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Evaluating the Magnocellular Deficit Theory of Dyslexia Using the Flash-Lag Effect

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The flash-lag effect (FLE) occurs when one perceives a moving object ahead of a stationary object while in reality the two are aligned (Nijhawan, 2001). Reducing magnocellular processing eliminates the effect (Chappell & Mullen, 2010). One prominent but controversial model of developmental dyslexia is the magnocellular deficit theory (e.g., Stein & Walsh, 1997). In the current experiment, participants with and without dyslexia viewed two FLE illusions, one designed to maximize and one to minimize magnocellular processing. Reducing brightness contrast (to minimize magnocellular processing) significantly reduced the magnitude of the FLE, which is consistent with previous findings. However, no effect of dyslexia was observed; this null finding does not support the notion that individuals with dyslexia suffer from a magnocellular deficit.

Keywords: Dyslexia, Magnocellular, Flash-lag Effect, Magnocellular Deficit theory of Dyslexia

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Dyslexia can be defined as “the inability to read effortlessly or with understanding” (Mayeux & Kandel, 1991, p. 850). Various neurological anomalies have been proposed to be causally related to this disorder. For example, a recent brain imaging study compared lateralization in children with and without dyslexia. The findings confirmed previous reports that the planum temporale, a cortical region in the temporal lobe, was asymmetrical (i.e., larger in the left hemisphere) in normal readers but symmetrical in readers with dyslexia, even when controlling for gender and handedness (Bloom, Garcia-Barrera, Miller, Miller, & Hynd, 2013). As the planum temporale plays a role in auditory processing, this result may relate to the phonological difficulties that represent the most common symptom of dyslexia (Ramus, 2003).

Other suggested neural or information-processing differences between dyslexic and non-dyslexic readers have focused on the visual system. For example, individuals with dyslexia differ from control participants in the ease with which they visually identify letters displaced from a fixation point. In control participants, letter identification accuracy drops as distance from the fixation points increases. Participants with dyslexia, however, often continue to accurately name letters far from the fixation point. Interestingly, the direction of the effect in readers with dyslexia corresponds to the direction of reading in their language (i.e., left to right versus right to left). Hebrew readers with dyslexia have enhanced visual perception for letters to the left of a fixation point, while in English readers with dyslexia, the advantage is to the right (Geiger, Lettvin, & Zeggara-Moran, 1992).

Another visual processing explanation for dyslexia involves the magnocellular pathway, which carries information about motion, overall shape, and small light-dark changes; the complimentary parvocellular system, originating from cones, carries information about detail and color. Both pathways lead to the thalamus, but action potentials from the magnocellular system arrive 7-10 ms sooner (Maunsell & Gibson, 1992). A deficit in magnocellular processing has long been hypothesized to be the root cause of developmental dyslexia (e.g., Stein & Walsh, 1997). However, the topic remains highly controversial, with many researchers arguing in favor of (e.g., Chase, Ashourzadeh, Kelly, Monfette, & Kinsey, 2005) as well as against (e.g., Skottun & Skoyles, 2005) the magnocellular deficit theory of dyslexia.

In attempting to resolve the dispute and pinpoint physiological causes for reading difficulties, many researchers have examined perceptual experiences believed to rely on magnocellular processing in readers with and without dyslexia, such as coherent motion and Ternus tests. Coherent motion tests consist of arrays of moving dots. Most of these dots move randomly from frame to frame, but some percentage of them all move in one direction. The percentage of dots that must move in one direction before a participant perceives directional rather than random motion is then recorded. In Ternus tests, participants view three identical objects in one frame which are then shifted to either the left or right. Participants either perceive that the object on the end jumps over the middle one (i.e., element movement) or that all three objects shift in the same direction (i.e., group movement). Perceiving group motion has been attributed to magnocellular processing (Slaghuis & Ryan, 1999).

Slaghuis and Ryan (1999) found that children with dyslexia exhibited lower sensitivity to contrast, required a higher percentage of dots moving in a common direction in order to perceive coherent motion, and showed an increased tendency to view element rather than group motion in Ternus stimuli. Similarly, Talcott, Hansen, Assoku, & Stein (2000) reported coherent motion detection impairment in adults participants with dyslexia.

