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Abstract

Research has shown improved reading following visual magnocellular training in individuals with dyslexia. Many studies have demonstrated how the magnocellular pathway controls visual spatial attention. Therefore, we have investigated the relationship between magnocellular pathway and visual spatial attention deficits in dyslexia in order to better understand how magnocellular-based interventions may help children to learn to read. Magnocellular function, visual spatial attention and reading abilities of thirty elementary school students with dyslexia, aged between 8-10 were measured. The experimental group received magnocellular-based visual motion training for 12 sessions, while the control group received neutral sessions. All tests were repeated at the end of the training and after 1 month. The magnocellular functioning, visual spatial attention, and reading abilities of the experimental group improved significantly compared to the controls. Additionally, improvement in reaction time of invalid conditions predicted improvements in saccadic eye-movements. We conclude that visual magnocellular training improved saccadic eye-movement control, visual spatial orientation, and reading ability.

Keywords: developmental dyslexia, magnocellular pathway, visual spatial attention, learning Disability, reading Improvement

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Developmental Dyslexia (DD), the most prevalent learning disorder, is characterized by difficulty in word recognition, poor decoding, and poor spelling. These problems occur despite normal educational opportunity and in the absence of any intellectual, neurological or sensory deficit (American Psychiatric Association, 2013). DD affects about 4-10% of school aged children (Esser et al., 2002; Lyon et al., 2003; Pouretemad et al., 2011). The underlying mechanism of DD, hence how best to help these children, is still a controversial topic. The magnocellular theory of dyslexia (Stein, 2001; Stein & Walsh, 1997) has been studied extensively in different languages and cultures as well as by interventional studies (e.g. Ebrahimi et al., 2019; Flint & Pammer, 2019; Lawton & Shelley-tremblay, 2017).

The visual magnocellular pathway (M-pathway) originates from the retina and projects to the primary visual cortex (V1) through the magnocellular layers of the Lateral Geniculate Nucleus (LGN) (Goodale & Westwood, 2004). The M-dominated dorsal stream, also called the "where stream", receives 90% of its visual input from M-pathway, projects to V5/MT (the motion-sensitive area), and afterwards to the Posterior Parietal Cortex (PPC) (Boden & Giaschi, 2007; Goodale & Westwood, 2004; Stein, 2014). This pathway mediates motion perception and also visual attention and eye movement control (Dickinson & Badcock, 2009; Stein, 2014).

Evidence for an M-pathway deficit in DD comes from both structural and behavioral studies, not only in alphabetic (e.g. Ahmadi et al., 2015; Benassi et al., 2010; Chouake et al., 2012; Ebrahimi et al., 2019; Eden et al., 1994; Flint & Pammer, 2019; Livingstone et al., 1991; Lovegrove & Williams, 1993; Wright et al., 2012), but also in logographic languages (e.g. Qian & Bi, 2015; Zhao et al., 2014). Smaller, disorganized, and abnormal magnocellular layers in the LGN (Giraldo-Chica et al., 2015; Livingstone & Galaburda, 1993; Livingstone et al., 1991), poorer performance in coherent motion detection and abnormal saccadic eye movement control (Al

Dahhan et al., 2014; Bakhshalizadeh, 2012; Benassi et al., 2010; Biscaldi et al., 2000; Boets et al., 2011; Jednoróg et al., 2011; Qian & Bi, 2015; Rayner et al., 2013) have all been found in individuals with dyslexia. Improving magnocellular functioning by training motion detection or saccadic eye movements has resulted in improved reading accuracy and also reduced reading errors (Corbetta & Shulman, 2011; Dushanova & Tsokov, 2019; Ebrahimi et al., 2019; Lawton, 2016; Lawton & Shelley-tremblay, 2017; Sabet et al., 2013; Wilmer et al., 2004; Bucci, 2019 for a review). These studies have clearly established the relationship between M-pathway deficits and DD, but how the M-pathway deficit affects reading remains unclear.

The M-pathway is the main regulator of Visual Spatial Attention (VSA) (Laycock et al., 2008). VSA is attention to the spatial location of a visually presented object (Vecera & Rizzo, 2003). The Posterior Parietal Cortex (PPC) is considered to be the main cortical region controlling VSA (Corbetta et al., 1998; Hopf & Mangun, 2000; Leonards et al., 2000; Rosen et al., 1999). According to the Vidyasagar neural model of attention (Vidyasagar, 1999), the PPC uses information provided by the M-pathway to generate a spatial map of the visual field. The contribution of the M-pathway to VSA has been confirmed repeatedly (Franceschini et al., 2018; Gori et al., 2016; Kinsey et al., 2004; Pammer et al., 2006; Stein, 2019; Vidyasagar & Pammer, 2010; but see Wright et al., 2012).

