

Increasing visual timing by movement discrimination exercises improves reading fluency, attention span, and memory retention in dyslexics

Teri Lawton*

Cognitive Neuroscience, Perception Dynamics Institute, Encinitas, CA 92023, USA

Abstract

Background: There is an ongoing debate about whether the cause of dyslexia (reading problems) is based on linguistic, auditory, or visual timing deficits.

Objectives: This review of the relevant research provides substantial evidence that visual movement-discrimination can be used to detect and remediate reading problems in all types of dyslexia. What emerges from multiple studies is the essential role for dorsal stream function in facilitating reading fluency, selective and sustained attention, and working memory in both dyslexic and typically developing students between the ages of 6-8 years old.

Methods: Specifically, training visual dorsal stream function on discriminating the direction of a dim test pattern (< 2% contrast) moving relative to a stationary textured background pattern is the key to facilitate reading acquisition in dyslexics and those at risk for reading problems.

Results: Visual movement-discrimination training improves reading, attention, and working memory through the hypothetical increase in temporal precision and neuronal sensitivity of magnocellular neurons relative to linked parvocellular neurons in the dorsal stream.

Conclusions: This research shows that visual movement figure/ground discrimination can be used not only to diagnose all types of dyslexia, but also to successfully remediate dyslexia. The results argue that a shift from phonologically-based to visually-based methods is indicated for the treatment of dyslexia.

Introduction

Good reading ability is required for academic success. There is no greater educational problem than students who struggle to read. Estimates on the prevalence of reading problems vary from 10% [1] up to 80% [2], often coupled with diagnoses of dyslexia or reading below proficiency. Students with dyslexia and other reading difficulties have problems in their ability to read that are disproportionate to their achievements in other academic areas. Reading difficulties are prevalent in the United States (U.S.) where 32 million students, 64% of students in grades 4-12 according to NCES (2013), do not read proficiently.

Reading is a composite skill that requires us to not only interpret the information that is coming in as we read each word, but also analyze and integrate information from different brain pathways in different areas of the brain to think about different ideas. Our brain must be capable of detecting and localizing the rapidly changing incoming patterns, e.g. the beginning and end of each word (this requires motion-sensitive magno cells) before performing detailed discrimination on those localized patterns to detect the letters in a word (this requires pattern-sensitive parvo cells). Incoming information must be discriminated and interpreted correctly by pattern-sensitive cells for both comprehension and higher-level language-based processing, as well as to guide subsequent processing in different areas of the brain. The more attention and resources expended to discriminate information at lower levels of processing, the fewer resources that are available for processing information at higher levels of processing, causing attention and cognitive deficits. Slow and inefficient word identification creates a bottleneck that diverts cognitive resources required for comprehension [3,4].

What is the underlying problem in dyslexia?

Dyslexia is a multifaceted reading disability [5,6], encompassing both visual processing-based and pronunciation-based reading issues, that is characterized by severe spelling and reading problems [7]. There is an ongoing debate about whether the cause of reading problems is based on visual timing, auditory timing, or linguistic deficits. The biological basis of dyslexia (reading difficulties) was for many years assumed to be in the brain regions responsible for the visual perception of text [8]. This theory has largely been replaced by other theories that propose reading problems results from problems in pronunciation or hearing the correct sounds, being caused by an auditory phonological processing deficit [9-18]. Many poor readers do demonstrate phonological problems, i.e. acquiring the skills of separating word sounds into separate phonemes to match with the letters that represent the syllables. The essential component of reading is decoding, translating letters into the sounds they stand for. However, the phonological theory does not explain why these children fail to learn to decode [19].

The phonological theory is set at too high a cognitive level to explain the cause of reading deficits [20]. Moreover, phonological deficits only

***Correspondence to:** Teri Lawton, Cognitive Neuroscience, Perception Dynamics Institute, P.O. Box 231305, Encinitas, CA 92023, USA, Tel: 310-903-6009; E-mail: tlawton@pathtoreading.com

Key words: Cortical plasticity, dyslexia, diagnosis, therapy, visual timing, perceptual learning, reading fluency, attention, working memory

Received: April 18, 2019; **Accepted:** May 20, 2019; **Published:** May 31, 2019

predict approximately 25% of future reading skills [21,22], and are not able to explain the full range of deficits found in struggling readers. Since there are a significant number of dyslexics who do not exhibit any phonological problems at all, a phonological deficit is not a necessary condition for dyslexia [23,24]. The phonological theory ignores the many children with dyslexia who complain of visual problems, in their difficulty to see printed words correctly [5,25-27]. Furthermore, word reading skills did not differ for those who completed an auditory intervention two years earlier, when compared with controls who had no auditory intervention [28], showing that improvements in phonological processing degrade over time.