However, Boets, Wouters, van Wieringen, & Ghesquière (2006) found no differences in coherent motion perception between children considered to be at high versus low risk of developing dyslexia due to family history.

Additionally, Skottun (2001) found Ternus tests to be ineffective at isolating the magnocellular pathway. In fact, shorter interstimulus intervals (ISIs), which elicit element rather than group motion perception, were associated with higher temporal frequencies and likely magnocellular, not parvocellular, stimulation. Finally, a review of contrast sensitivity studies revealed that although findings of reduced contrast sensitivity in those with dyslexia exist, those were “outnumbered by both the studies which have found no loss of sensitivity and the studies which have found contrast sensitivity reductions inconsistent with a magnocellular deficit” (Skottun, 2000, p. 125).

An alternative, novel method of assessing magnocellular processing may be perception of the flash-lag effect (FLE). The FLE occurs when one projects a moving object ahead of its true location to compensate for the lag between the time when a light stimulus enters the eye and the time this stimulus is processed by the brain as an image (Nijhawan, 2001). A recent explanation of the FLE relies on the magnocellular pathway. As this pathway is responsive to brightness but not color differences, making the moving object and its background equiluminant should minimize magnocellular processing (Chappell, Hine, & Hardwick, 2002). This manipulation, however, did not consistently reduce FLE magnitude in most participants (Chappell, Hine, & Hardwick, 2002), apparently because the moving stimulus still activated magnocellular cells (Chappell & Mullen, 2010). Conversely, the FLE was eliminated by both removing the contrast between the moving object and the background and immersing it in luminance noise (Chappell & Mullen, 2010).

In the present experiment, readers with and without dyslexia were tested with two FLE stimuli. One of these was designed with high brightness contrast between the moving object and its background, in order to maximize magnocellular processing. If dyslexia does involve a magnocellular deficit, then participants without dyslexia should perceive the moving object to be further ahead of the flashed object than should those with dyslexia. Additionally, we designed a second FLE stimulus to test the hypothesis that making the moving stimulus equiluminant with its background would reduce the FLE. In addition to using equiluminant background and moving stimuli, we added chromatic noise to the background in order to increase parvocellular but not magnocellular stimulation. We predicted that this would decrease the magnitude of the FLE illusion in participants with and without dyslexia.

Method

Participants

Participants were recruited primarily from the student population of the University of Alaska Anchorage (UAA). Additionally, a special effort was made to recruit participants with dyslexia. This was accomplished by snowball sampling and by posters in the UAA Disability Support Services office. Twenty-four percent of our sample reported that they had dyslexia, suggesting that our recruitment effort was successful.

Participants viewed a brief description of dyslexia (see Snowling, Dawes, Nash, & Hulme, 2012) before responding to the question, “Do you think you have dyslexia?” We categorized participants who responded “yes” or “maybe” as dyslexic. This brief self-

report method has been shown to differentiate effectively between those with high and low literacy and to correlate with scores on the Adult Reading Questionnaire (Snowling et al., 2012). Using this system, 45 participants (18 men and 25 women, average age 27.87) were categorized as being dyslexic, and 143 (74 men and 67 women, average age of 23.03) were considered normal readers. Four participants did not specify a gender.

Stimulus materials

The FLE illusion that participants viewed consisted of a small green square that moved in a rectangular motion path on a larger square background. The participants were asked to fixate on a dot in the middle of the stimulus while the green square completed two motion paths. When the square was near the end of the second motion path, a white square flashed directly above the green square. The flashed object was present for 1 frame, and the animation moved at 36 frames per second. On a typical 19-inch monitor, the movement path was 728.8 mm long and the green square moved at a speed of 74.15 mm per second.

Two versions of this FLE animation were created to stimulate the magnocellular and parvocellular pathways. The magnocellular pathway was targeted by creating high contrast between the green moving object and the background, which was accomplished by making the background black. The parvocellular pathway was targeted by reducing brightness contrast between the moving object and the background. This was accomplished by making the background equiluminant, but different in hue, from the moving stimulus. Chromatic noise was added to the background by creating a two-tone bitmap, presented in Figure 1.