There is strong evidence that there are 2 distinct systems supporting attentional control: endogenous (goal-driven) and exogenous (stimulus-driven) VSA (Chica et al., 2013; Valdois et al., 2019). Exogenous VSA seems to be mainly affected in DD individuals (e.g. Facoetti et al., 2010; Facoetti et al., 2006; Liu et al., 2018; Roach & Hogben, 2004; Ruffino et al., 2014) and at-risk pre-readers (Franceschini et al., 2012). Sluggish Attentional Shifting theory (SAS) (Hari & Renvall, 2001) suggests that individuals with DD are slower than non-DD in both engaging and

disengaging their attention and therefore show difficulties in orienting their attention during reading (Fu et al., 2019). The compelling evidence for this theory comes from studies examining exogenous VSA orientation applying the Posner (Posner, 1980) spatial cueing task with peripheral cues (e.g. Facoetti et al., 2003; Ruffino et al., 2014). This task to assess the orienting of VSA, consists of two different target locations (valid vs. invalid) with two different Stimuli Onset Asynchronies (SOA) between cue and target. Studies have revealed overall longer reaction times (RT) of DD relative to non-DD individuals in both different target locations and different SOAs. Also, studies have shown that DD individuals have shorter RTs after long compared with shorter SOAs. These findings confirm slower shifting of attention in DD individuals (Ding et al., 2016; Facoetti et al., 2003; Facoetti et al., 2008; Franceschini et al., 2018; Lallier et al., 2009; Ruffino et al., 2010).

The slower shifts of VSA in DDs probably arise from a deficit in M-pathway functioning (Franceschini et al., 2018). Likewise, neural and psychophysical studies have confirmed that the M-dominated dorsal stream mediates the early selection of features in space (Cheng et al., 2004; Martínez et al., 1999; Roelfsema et al., 1998). Hence the reading difficulties of DD individuals with M-pathway deficits can probably be attributed to VSA deficits. Nevertheless, some studies have not found any relationships between VSA and M-pathway weakness (Wright et al., 2012) or between VSA and reading abilities (Kermani et al., 2018). But most of the studies in this field were correlational and these cannot prove causation. However, intervention studies can provide such evidence. There is a discussion over trainability of cognitive functions. More recent studies suggest that this type of training can be more successful if it is domain specific and focused on specific processes like those that contribute to reading (i.e. Franceschini et al. 2017; Lawton, 2016; Peng & Miller, 2016).

Therefore, in the current study, we have investigated whether interventionally driven changes in the functioning of the magnocellular pathway leads to changes in visual spatial attention. Understanding this mechanism might help us elucidate the mechanism by which magnocellular training can help children to improve their reading.

Method

Participants

Thirty primary school students (mean age: 8.3 years) with DD participated in the study (2 females). They were recruited from grade two and three of elementary schools in Tehran, Iran. Participants were assigned randomly to experimental and control groups (15 participants in each group). All of them had a reading score below 71 on the reading test and a Random Dot Kinematogram (RDK) mean score above 34 as inclusion criterion. These cut offs were stablished for similar populations relative to normal readers by Bakhshalizadeh (2012). They did not have any neurological or sensory deficits and they had normal IQ with no clinical interventions during or before the study. Using Conners rating scale, DD participants with probable comorbid ADHD were excluded. The two groups were matched for age, IQ, educational level, reading accuracy, magnocellular functioning, and VSA performance.

Ethics - All the children agreed voluntarily to join the study, and their parents provided written informed consent under a protocol that was approved by the Shahid Beheshti University, Iran and all methods were performed in accordance with the relevant guidelines.

Assessments

Reading abilities

Persian Reading Ability Assessment (APRA). Participants' reading ability was assessed by the APRA (Pouretemad et al., 2011). This Persian reading test includes 10 texts which evaluate

reading accuracy. Furthermore, three categories of reading errors are obtained as follow: visual errors: which contains omission (omitting a word partially or fully), addition (adding an extra word or letter to the text), and reversal (reversing the letters of a word), phonological errors: which contains mispronunciation (pronouncing the word wrongly), substitutions (using incorrect word with same initial phoneme instead of the target word), and fragmentations (breaking the words into its components and then combining it to read) and pragmatic errors: which contains refusal (pausing 5-7 seconds with no effort to read) and repetition (rereading the whole word). Following each text, participants were asked questions about the given text. The scores were calculated as comprehension scores. This test has been used in previous studies of Persian speaking students' reading abilities (e.g. Ahmadi et al., 2015).

Magnocellular functioning

Random Dot Kinematogram (RDK). A two-panel RDK -containing two sets of moving dots- was used in order to assess the M-pathway functioning (Stein, 2001). In each trial, the participant is required to select a panel in which the dots move more coherently together. The proportion of the dots moving coherently was varied based on the previous answer. A correct answer decreased it by 1dB and a wrong one increased it by 3 dB. The threshold calculated from this 'staircase' is the proportion of coherently moving dots which participant can recognize correctly on 75% of trials.

Saccadic eye movement. According to the method of (Jafarzadehpur et al., 2007; Sabet et al., 2013) two targets were placed at the ends of a 30cm horizontal ruler. The ruler was placed 20cm from the participant. Examiner sat in front of the participant and asked them to shift gaze from one target to the other as quickly as possible to see the targets clearly, while the head was

held still. The number of cycles -(a left followed by a right saccade)- per minute was recorded by examiner.