A careful examination of the neuroimaging studies responsible for this paradigm shift from visual processing deficits to auditory processing deficits reveals that visual word form areas and other visual processing areas were also implicated in many of these studies. For instance, Shaywitz et al. [1] states that "Brain activation patterns differed significantly between the groups with dyslexic readers showing relative underactivation in posterior regions (Wernicke's area, the angular gyrus, and striate cortex) and relative overactivation in an anterior region (inferior frontal gyrus)". The finding that the visual (striate cortex) processing area is underactive in persons with dyslexia is supported by other studies [1,17,29-33] and reliably co-occurs with abnormal patterns of cortical activity in areas typically associated with auditory analyses. Therefore, these neuroimaging studies provide no evidence that dyslexia is caused by auditory processing deficits as opposed to visual processing deficits.

Dyslexia is characterized by slow reading caused by spatial and temporal sequencing deficits

Dyslexics typically have slow reading speeds [34,35]. Children with dyslexia are reported to have some combination of: 1) **temporal** [9,16,17,36], and/or 2) **spatial** [5,6,20,37-46] sequencing deficits. These temporal and spatial sequencing deficits are prevalent in patient reports that words on the page appear distorted, displaced, or crowded together [47], often resulting in headaches and eyestrain [48]. Children with dyslexia are slower at recognizing individual letters, and slower at sequencing them correctly [49]. Successful sequencing during reading depends on the accurate timing of auditory and visual sensory inputs. This timing is also known as transient or temporal processing.

Temporal and spatial sequencing deficits, found when subjects are shown images that are rapidly presented or moving, have been hypothesized to result from neural timing deficits associated with sluggish magnocellular neurons [5,6,31,40-46,49-55]. These studies suggest that the visual timing deficits cause impairments in the integration of information between magnocellular ('where') and parvocellular ('what') neurons. A normally functioning magnocellular pathway is sensitive to *low-contrast* achromatic patterns [56,57]. All dyslexics exhibit high contrast thresholds for discriminating the direction of moving patterns relative to a stationary textured background pattern [40-45,51,58], suggesting that visual magnocellular deficits may explain the timing deficits found in dyslexia.

The role of magnocellular deficits in dyslexia

The visual system has been hypothesized to exploit the dichotomy of a fast-magnocellular channel and a slower parvocellular channel for the purpose of selective attention [52,59]. Receiving predominantly magnocellular input [60-62], the major dorsal stream pathway, specialized for processing the location and movement of objects in space [60,63,64], projects from the primary visual cortex, V1, through

visual area MT (middle temporal cortex) to the medial superior temporal area (MST) [65], as well as projecting to V3, V3A and V6 [66] and into the intraparietal sulcus of the posterior parietal cortex (PPC), a selective spatial attention area [67] that is also used to analyze event timing [68]. The PPC provides the input to the dorsal lateral prefrontal cortex (DLPFC), where working memory is encoded, the predominant cortical areas involved in the Executive Control Network [69]. This is in contrast to the ventral stream which receives both magnocellular and parvocellular inputs as it projects from V1 through area V4 and on to the infero-temporal (IT) cortex, an area specialized in extracting the details related to an object's color and shape [60,63,64]. The faster transmission time of the magnocellular neurons projecting predominantly to the dorsal stream is gated via attentional feedback to the striate cortex [52], which can then be used by parvocellular neurons in the ventral stream as a starting point for deciphering the individual letters in a word [44,45,52-54,59]. Moreover, feedback in the dorsal stream from MT to V1 improves figure/ground discrimination [70], a task used when reading by discriminating the letters in the word from the remaining text. Furthermore, feedback from MT has its strongest effects for low salience stimuli [70], such as low contrast patterns having less than 10% contrast, i.e. those patterns that maximally activate magnocellular neurons [56,57].