Following each animation, participants viewed a screen containing seven images corresponding to various amounts of projection or lag. They selected the option that best represented their perception of where the moving square was when the flashed stimulus was presented. One option represented the actual moving square position, three options represented projection of moving square, and three options represented lag. This response screen is depicted in Figure 2.

Figure 1: FLE stimuli designed to stimulate the parvocellular (left) and magnocellular (right) visual pathways.

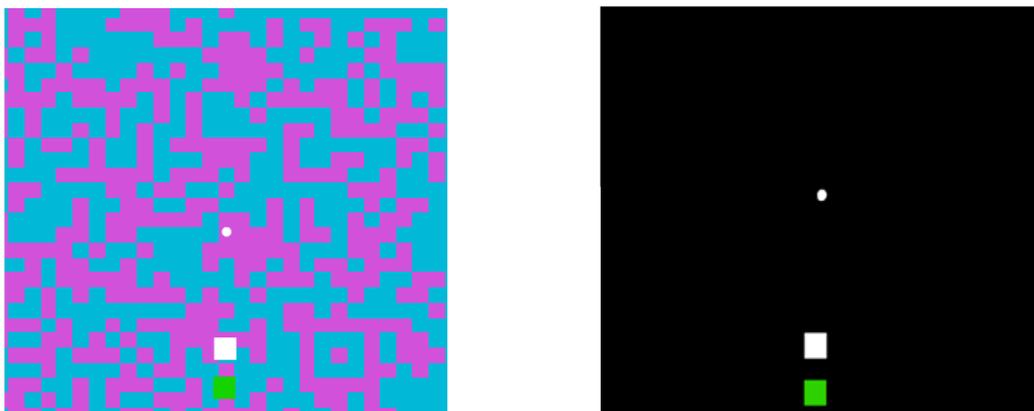
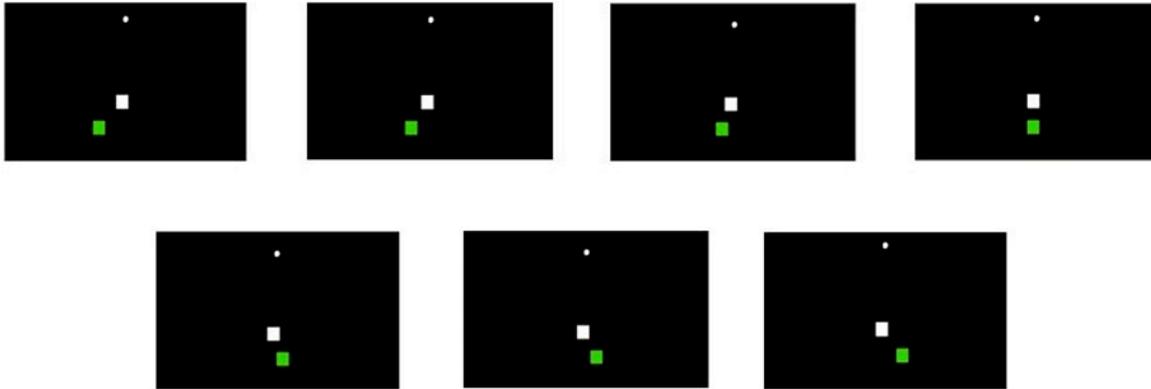


Figure 2: Screen image used to collect participant responses.



Please select the image that shows where the green square was when the white square flashed.

Procedure

In order to maximize our sample size, we used Qualtrics to make our study available online. Most students completed the experiment in an on-campus computer laboratory, but some completed it from their homes. All participants consented to the test by responding to an online consent form before viewing 2 FLE illusions. One of these tests was designed to facilitate magnocellular processing by maximizing brightness contrast between the moving item and the background, whereas the other FLE test was designed to minimize magnocellular processing by reducing this contrast. The two types of FLE illusions were presented in counterbalanced order. Once the participants completed the computerized test, they viewed a debriefing statement and received their incentive: a Red Bull, a candy bar, and/or extra credit in a Psychology course at UAA.