Visual spatial attention (VSA)

The VSA tasks were designed according to the Posner paradigm and based on Facoetti et al. (2010), Facoetti et al. (2010), and Ruffino et al. (2014).

VSA reaction time (RT). Stimuli were white and were presented on a black background. Two circles (2/5°) were presented to the left and right of the fixation point. A cue (the thickening of one of the circles) as to which side the target would appear was presented for 40 msec; it could be either valid or invalid. The target stimulus was presented 60 or 210 msec (Inter Stimulus Interval -ISI) after the cue; it was a 0.5° dot located in the center of one of the circles (lasting for 40 msec). The participant was asked to press a key on the keyboard as quickly as possible when he saw the target. Performance of the participant in valid or invalid trails and for the two SOAs was calculated as an index of VSA orientation. Additionally, catch trials that did not consist of any target stimulus were intermixed with response trials. A total of 128 trials were presented including 104 target trials (equal for each condition and each SOA) and 24 catch trials. Simple RT, errors, and also cueing effect (invalid-valid RTs for each SOA) as an index of attention orientation were recorded (Fig. 1A).

VSA accuracy- Two horizontal bars were located peripherally to the right and left of the fixation point. The cue, which was thickening of one of the bars for 50 msec, was followed by the target stimulus. The target was an ellipse rotated ±30° or ±60°, displayed for 180 msec. Responses were not time-limited, and the participant was asked to select the orientation of the target just seen from among the four possibilities. A total of 36 trials were presented. The percentage of correct responses was recorded (Fig. 1B).

Magnocellular based visual motion training:

This training program was the same as Ebrahimi et al. (2019) .The efficacy of this training on magnocellular functioning and reading improvement has been well established. This training consists of both computerized and non-computerized training and also between session training.

RDK (version modified for training)

This was similar to the RDK task except that an auditory feedback was added to inform the participant of errors. This training is referred to as perceptual learning task relevant.

Saccadic eye movement training

This was again similar to the saccadic eye movement assessment except that in each trial the distance between the two targets was reduced. In each trial, the head was kept fixed, and correct saccadic eye movements were counted out loud in order to provide feedback. Each training session consisted of 10x one-minute saccadic eye movements with a 1-minute break between each.

Digit counting

In this computerized program digits 0-9 were presented from right to left (the Persian reading direction) at random or consecutively. This program had 10 levels. The presentation time of digits in level 1 was 560 msec and it decreased 37 msec each level i.e. in the level 10 the presentation time of digits was 190 msec. The font size was reduced also. The participant was asked to choose a favorite number and report how many times that number was presented. Both correct and incorrect answers were fed back.

Dot counting

Dots were presented in a virtual square sequentially; each dot disappeared before the next appeared. The participant was asked to trace the dots and report the number in each trial. Feedback

was provided. When the participant responded correctly on 80% of the trials at one level they proceeded to a higher level (decreased interval time).

Between session training

Participants were given a printed version of counting digits in a notebook containing 7 pages of rows of random digits 0-9. Throughout the notebook, the number of digits increased and their font size decreased. The participants were asked to count the numbers as quickly as possible while their head was kept fixed. Parents were instructed on how to perform the training and report the results. Some of the studies suggest that these tasks could implicitly influence the visual spatial attention and so this factor should take into account in the interpretation of their effects.

Procedure

All the assessments as well as the computerized magnocellular based visual-motion training were carried out on MacBook Pro 13-inch personal laptop 50 cm away from the participants in a dimly lit and quiet room. All the sessions were held in the school and the examiner was with the participants during the session in both group. During all the tests and training sessions, a chinrest was used to keep the head fixed. Baseline 'pre-tests' were conducted at the beginning. The experimental group received 12 sessions of magnocellular based visual-motion training lasting 30-40 minutes two days a week. The control group received the same pattern of neutral sessions consisting of a non-training computerized game. This game was about cooking cakes and cookies. This allowed us not to have a completely passive control group and have better comparable condition across groups. The post-test was conducted immediately after the last session. Assessments were repeated again 1 month later.

Data analysis

Missed responses and responses on catch trials in the visual spatial attention task were excluded from the results, and not analyzed. The accuracy rate in each condition was above 70 percent. Two participants in the control group missed their follow-up assessments, therefore they were excluded from the final analysis. Also, data of two participants in the experimental group were recognized as outliers and were not included in analysis. Smirnov-Kolmogorov test revealed normal distribution for data ($p > 0.005$). We carried out a repeated measured ANOVA, If the Mauchuly's assumption had been violated, Greenhouse-Geisser corrections were performed. A set of t-tests was also carried out. $p<0.005$ was considered for significance in all analysis.

Results

Pre-test comparisons

Independent t-tests were conducted at pre-test to check for differences between the two groups. There were no significant differences between the groups in age, IQ performance, reading accuracy, reading comprehension, M-pathway functioning (RDK, saccadic eye movements-potentially saccadic eye movements are training of over attentional orienting and that is preceded by an covert attentional orienting-), VSA performance (Mean RT on SOA 100, Mean RT on SOA 250, Mean RT on valid condition, Mean RT on invalid condition, Mean accuracy in valid condition, or Mean accuracy in invalid condition. (see Table 1).