Dyslexics have magnocellular responses that were found to be 20-40 ms slower than typically developing observers [31], being 2-4-fold slower than the normal magnocellular lead time of 10-20 ms [71,72]. Some investigators hypothesize that in dyslexics a lack of synchronization in timing between magnocellular and parvocellular activations may prevent effective sequential processing, pattern analysis, and figure/ground discrimination, and hence impede the development of efficient reading and attention skills [5,6,40-44,52-54,73]. Our working hypothesis [40,45] is that magnocellular neurons in the dorsal cortical visual pathway (V1-MT) of dyslexics are sluggish, causing visual timing deficits at lower levels of visual processing [31,74] that disrupt processing at higher levels of dorsal stream processing, as shown by dyslexics having little or no activity in MT [29,30], including the development of these visual and attention pathways. These visual timing deficits limit reading acquisition in dyslexics.

Convergent evidence finds that many dyslexic readers demonstrate impairments in movement discrimination tasks that rely upon magnocellular functioning. Dyslexics were found to have motion perception deficits at each of these levels of processing in the dorsal stream:

1. the retinal level when measured using the frequency doubling illusion [75-79],
2. the Lateral Geniculate Nucleus (LGN) where the magnocellular layers were found to be 30% smaller and more disorganized [31],
3. V1 measured using VEPs [31,33,74,80],
4. V1 and MT using both fMRI brain imaging [29,30] and MEG brain imaging [81], and psychophysical tasks of movement discrimination relative to a stationary background [40-45,51],
5. MT using motion coherence for direction discrimination [37-39,50,82,83],
6. the Lateral Intraparietal cortex (LIP) and Frontal Eye Fields (FEF), anterior cortical areas activated by saccades, based on saccade and anti-saccade training tasks [84], causing text to appear to move, a symptom that many dyslexics report [26,27], and

7. parietal structures, prefrontal language systems, cerebellum, basal ganglia [35], and hubs of the attention networks [81].

These results suggest a strong relationship between dorsal stream processing and reading ability, such that poor dorsal stream processing caused by sluggish magno (motion) cells is associated with slower timing and poor reading skills [5,6,19,20,41-45,51-53,77,82,85]. In fact, motion sensitivity in individuals predicts orthographic reading skills in good and poor readers [86,87]. Dyslexics have sluggish motion cells that do not properly signal the pattern-sensitive cells, causing difficulty in isolating and identifying the critical elements needed for reading, such as the beginning and end of the word before sequentially analyzing the letters in the word. Dyslexics thereby lack the ability to process sequential information quickly and accurately, causing deficits in both reading speed and comprehension.

The claim that there is no evidence for visual deficits leading to dyslexia [88] ignores the multiple lines of converging evidence pointing to poor visual dorsal stream functioning being associated with poor reading skills, as shown by previous reviews [35,89,90]. Earlier studies [41-45,51] demonstrate that when a movement figure/ground discrimination assessment is used, *poor reading skills* are always associated with poor visual dorsal stream functioning for all types of dyslexics. Moreover, reading skills can be remediated rapidly by training that is designed to improve dorsal stream function.

Background frame of reference required to reveal movement discrimination deficits in all dyslexics

The key stimulus attribute needed to detect motion discrimination deficits are direction discrimination thresholds obtained by measuring the contrast sensitivity for the direction of motion relative to a stationary textured background [91]. Only when the direction of motion is discriminated against a stationary textured background frame of reference do both dysphonetic and dyseidetic dyslexics, those dyslexics with pronunciation and/or spelling problems, exhibit a significantly impaired ability to discriminate the direction of motion [40-45,51,58,73], whereas when movement discrimination was done against no background, only dysphonetic dyslexics [92] had movement discrimination deficits. Since direction-discrimination employs inhibitory circuits [93], this indicates that dyslexics have a developmental deficit in the functioning of their inhibitory circuits, which is supported by their impulsive behavior. Patterned backgrounds, as opposed to featureless backgrounds, require figure/ground discrimination, suggesting that a core deficit in dyslexics may be difficulty in discriminating the movement of an object relative to its background, this task being analyzed within the dorsal stream. Movement figure/ground discrimination deficits are consistent with the dyslexic's deficits: 1) being primarily due to deficits in the spatiotemporal parsing of the letter stream [52-54,59], normally transmitted both by feedforward magnocellular (low-contrast movement) input, and from feedback at the attended location from lateral inferior-parietal (LIP) to middle temporal (MT) [85], and from MT to V1 (primary visual cortex) [70], and 2) in excluding noisy backgrounds [94,95].