Results

Participants' responses were coded such that if they selected the image that was exactly the same as the presented stimulus, the response was coded as a 0. The responses that corresponded with projection and lag were coded 1 to 3 and -1 to -3, respectively, depending on the level of projection or lag displayed in the response. In the magnocellular stimulation condition, participants with dyslexia reported the moving stimulus to be slightly further ahead of the flashed object on average $M = 1.21$, 95% CI [0.68, 1.74] than did non-dyslexic participants $M = 0.80$, 95% CI [0.50, 1.09]. In the parvocellular stimulation condition, participants with and without dyslexia reported perceiving less projection of the moving square relative to the flashed object. Participants with dyslexia reported very similar perceptions of the moving object on average $M = 0.51$, 95% CI [-0.06, 1.08] to what was reported by participants without dyslexia $M = 0.54$, 95% CI [0.22, 0.88].

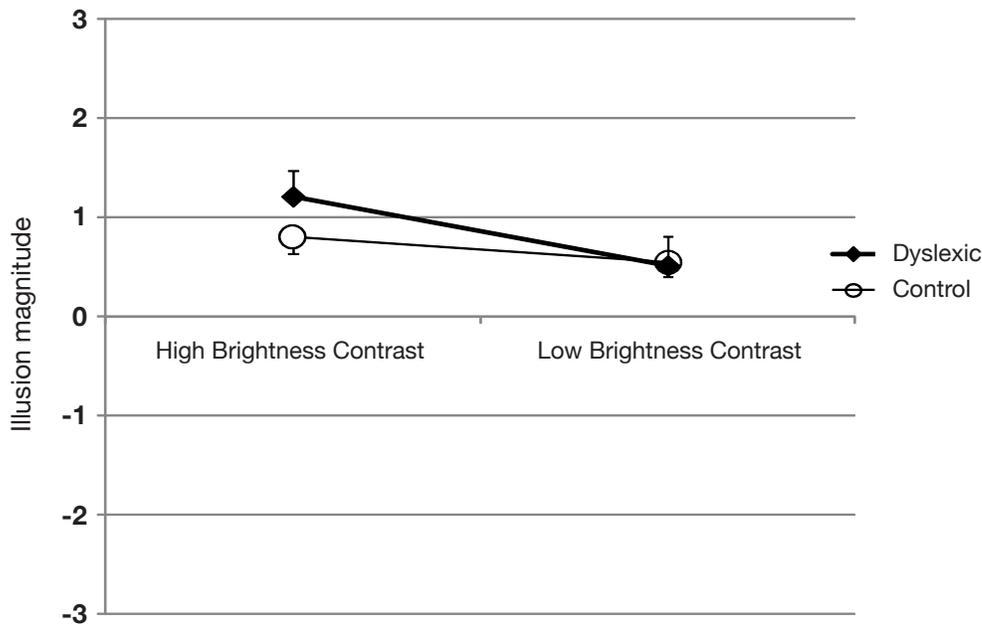


Figure 3: Average extent to which participants with and without dyslexia projected the moving stimulus ahead of the flash in two FLE tests. Error bars represent standard error.

Participants' responses are presented in Figure 3 and were analyzed with a 2 (dyslexic, yes or no) x 2 (magnocellular or parvocellular stimulation) repeated measures ANOVA. The stimulus manipulation had a significant effect $F(1, 174)=4.82, p=.03$, partial $\eta^2 = .027$; participants reported the moving object to be further along its path relative to the flashed object in the magnocellular condition than in the parvocellular condition. There was no significant effect of dyslexia $F(1, 174)=.65, p=.42$, partial $\eta^2 = .006$. There was also no interaction between factors $F(1, 174)= 1.09, p = .30$, partial $\eta^2 = .006$.

Discussion

Our results with the equiluminant stimuli differ slightly from those obtained by Chappell, Hines, & Hardwick (2002). While they did not find reliable significant decreases in FLE magnitude, we found a small but statistically significant decrease. This result may stem from either our larger sample size or from our addition of chromatic noise to our background. Either way, this result indicates that minimizing brightness contrast and adding chromatic background noise reduces magnocellular processing.