M-pathway functioning: treatment comparison

RDK.

There were significant main effects of group ($F[1,24] = 13.27$, $p < 0.001$, $\eta^2_p = 0.35$), the assessment session ($F[1.36,32.68] = 30.61$, $p < 0.001$, $\eta^2_p = 0.56$) and also a significant group × assessment session interaction ($F[1.36,32.68] = 13.98$, $p = 0.001$, $\eta^2_p = 0.36$) for the RDK

threshold scores. The two groups had no significant differences in pre-test, but students with dyslexia in the experimental group had lower thresholds at post-test ($t(24) = 4.69$, $p < 0.001$) and at the 1 month follow-up ($t(24) = 5.43$, $p < 0.001$) compared to control group. Also, pairwise comparisons within experimental group showed significant reduction in RDK scores from pre-test to post-test (mean difference = 24.62, $p < 0.001$) and at the 1 month follow-up (mean difference = 26.08, $p < 0.001$) indicating significant improvements in motion detection in the experimental group after the intervention. But the control group showed no significant reductions at any of the assessment sessions ($p > 0.05$; Fig. 2A).

Saccadic eye movement.

Repeated measure ANOVA revealed significant main effects for group ($F[1,24] = 33.41$, $p < 0.001$, $\eta^2_p = 0.58$), assessment session ($F[1.07,25.86] = 126.31$, $p < 0.001$, $\eta^2_p = 0.84$) and significant group × assessment session interaction ($F[1.07,25.86] = 99.22$, $p < 0.001$, $\eta^2_p = 0.80$) for number of saccadic eye movements. There was no significant difference between experimental and control group at pre-test, but experimental group had higher saccade scores in post-test ($t(24) = 7.42$, $p < 0.001$) and follow-up ($t(24) = 9.03$, $p < 0.001$) compared to control group. Pairwise comparisons within experimental group, as well, showed significant improvement in saccadic eye movement from pre-test to post-test (mean difference = -43.00, $p < 0.001$) and to follow-up (mean difference = -48.54, $p < 0.001$), but there was no significant improvement in control group ($p > 0.05$; Fig. 2B).

Visual spatial attention-Reaction time: treatment comparison

A repeated measure ANOVA was carried out with cue-target SOA (100 vs. 250) and target condition (valid vs. invalid) as within-subject factors and group (experimental vs. control) as between-subject factor. There were significant main effects of assessment session (F[1.01,25.41]

= 2.21, p = 0.02, η^2_p = 0.20), cue-target SOA (F[1,24] = 23.94, p < 0.001, η^2_p = 0.49) and target condition (F[1,24] = 13.37, p = 0.001, η^2_p = 0.35), but the main effect of group was not significant (F[1,24] = 2.57, p = 0.12). Also, there were significant group × assessment session interaction (F[1.01,25.41] = 7.57, p = 0.01, η^2_p = 0.24), and target condition × assessment session interaction (F[1.1,26.55] = 6.05, p = 0.01, η^2_p = 0.20). Crucially the group × assessment session × target condition interaction as an index of VSA orientation was also significant (F[1. 1,26.55] = 4.30, p = 0.04, η^2_p = 0.15). The other interactions were not significant (p > 0.05). For more analysis, we calculated the mean RT for valid, invalid, SOA 100 and SOA 250 trials and repeated the repeated measure ANOVA.

Cue-target SOA.

Mean RT on SOA 100- There were significant main effects of assessment session (F[1.04,25.18] = 4.50, p = 0.04, η^2_p = 0.15) and also a significant group × assessment session interaction (F[1.04,25.18] = 5.44, p = 0.02, η^2_p = 0.18), but the main effect of group was not significant (F[1,24] = 2.23, p = 0.14). An independent samples t-test showed a significant difference between the experimental and the control group mean RT on SOA 100 only in follow-up (t(24) = 2.25, p = 0.03) driven by a shorter RT in the experimental group, which shows that students with dyslexia in experimental group had lower mean RT on SOA 100 after intervention. Also, pairwise comparisons within the experimental group showed significant reductions in mean RT on SOA 100 from pre-test to follow-up (mean difference = 0.94, p = 0.01), but the control group showed no significant reductions through assessment sessions (p > 0.05; Fig. 3A).

Mean RT on SOA 250- There was a significant main effect of assessment session (F[1.02,24.47] = 6.65, p = 0.01, η^2_p = 0.21) and also a significant group × assessment session interaction (F[1.02,24.47] = 7.84, p = 0.009, η^2_p = 0.24). But the main effect of group was not

significant (F[1,24] = 2.47, p = 0.12). There were no significant differences between the two groups at pre-test, but individuals in experimental group had shorter RTs at follow-up (t(24) = 2.77, p = 0.01) compared to the control group. Pairwise comparisons within the experimental group showed significant reductions in mean RT on SOA 250 from pre-test to post-test (mean difference = 0.97, p = 0.04) and to follow-up (mean difference = 0.136, p = 0.008), but the control group showed no significant reductions at any of the assessment sessions (p > 0.05; Fig. 3B).