Previous results [44,45,51,96,97] support the hypothesis that multifrequency backgrounds confer an advantage when discriminating the direction of motion, by providing a wider, more structured background frame of reference. Even though the dorsal stream consists of predominantly magnocellular neurons, there is input to the dorsal stream from parvocellular neurons [61,98-100] from the LGN, V1, and V4, all projecting to MT, enabling parvocellular activity to provide a background frame of reference for discriminating the direction of

movement in the dorsal stream. Movement discrimination relative to a stationary background takes advantage of MT's center-surround organization [101] to facilitate figure/ground discrimination, enabling the person with dyslexia to: improve their reading fluency and processing speed, attend to wider regions of space, have better cognitive flexibility, and remember more easily. This stationary background frame of reference makes motion discrimination easier and reveals that all dyslexics have a magnocellular deficit [40-45,51,58,73].

Training to improve movement discrimination improves reading, attention and memory

The patented [40,73] PATH (Perception Attention Therapy) to Reading (PATH) training, pathreading.com, employs movement direction-discrimination to measure the contrast that is needed for figure/ground discrimination of sinewave gratings (dim gray stripes less than 2% contrast) that move left or right relative to a stationary background [45]. These backgrounds increase the task's complexity by increasing the number of background spatial frequencies from 1 to 3, providing a wider background frame of reference by recruiting additional spatial frequency channels, the background contrast from 5% to 10% to 20%, thereby activating more parvocellular neurons, with left-right movement increasing in speed after each four complexity levels, from 6.7 Hz to 8 Hz to 10 Hz to 13.3 Hz, as the training progresses. Movement could not be discriminated at faster speeds until the person was trained to discriminate left-right movement at the slower speeds, which is why PATH training progresses from slower to faster speeds of movement. These movement-discrimination training patterns, vertical sinewave gratings, are designed to differentially activate motion-sensitive (magnocellular) neurons in the V1-MT network [63,70,100-102] relative to pattern-sensitive (parvocellular) neurons, thereby being an effective training stimulus to improve magno-parvo integration timing deficits at both early and higher levels of visual motion processing.

Since motion coherence only activates the motion-sensitive neurons at MT and at higher processing levels [103,104], direction discrimination using motion coherence, instead of the vertical sinewave gratings that are used for PATH training, has not been found to be an effective training paradigm [105]. Studies that have questioned the hypothesis that dyslexics have magnocellular deficits [106-108] examined a dyslexic's sensitivity to stimuli that are not optimal for activating direction-selective cells in the V1-MT network [102,109], using either flicker or high contrast random dot patterns against *no* background pattern.

Visual timing deficits were detected and remediated for all types of dyslexics when PATH movement-discrimination training was done for only 15-20 minutes 2-3 times a week for 12 weeks. This training also significantly improved reading fluency, processing speed, attention, and working memory, which are all high-level cognitive functions [41-45,51]. Reading speed has been shown to correlate with comprehension, grade level, spelling ability, and a host of other reading skills. PATH neurotraining improved working memory and attention by improving dorsal stream function at both low and high levels of processing [51]. Based on the data from a previously published study [51] obtained from 21 participants undergoing PATH neurotraining, an effect size (Cohen's *d*) of 0.9, 1.2, 0.8, 1.0, 1.1, and 1.1 was found for reading speed, reading comprehension, pronunciation, attention, visual working memory, and auditory working memory, respectively, by examining the interaction term between Training and Time. These effect sizes are substantially larger than reported in a meta-analysis examining methods to improve

reading skills in dyslexics [110]. After a short amount of PATH movement-discrimination training was completed by dyslexic fourth graders, three times/week for 6 weeks, their dorsal stream activity improved significantly when measured by Visual Evoked Potentials [33]. These results are consistent with a recent pilot study using magnetoencephalography (MEG) source imaging [81] that found improved functioning in both the dorsal stream (V1, V3, MT, MST areas) and fronto-parietal attention networks (ACC, precuneus/PCC, DLPFC) following 8 weeks of movement-discrimination training for 10-15 minutes twice a week for an adult who was a 29 year-old dyslexic.