The current results obtained using stimuli incorporating luminance contrast are consistent with previous findings (Chappell & Mullen, 2010; Nijhawan, 2001); participants projected the moving stimulus ahead of its actual location. These results, however, are not supportive of the theory that a deficit in magnocellular processing contributes to dyslexia. If people with dyslexia had a consistent deficit in the magnocellular pathway, it is reasonable to suspect that they would perceive less projection of the moving object than would participants without dyslexia. In fact, when tested with the standard FLE stimulus, participants with dyslexia projected the moving stimulus further ahead of the flashed object

on average than control participants, although this difference was not statistically significant.

A determination of whether any magnocellular deficit underlies dyslexia is important because several recent experimental treatments have been based on targeting the magnocellular pathway. One example of such a treatment comes from Choake and colleagues (2012), who trained individuals with dyslexia for five days using a motion-detection task (magnocellular stimulation) or a parallel line detection task (parvocellular stimulation). Participants in both training groups improved their speed on a subsequent lexical decision test, but neither group improved their accuracy at recognizing words (in fact, a small decrease in accuracy for both groups was observed). However, only individuals in the magnocellular training group had final accuracy scores which correlated positively with increased speed from the first to the fifth and final motion-detection training sessions; this was reported as evidence that magnocellular training can improve the ability to recognize words--even though no improvement from test 1 to test 2 was observed.

Another controversial treatment for dyslexia involves the use of colored lenses or transparencies placed over pages of text. Ray, Fowler, and Stein (2005) argued that short wavelength (blue) cones inhibit magnocellular neurons; therefore, yellow transparencies should decrease this inhibition and improve reading performance. Additionally, colored lenses and overlays are sometimes used to treat a subtype of dyslexia called "Irlen syndrome," which has been described as oversensitivity to specific wavelengths of light (Kruk, Sumbler, & Willows, 2008).

Although reading improvement has been reported from the use of this treatment in some studies, many others have failed to find any benefit. For example, children diagnosed with Irlen syndrome performed similarly on the Wilkins Rate of Reading Test (WRRT) when using no colored overlay, an overlay made with their prescribed color, or an overlay made with the color complimentary to their prescribed color (Ritchie, Sala, & McIntosh, 2011). Importantly, the children were blind to what color would supposedly assist their reading. The only children showing improved reading ability when reading through their prescribed color of overlays were those familiar with what color they had been prescribed, suggesting a placebo effect. In another recent investigation, colored overlays did produce faster reading on the WRRT in readers with and without dyslexia. However, neither group of readers exhibited improvements in reading comprehension when using the overlays (Henderson, Tsogka, & Snowling, 2013).

In 2009, a Joint Statement from the American Academy of Pediatrics declared that "Currently, there is no adequate scientific evidence to support the view that subtle eye or visual problems cause learning disabilities... the evidence does not support the concept that vision therapy or tinted lenses or filters are effective, directly or indirectly, in the treatment of learning disabilities" (p. 842). On the other hand, more conventional treatments of reading difficulties are effective. For example, simply practicing reading, letter identification, and phoneme awareness led to reading improvement of approximately .27 standard score points per hour (Hatcher et al., 2006). Similarly, a computerized treatment including of phoneme knowledge and association with graphemes, practice dividing words into syllables, and familiarity with "loan words" (i.e., words coming from another language) raised the reading level of children with dyslexia to near that of non-dyslexic readers (Tijms, 2011).

The search for physiological differences between readers with and without dyslexia is an important one which may ultimately lead to extremely effective reading therapies. The results of the current experiment certainly do not rule out the possibility that some visual processing irregularities can contribute to the reading difficulties that characterize

developmental dyslexia. However, unless more conclusive support of the magnocellular deficit theory of dyslexia emerges, those with dyslexia may be well-advised to rely on established reading enhancement strategies over those that target poorly understood and inconsistently supported physiological aspects of dyslexia.

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