***Target condition*.**

Mean RT on valid conditions- There was a significant group × assessment session interaction (F[1.02,24.46] = 4.36, p = 0.04, η^2_p = 0.15) . But the main effect of group (F[1,24] = 1.8, p = 0.1), and main effect of assessment session (F[1.02,24.46] = 2.88, p = 0.1) were not significant. An independent samples t-test showed a significant difference between the experimental and the control group mean RT on valid cue trials only in follow-up (t(24) = 2.09, p = 0.04) caused by a shorter RT in the experimental group, i.e. the students with dyslexia in experimental group had a shorter mean RT on valid conditions after the intervention. Also, pairwise comparisons within the experimental group showed a significant reduction in mean RT on valid cue trials from pre-test to follow-up (mean difference = 0.78, p = 0.04), but control group showed no significant reductions at any of the assessment sessions (p > 0.05; Fig. 4A).

Mean RT on invalid conditions- There were significant main effects of assessment session (F[1.02,24.64] = 8.20, p = 0.008, η^2_p = 0.25) and also a significant group × assessment session interaction (F[1.02,24.64] = 8.85, p = 0.006, η^2_p = 0.26), but the main effect of group was not significant (F[1,24] = 2.76, p = 0.1). The two groups had no significant differences in pre-test, but individuals in the experimental group had marginally significant lower RT at post-test (t(24) = 1.81, p = 0.08) and a significantly lower RT at follow-up (t(24) = 2.7, p = 0.008) compared to the

control group. Pairwise comparisons within the experimental group showed significant reduction in mean RT on invalid cue trials from pre-test to post-test (mean difference = 0.113, $p = 0.03$) and at follow-up (mean difference = 0.151, $p = 0.004$), but the control group showed no significant reductions through assessment sessions ($p > 0.05$; Fig. 4B).

Cueing effect

The main effect of assessment session ($F[1.08,25.97] = 6.82$, $p = 0.01$, $\eta^2_p = 0.22$), and the group × assessment session interaction was marginally significant ($F[1.08,25.97] = 3.07$, $p = 0.08$, $\eta^2_p = 1.00$) for cueing effect for SOA 250, indicating faster orienting of attention in experimental group after training in longer SOA. An independent samples t-test showed a significant difference between the experimental and the control group cueing effect for SOA 100 ($t(24) = 2.06$, $p = 0.05$) at follow-up.

Visual spatial attention-Accuracy: treatment comparison

A repeated measure ANOVA was carried out with target condition (valid vs. invalid) as within-subject factor and group (experimental vs. control) as between-subject factor. There were significant main effects of assessment session ($F[1.19,28.63] = 6.19$, $p = 0.01$, $\eta^2_p = 0.20$), and group ($F[1,24] = 9.13$, $p = 0.006$, $\eta^2_p = 0.27$), but the main effect of target condition was not significant ($F[1,24] = 2.48$, $p = 0.12$). Also, there was marginally significant group × assessment session interaction ($F[1.19,28.63] = 3.2,9$ $p = 0.07$, $\eta^2_p = 0.12$). The other interactions were not significant ($p > 0.05$). For more analysis, we calculated the mean accuracy for valid and invalid trials and repeated the repeated measure ANOVA.

Mean accuracy on valid trials

None of the effects were statistically significant ($p > 0.05$; Fig. 5A).

Mean accuracy on invalid trials

Repeated measure ANOVA revealed significant main effects for assessment session (F[2,48] = 13.38, p < 0.001, η^2_p = 0.35), group (F[1.24] = 12.28, p = 0.002, η^2_p = 0.33) and a significant group × assessment session interaction (F[2,48] = 7.93, p = 0.004, η^2_p = 0.24). There were no significant differences between experimental and control groups at pre-test, but the experimental group had higher accuracy scores post-test (t(24) = 3.65, p = 0.001) and at follow-up (t(24) = 4.40, p < 0.001) compared to the control group. Also, pairwise comparisons within the experimental group showed significant improvements in mean accuracy on invalid cue trials from pre-test to post-test (mean difference = -20.08, p = 0.003) and to follow-up (mean difference = -19.56, p = 0.002), but the control group had no significant improvement through assessment sessions (p > 0.05; Fig. 5B).

Cueing effect

None of the effects were statistically significant (p > 0.05).

Reading abilities: treatment comparison

Reading accuracy.

There were significant main effects of group (F[1,24] = 5.39, p = 0.02, η^2_p = 0.18), assessment session (F[1.08,28.67] = 144.2, p < 0.001, η^2_p = 0.85) and also a significant group × assessment session interaction (F[1.10,28.67] = 34.09, p < 0.001, η^2_p = 0.58) for reading accuracy scores. There were no significant differences between two groups in pre-test, but students with dyslexia in experimental group had higher scores in reading accuracy post-test (t(24) = 3.32, p = 0.003) and at the 1 month follow-up (t(24) = 3.16, p = 0.004) compared to the control group. Pairwise comparisons within the experimental group showed a significant improvement in reading accuracy from pre-test to post-test (mean difference = -21.81, p < 0.001) and to follow-up (mean difference = -25.21, p < 0.001), and also pairwise comparisons within control group showed

significant improvements in reading accuracy from pre-test to post-test (mean difference = -5.82, $p = 0.006$) and to follow-up (mean difference = -10.00, $p < 0.001$; Fig. 6A).