These improvements in cognitive skills found for dyslexics following PATH movement-discrimination training were not found for computer-based repeated reading using Raz-Kids [51]. When compared to PATH neurotraining, repeated reading interventions do less to improve reading fluency, as supported by finding: 1) reading speeds improved only 2-fold following repeated reading exercises [4], instead of from 3-fold to 10-fold, when reading aloud with a student following PATH neurotraining [44], and 2) improvements in comprehension using repeated reading interventions are much lower than found using PATH neurotraining. For example, Vadasy & Sanders [4] found that repeated reading aloud improved comprehension 8%, assessed by the Gray Oral Reading Test (GORT), whereas PATH neurotraining improved comprehension, when assessed by the GORT, 28% for dyslexic students and 37% for typically-developing students [51], even though each group trained for half as much time as done by Vadasy & Sanders [4]. The improvements in cognitive skills following PATH training were also found in typically developing children [51] who were in second and third grade (6 to 8 years old), which is the age when the temporal lobe shows peak synaptogenesis [111]. PATH movement-discrimination training was also found to significantly improve reading fluency in typically developing 6 to 8 year-old children previously [40,42,43,51]. Since timing deficits can be reduced following training using PATH movement-discrimination brain exercises [45,51], this supports the hypothesis that magnocellular pathways provide the gateway for attentive processing [52,59] and reading.

The movement direction-discrimination intervention (PATH neurotraining) is believed to change the timing of neural responses to accelerate [40,44,45,73] via intensive training of the dorsal stream, improving magnocellular relative to parvocellular activity, thereby improving inhibitory and excitatory circuits, based on the data from neural plasticity. This theory is based on the idea that the synchronous firing of neurons is what controls communication in the brain between different areas [112]. If neurons in one area are "sluggish" with respect to neurons in another area, then they will be unable to synchronize, since processing speed will be slowed down, and therefore communication, and hence learning, will be compromised. By extensive training on movement-discrimination, we hypothesize that we are improving the attention, executive control, and reading networks. Visually based movement discrimination exercises in both normal subjects [40,42,43,51,96,97,113] and dyslexics [40-45,51] have demonstrated neuroplasticity in the domain of processing speed using massed practice. These studies found that the more movement discrimination was practiced, the more contrast sensitivity for movement-discrimination, reading, attention, and memory skills improved, with gains in speed, accuracy, comprehension, attention, and working memory being measured using age-appropriate standardized tests for these cognitive skills. Not only was PATH training more effective, but it required less than half the training time used by other reading interventions.

Improving magnocellular function improves feedback from high cortical areas to lower levels

When reading, it has been proposed that the PPC uses the spatial information of the location and overall shape and form of a word that is received through the rapid magnocellular pathway to gate the information that is going into the temporal stream [52]. The information is gated via attentional feedback to the striate cortex and to other regions in the occipito-temporal cortex [52,59,114,115,116] most likely done by top-down feedback which uses synchronized neuronal oscillations at the lower end of the gamma frequency range [59], which in turn is used by the parvocellular neurons in the ventral stream, using coupled alpha/gamma oscillations regulated by the pulvinar for sequential processing [117], as a starting point for deciphering the individual letters [52,59]. In fact, the visual word form area (VWFA) in the ventral stream, where the visual shapes of words are analyzed in detail [10] receive significant magnocellular input from the dorsal stream to direct the VWFA's attention to which word it should analyze next [59,118]. It is likely that the dyslexic reader's deficit in attentional focus [52,53,119,120] is a consequence of sluggish magnocellular neurons that prevents the linked parvocellular neurons from being able to isolate and sequentially process the relevant information that is needed for reading [49,52,53], and not from an information overload as was proposed previously [121].

Each cycle of gamma oscillation focuses an attentional spotlight on the primary visual cortical representation of just one or two letters, before sequential recognition of those letters and their concatenation into word strings [59]. The timing, period, envelope, amplitude, and phase of the synchronized oscillations that is modulating the incoming signals to the striate cortex have a profound influence on the accuracy and the speed of reading [59]. The speed determined by the gamma frequency oscillation is the essential rate-limiting step in dyslexia [59]. Movement figure/ground discrimination training is likely to strengthen coupled: 1) theta/gamma activity for the test patterns moving at 6.7 and 8 Hz, and 2) alpha/gamma activity for the test patterns moving at 10 and 13.3 Hz, the patterns that improved in contrast sensitivity the most after doing PATH neurotraining [44,45,51]. Since the highest contrast sensitivities were found for patterns moving from 10-13 Hz, unable to be discriminated before training at slower speeds, these results contradict Goswami's [122] temporal sampling framework theory that states the key timing deficits in dyslexia are for movement less than 10 Hz. These results indicate that the visual movement-discrimination training paradigm improved not only magnocellular function and attention, but also improved magno-parvo integration, figure/ground discrimination, and coupled theta/gamma and alpha/gamma frequency oscillations.

These studies [45,51] found that improving visual motion direction-discrimination sensitivity and timing (low-levels in the dorsal stream) significantly improved processing in the neural networks at high levels of cognitive processing, those that mediate attention, reading, and working memory (executive control) in both typically-developing and dyslexic students. MEG brain imaging studies [81,123] indicate that these improvements were found by improving the V1-MT network (low levels in the dorsal stream), which improved functioning at higher levels in the dorsal stream, including the PPC, the DLPFC, and the attention networks. Improving the function of the PPC is consistent with the behavioral improvements found after PATH neurotraining in: 1) attention, since selective endogenous attention is encoded in the PPC [67,124,125], and 2) working memory, since the PPC projects to the DLPFC, where working memory is encoded [69]. Moreover, the control

of spatial attention in early visual cortex is directed by regions of the PPC [114-116], as well as the DLPFC [126,127]. Furthermore, since both phonological processing and auditory working memory were shown to improve following visual movement-discrimination training, these improvements demonstrate that visual movement-discrimination training also improves auditory skills, providing more evidence that PATH neurotraining improves the PPC, where there is a convergence of both auditory and visual inputs [128]. By improving attention, students were able to hear the sequential ordering of sounds more accurately, improving phonological processing and auditory working memory [45].

Reading skills improved more by increasing visual timing than by increasing auditory timing

Improving visual timing was shown to improve phonological deficits and auditory working memory more than auditory timing interventions like FastForWord [45]. FastForWord training lengthens individual phonemes so that phonological processing improves, with the length of the phonemes being decreased as the training progresses. PATH training, on the other hand, measures the contrast needed for visual movement direction-discrimination. Only visual movement-discrimination training when it was compared to phonological training, either by improving auditory timing (FastForWord) or word building strategies (Learning Upgrade), significantly improved both low and high level cognitive functions [45]: 1) motion direction sensitivity, 2) speed of processing for both motion direction-discrimination and reading rates, 3) reading comprehension, 4) phonological processing, 5) attention, and 6) both auditory and visual working memory, including delayed recall. Moreover, when PATH neurotraining was followed by oral guided reading for 5 minutes [44], reading speeds increased by as much as 11-fold, and on average were over three times faster than found previously, providing more evidence that PATH neurotraining improves PPC functioning. These results indicate that movement-discrimination training improves the sensitivity and timing of sluggish magnocellular neurons (improving dorsal stream function), relative to parvocellular neurons early in the dorsal stream, as evidenced by improved movement-discrimination contrast sensitivity at higher background contrasts and temporal frequencies following movement-discrimination training.

Studies using movement figure/ground discrimination (PATH neurotraining) provide additional evidence that *visual* motion processing is fundamental for paying attention, good reading performance, and remediating reading deficits, improving high level cognitive processes, which is contrary to common practice based on the assumption that only auditory-based phonological processing can be used to remediate reading deficits [7,10,11,12,15-17]. Students given training aimed at improving the auditory timing of magno cells, as embodied by the FastForWord program, improved in reading fluency, but the improvements were not significant when they were compared to the improvements made by controls [45], as also found in a review of FastForWord studies [129].

Moreover, the more students improved on movement direction-discrimination, the more they improved on higher level cognitive skills, especially in reading speed and comprehension [42,44]. Remediating visual timing deficits in the dorsal stream improved reading and attention, which suggests a causal role of visual movement-discrimination training and attention in reading acquisition. These studies: 1) support the hypothesis that faulty timing in synchronizing the activity of magnocellular with parvocellular visual pathways in the dorsal stream is a fundamental cause of dyslexia, 2) argue against

the assumption that reading deficiencies in dyslexia are caused by phonological or language deficits, and 3) demonstrate that visual movement discrimination is not only a correlate of dyslexia for children and typically-developing students at-risk for reading problems, but it is also a successful treatment. Therefore, a paradigm shift in the detection and the treatment of dyslexia from improving phonological processing to improving visual movement figure/ground discrimination is needed.