Reading comprehension.

There was a significant main effect of assessment session ($F[1.09,26.34] = 6.4$, $p = 0.01$, $\eta^2_p = 0.21$) and also a significant group × assessment session interaction ($F[1.09,26.34] = 4.6$, $p = 0.03$, $\eta^2_p = 0.16$) for reading comprehension scores, but the main effect of group was not significant ($F[1,24] = 0.07$, $p = 0.79$). Pairwise comparisons within the experimental group showed significant improvements in reading comprehension scores from pre-test to post-test (mean difference = -12.39, $p = 0.01$) and to follow-up (mean difference = -16.82, $p = 0.004$), but there was no significant improvement in the control group ($p > 0.05$; Fig. 6B).

Reading errors.

Visual errors- There was a significant main effect of assessment session ($F[1.65,36.99] = 18.86$, $p < 0.001$, $\eta^2_p = 0.44$) and a significant group × assessment session interaction ($F[1.65,36.99] = 5.94$, $p = 0.008$, $\eta^2_p = 0.19$) for mean of visual errors. Also, there was a marginally significant main effect of group ($F[1,24] = 3.05$, $p = 0.09$, $\eta^2_p = 0.11$). There were no significant differences between the two groups pre-test, but the students with dyslexia in the experimental group had lower visual errors post-test ($t(24) = 2.41$, $p = 0.02$) and at the 1 month follow-up ($t(24) = 2.81$, $p = 0.01$) compared to the control group. Pairwise comparisons within the experimental group showed significant reductions in visual errors from pre-test to post-test (mean difference = 5.16, $p < 0.001$) and at follow-up (mean difference = 6.30, $p < 0.001$), but the control group had no significant reductions throughout the assessment sessions ($p > 0.05$; Fig. 7A).

Phonological errors- There were a significant main effect of group ($F[1,24] = 9.22$, $p = 0.006$, $\eta^2_p = 0.27$), and a significant group × assessment session interaction ($F[1.54,37.11] = 6.57$,

$p = 0.007$, $\eta^2_p = 0.21$) for mean of phonological errors, but there was no significant main effect of assessment session (F[1.54,37.11] = 1.68, $p = 0.2$). Pairwise comparisons revealed a significant increase in phonological errors in the experimental group, between pre-test and post-test (mean difference = -3.84, $p = 0.003$) and follow-up (mean difference = -2.18, $p = 0.04$), but the control group showed no significant changes throughout the assessment sessions ($p > 0.05$; Fig. 7B).

Pragmatic errors- none of the effects were significant ($p > 0.05$; Fig. 7C).

Regression analysis

A two-step multiple regression analysis was performed on the entire sample (28 students with dyslexia). The dependent variable was reading accuracy changes (the difference between reading accuracy scores between the 1 month follow-up and pre-test) and the predictors were: (1) age (2) RDK mean score changes, (3) saccadic eye movement changes, (4) mean RT on SOA 100 changes, (5) mean RT on SOA 250 changes, (6) mean RT on valid trials changes, and (7) mean RT on invalid trials changes. All the scores were calculated as the difference between follow-up and pre-test scores. Results showed that saccadic eye movement changes and age accounted for 52.9% of variance in reading accuracy ($p < 0.001$). Improvement in saccadic eye movement changes accounted for 46.4% of the changes in reading accuracy ($p < 0.001$; Table 2). In order to elucidate the predictor factors for the saccadic eye movement changes, we performed a three-step multiple regression analysis with (1) age (2) RDK mean score changes, (3) mean RT on SOA 100 changes, (4) mean RT on SOA 250 changes, (5) mean RT on valid trials changes, and (6) mean RT on invalid trials changes as predictors. The mean RT on invalid trials changes, the RDK score changes and age accounted for 44.9% of variance in reading accuracy changes ($p = 0.001$). Both mean RT on invalid trials changes and the RDK score changes accounted for 35.2% and

Improvement in RT on invalid trials itself accounted for 25% of improvement in saccadic eye movement ($p = 0.005$; Table 3). The correlation matrix is provided in table 4.

Discussion

In this study, we asked whether training magnocellular pathway (M-pathway) function in children with developmental dyslexia through visual motion training, can influence their visual spatial attention (VSA) and reading abilities. Our results indicate that visual motion training can improve the functioning of the M-pathway. This improvement was associated with faster target detection and improved accuracy in VSA. In addition, the visual motion training resulted in improved reading in children with DD compared with those who didn't receive it. We also showed that the improvement in reading can best be explained by the changes in saccadic eye movement control. This improvement in saccadic eye movements was best predicted by the changes in RT during invalid trials.