Improving visual dorsal stream function remediates reading fluency, attention span, and memory retention

The sluggish magnocellular neurons in dyslexics not only result in attention deficits, an impairment in the low gamma frequencies that reduce feedback to visual cortical areas [59], but it also disrupts processing in LIP and FEF, either within a fixation, between a fixation sequence, or both [52,84,130,131], causing very slow reading speeds. Moreover, finding that movement-discrimination training improved not only reading fluency, but also selective and sustained attention, and working memory when done before reading [45,51,81,123] indicates that movement-discrimination training helps develop the attention and executive control networks, since fewer resources are used to decode incoming information, so that more resources can be used to analyze the information, which improves visual, attention, reading, and memory skills. These results provide more evidence showing that abnormal visual motion processing is a fundamental cause of reading and attention problems in dyslexia and other cognitive slow-downs, like those caused by a concussion [123]. By improving the attention network's functioning, movement-discrimination training provides a wider window of attention, so that more objects are perceived in their correct location in a single glance [132]. Movement direction-discrimination training improves the ability to detect the synchronicity of multiple objects in space and see their trajectories over time, most likely by increasing the ease of magno-parvo integration, thereby facilitating figure/ground discrimination within a wider window of focused attention [51]. Moreover, there is evidence that improvements in reading speed after movement-discrimination training are sustained over time [44], whereas improvements in word reading found following auditory interventions to improve phonological processing degrade over time, two years later showing no difference in word reading skills when compared to controls who did not complete the auditory intervention [28].

Improving cognitive function by training left-right movement discrimination relative to a background is a novel method [40,73] that was found to be both rapid and effective in improving cognitive skills in dyslexics. Only when low-level visual timing deficits are remediated in those with dyslexia are the improvements in high-level cognitive functions, such as reading fluency (speed and comprehension), attention, and working memory improved quickly, with improvements that are sustained over time [44]. PATH neurotraining is the first visually-based intervention that was found to improve both low-level movement discrimination in the dorsal stream and high-level cognitive functioning, both behaviorally and using MEG brain imaging, improving the attention and the executive control networks. Since movement-discrimination (PATH) neurotraining is so rapid and effective, it offers a new approach that represents a paradigm shift in the treatment of dyslexia, one that is based on improving visual timing instead of improving phonological timing. When reading, students who allocate all their resources to identify the letters in the word, instead of allocating their resources to interpret a sentence, understand its meaning, and integrate the information into their existing knowledge need movement figure/ground discrimination training to remediate their visual timing deficits.

Conclusions

Visual movement figure/ground discrimination can be used to not only diagnose all types of dyslexia, but also for the successful treatment of dyslexia, so that reading, and learning can be done more automatically. What emerges from multiple studies is the essential role of dorsal stream function to facilitate reading fluency, selective and sustained attention, and working memory. Training visual dorsal stream function at low levels (V1-MT pathways) significantly improved these high-level cognitive functions, hypothesized to result from increasing the temporal precision and neuronal sensitivity of magnocellular neurons relative to linked parvocellular neurons in the dorsal stream. PATH movement-discrimination training was faster and more effective in improving reading, attention, and memory than found after training on: 1) repeated reading interventions, 2) interventions designed to improve auditory timing, or 3) linguistic-based reading interventions. Remediating visual timing deficits in the dorsal stream revealed the causal role of visual movement discrimination training to facilitate reading acquisition in dyslexics and typically developing students between the ages of 6 to 8 years old. Moreover, this research supports the hypothesis that faulty timing in synchronizing the activity of magnocellular with parvocellular visual pathways in the dorsal stream is a fundamental cause of dyslexia and argues against the assumption that reading deficiencies in dyslexia are caused by phonological or language deficits. These studies indicate that a paradigm shift in treating dyslexia from phonologically-based to visually-based methods is essential.

Acknowledgements

This research review did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors. The author has a potential conflict of interest, since she is the developer of *Path To Reading (PATH)* neurotraining, and is the Director of Research and Founder of Perception Dynamics Institute.