The results of this study suggest that practice and repeated exposure can improve motion detection and eye movement control. This is in line with many other studies in this field (Dushanova & Tsokov, 2019; Ebrahimi et al., 2019; Lawton, 2016; Lawton & Shelley-tremblay, 2017; Sabet et al., 2013; Wilmer et al., 2004). Also, improvement in motion detection mediated by the M-pathway, may improve saccadic eye movement since the M-input dominates the dorsal stream forwards to the PPC, which is a crucial area for saccadic eye movement control (Dickinson & Badcock, 2009; Stein, 2014).

After the magnocellular based training, participants in the experimental group had lower RTs in the visual spatial attention task and made more accurate responses. This finding is in line with studies using action video games as training of M-D, visual spatial attention and reading (e.g., Bertoni et al., 2021; Franceschini & Bertoni, 2019; Peters et al., 2019)

Before training, there seems to be no difference between 100 and 250 SOAs RTs which indicates the absence of warning effect. Besides, at the post-test and better at the follow-up the warning effect appears, indicating improvement in the alert mechanisms mainly controlled by the right fronto-parietal network (Posner & Petersen, 2012). Furthermore, results show that improvements in RTs of experimental group are grater at SOA 250 than at SOA 100 between pre and post as well as pre and follow-up, which indicates greater improvement in the long SOA. The reason for this finding is perhaps because it is more difficult to achieve improvement in shorter SOA because of the SAS (Hari and Renvall, 2001; Facoetti et al., 2010). Additionally, the treatment effect appears to be greater in the invalid cue condition than in the valid cue condition, suggesting a greater effect of M-pathway training on disengagement than engagement of VSA. Also, the accuracy difference seems to be greater in the invalid condition between the two groups in the post and follow-up, confirming the results with the RTs.

The SAS theory (Hari & Renvall, 2001) suggests that when DD individuals face a rapid sequence of stimuli, they are slower to disengage from one item and move on to the next, and this is thought to be the result of a failure of the automatic attentional system (Lallier et al., 2010). This impairment could result in impoverished phoneme/grapheme representations and thus explain the reading difficulties (Lallier et al., 2013). Neuroimaging studies have revealed increased cortical activity in the PPC while shifting attention to peripheral locations (e.g. Corbetta et al., 1998; Giesbrecht et al., 2003). As discussed earlier, the PPC is believed to be a major endpoint of the M-dominated dorsal stream (e.g. Boden & Giaschi, 2007) and therefore this stream has been hypothesized to mediate the visual direction of attention and also visual guidance of eye movements during reading (e.g. Cheng et al., 2004; Stein, 2014).

Abnormal or weakened input of the M-pathway to the dorsal stream (Boden & Giaschi, 2007; Stein, 2019; Vidyasagar & Pammer, 2010) could be the neurobiological substrate for sluggish attentional disengagement in DD (Ruffino et al., 2010). Facoetti et al. (2000) suggested that DD children with M-pathway deficits are not able to focus their attention or to inhibit irrelevant stimuli during reading. Accordingly, it has been suggested that any improvement in M-pathway functioning would contribute to VSA improvement. Our results are in line with other studies which found a relationship between M-pathway and VSA (Franceschini et al., 2018; Gori et al., 2016; Kinsey et al., 2004; Pammer et al., 2006; Stein, 2019). Nevertheless, Kermani et al. (2018) have suggested that impairment in VSA is a result and not the cause of reading difficulties. If this were true, only training directly engaging reading would improve VSA, yet our training didn't involve reading at all.

Additionally, our training did improve reading. This is consistent with Ebrahimi et al. (2019) together with many other studies showing that magnocellular training can improve reading (e.g. Dushanova & Tsokov, 2019; Lawton, 2016; Lawton & Shelley-tremblay, 2017; Wilmer et al., 2004). The role of the M-pathway in letter decoding and hence reading abilities is now well established. According to Morrison (1984), while processing a fixated word during reading, multiple internal orientations of VAS occur across the different spatial locations of the text in order to process words parafoveally and thus to program the next saccadic eye movement. This mechanism improves accuracy and fluency (Rayner et al., 2013) and is vital for accurate reading (Franceschini et al., 2018). So, improvement in this mechanism is expected to lead to more accurate reading.

Despite improved reading accuracy and comprehension following training of the M-pathway, the number of phonological errors increased in our experimental group. One explanation

can be found in Siegel (1993) study, who found that individuals who had impairments in the sub-lexical route (phonological skills) relied more on the lexical route to compensate. It is possible that during our training, individuals learned to use the lexical information more than the sub-lexical and as a result, their phonological errors increased temporarily. Interestingly, by the follow-up after one month, the number of these errors had decreased. Once an association between VSA and the impaired sub-lexical route (phonological decoding) has been established (Facoetti et al., 2010, 2006; Ruffino et al., 2014), improvement in VSA was then accompanied by a decrease in phonological errors.

Regression analysis revealed that improvement in saccadic eye movement control predicted reading accuracy improvement. In addition, a faster RT on invalid trials predicted improvement in saccadic eye movement control. Faster RT on invalid trials can be attributed to faster disengagement of attention. As discussed above, faster VSA disengagement can programs the next saccadic eye movement which could lead to more accurate reading (Morrison, 1984).

This study had some limitations and cautions for the interpretation of the findings. First, it was carried out on Farsi speaking students which differs systematically from English or Latin languages. For instance, in Farsi we use the Arabic alphabet, it is completely cursive (‘joined up’), and written from right to left. So, generalization of our findings to other languages should be done with caution. Second, we used the same texts in all our assessment sessions which might have enabled a learning effect. This could explain why the scores of reading accuracy were improved in the control group too. Further studies that use different text for pre- and post-test can give us a clearer image. Third, in VSA tasks participants are required to fix their eyes on the fixation spot and are not permitted to make eye movements. Using eye-tracking would better ensure that the eyes didn’t move.

Conclusion

Taken together these results show that changes in M-pathway function are associated with improvements in visual spatial attention task in DD. Thus, furthers studies are needed to investigate if M-pathway training improve reading through changes in VSA.

Conflicting Interests

The authors declare no conflicting interests.

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Table 1.

Mean (M), standard deviation (SD), and comparison of age, IQ, reading accuracy Reading comprehension, M-pathway functioning (RDK and saccadic eye movement), and VSA performance (Mean RT on SOA 100-250, Mean RT on Valid-invalid condition, and Mean accuracy on valid-invalid condition) at pre-test between experimental (E) and control (C) groups.

	E (n = 13)		C (n = 13)		comparison	
	M	*SD*	*M*	*SD*	*t(24)*	*p*
Age (years)	8.5	1.05	8.2	0.59	0.91	0.36
IQ (Raven)	97.3	4.9	95	2.8	1.46	0.15
Reading accuracy	48.4	14	46.1	15.6	0.39	0.69
Reading comprehension	36.7	17.9	47.5	20.5	1.42	0.16
RDK	58.1	7.9	56.5	16	0.32	0.74
Saccadic eye movement	23.3	14.4	24.4	12.8	0.20	0.84
Mean RT on SOA 100 (sec)	0.48	0.08	0.47	0.08	0.06	0.94
Mean RT on SOA 250 (sec)	0.46	0.08	0.42	0.08	1.02	0.31
Mean RT on valid condition (sec)	0.43	0.07	0.42	0.07	0.18	0.85
Mean RT on invalid condition (sec)	0.51	0.09	0.48	0.08	0.83	0.41
Mean accuracy on valid condition	46.5	17.6	0.42	19.9	0.52	0.60
Mean accuracy on invalid condition	37.5	13.2	35	11.3	0.51	0.60

Table 2.
Two step regression analysis predicting reading accuracy changes

		Unstandardized coefficients		*Unstandardized coefficients*					
step	*predictor*	*B*	*SE*	*ß*	*p*	R^2	*Adjusted* R^2	*F*	*p*
1						0.48	0.46	22.60	0.0001
	Saccadic eye movement changes	0.27	0.05	0.69	0.0001				
2						0.56	0.52	15.03	0.0001
	Saccadic eye movement changes	0.30	0.05	0.77	0.0001				
	Age	-3.54	1.70	-0.29	0.049				

Table 3.
three step regression analysis predicting saccadic eye movement changes

		Unstandardized coefficients		*Unstandardized coefficients*					
step	*predictor*	*B*	*SE*	*ß*	*p*	R^2	*Adjusted* R^2	*F*	*p*
1						0.28	0.25	9.3	0.005
	Mean RT on invalid trials changes	-101.2	33.1	-0.52	0.005				
2						0.40	0.35	7.7	0.003
	Mean RT on invalid trials changes	-77.5	32.6	-0.40	0.02				
	RDK mean score changes	-0.57	0.26	-0.37	0.03				
3						0.51	0.44	7.8	0.001
	Mean RT on invalid trials changes	-8.52	30.5	-0.46	0.008				
	RDK mean score changes	-0.54	0.24	-0.35	0.03				
	Age	10.20	4.5	0.33	0.03				

Table 4.

Descriptive statistics and correlations for changes score.

variable	*n*	*M*	*SD*	1	2	3	4	5	6	7	8
1.Age	26	8.38	0.85	-							
2. Reading accuracy changes	26	17.61	10.21	-0.097	-						
3.RDK mean score changes	26	-15.46	16.72	0.01	-0.483*	-					
4.Saccadic eye movement changes	26	25.53	25.63	0.258	0.696**	-0.507**	-				
5.Mean RT on SOA 100 changes	26	-0.045	0.097	0.046	-0.0459*	0.154	-0.471	-			
6.Mean RT on SOA 250 changes	26	-0.064	0.13	0.139	-0.556**	0.400*	0.596*	0.853**	-		
7.Mean RT on valid trials changes	26	-0.035	0.097	0.003	-0.443*	0.236	-0.411*	0.905**	0.894**	-	
8.Mean RT on invalid trials changes	26	-0.074	0.13	0.167	-0.555**	0.331	-0.529**	0.904**	0.948**	0.804**	-

*$p<0.05$; **$p<0.01$