References

- Shaywitz SE, Shaywitz BA, Pugh KR, Fullbright RK, Constable RT, et al. (1998) Functional disruption in the organization of the brain for reading in dyslexia. *Proc Natl Acad Sci U S A* 95: 2636-2641. [Crossref]
- <http://nces.ed.gov/nationsreportcard/subject/publications/main2013/pdf/2014451.pdf>
- LaBerge D, Samuels SJ (1974) Toward a theory of automatic information processing in reading. *Cognitive Psychology* 6: 293-323.
- Vadasy PF, Sanders EA (2008) Repeated reading intervention: Outcomes and interactions with readers' skills and classroom instruction. *J Educ Psychol* 100: 272-290.
- Stein J, Walsh V (1997) To see but not to read; the magnocellular theory of dyslexia. *Trends Neurosci* 20: 147-152. [Crossref]
- Stein J (2001) The magnocellular theory of developmental dyslexia. *Dyslexia* 7: 12-36. [Crossref]
- Vellutino FR, Fletcher JM, Snowling MJ, Scanlon DM (2004) Specific reading disability (dyslexia): what have we learned in the past four decades? *J Child Psychol Psychiatry* 45: 2-40. [Crossref]
- Hinshelwood J (1917) Congenital Word-Blindness, H.K. Lewis & Co., 136 Gower St, London.
- Bradley L, Bryant PE (1983) Categorizing sounds and learning to read: A causal connection. *Nature* 301: 419-421.
- Dehaene S (2009) Reading in the Brain. Viking: Penguin Group (NY): p235-261.
- Olulade OA, Napoliello EM, Eden GF (2013) Abnormal visual motion processing is not a cause of dyslexia. *Neuron* 79: 180-190. [Crossref]
- Shaywitz, SE (2003) Overcoming Dyslexia. Alfred A Knopf, New York.
- Snowling MJ (2001) From language to reading and dyslexia. *Dyslexia* 7: 37-46. [Crossref]
- Stanovich K, Siegel LS (1994) Phenotypic performance profile of children with reading disabilities: A regression-based test of the phonological-core variable difference model. *J Educ Psychol*, 86: 24-53.
- Tallal P (1980) Auditory temporal perception, phonics, and reading disabilities in children. *Brain Lang*, 9: 182-198. [Crossref]
- Tallal P, Miller S, Fitch RH (1993) Neurobiological basis of speech: a case for the preeminence of temporal processing. *Ann N Y Acad Sci* 682: 27-47. [Crossref]
- Temple E, Deutsch GK, Poldrack RA, Miller SL, Tallal P, et al. (2003) Neural deficits in children with dyslexia ameliorated by behavioral remediation: Evidence from functional MRI. *Proc Natl Acad Sci U S A* 100: 2863-2865. [Crossref]
- Torgesen JK, Wagner RK, Rashotte CA (1994) Longitudinal studies of phonological processing, and reading. *J Learn Disabil* 27: 276-286. [Crossref]
- Stein JF (2018a) Does dyslexia exist? Language, Cognition, and Neuroscience. 33: 313-320.
- Stein JF (2018) The current status of the magnocellular theory of developmental dyslexia. *Neuropsychologia* pii: S0028-3932(18)30115-5. [Crossref]
- Mann VA, Liberman IY (1984) Phonological awareness and short-term memory *J Learn Disabil* 17: 592-599. [Crossref]
- Wagner RK, Torgesen JK, Rashotte CA, Hecht SA, Barker TA, et al. (1997). Changing relations between phonological processing abilities and word-level reading as children develop from beginning to skilled readers: A 5-year longitudinal study. *Dev Psychol* 33: 468-479. [Crossref]
- Castles A, Coltheart M (1993) Varieties of developmental dyslexia. *Cognition* 47: 149-180. [Crossref]
- Peterson RL, Pennington BF, Olson RK, Wadsworth SJ (2014) Longitudinal stability of phonological and surface subtypes of developmental dyslexia. *Sci Stud Read* 18: 347-362. [Crossref]
- Northway N (2012) Why do words jump? An exploration of visually symptomatic readers. *Br Ir Orthopt J* 9: 3-8.
- Singleton C, Trotter S (2005) Visual stress in adults with and without dyslexia. *J Res Read* 28: 365-378.
- Harries P, Hall R, Ray N, Griffiths PG, Stein J (2015) Using coloured filters to reduce the symptoms of visual stress in children with reading delay. *Scand J Occup Ther* 22: 1-8. [Crossref]
- Wise BW, Ring J, Olson RK (2000) Individual differences in gains from computer-assisted remedial reading. *J Exp Child Psychol* 77: 197-235. [Crossref]
- Demb JB, Boynton GM, Heeger DJ (1998) Functional magnetic resonance imaging of early visual pathways in dyslexia. *J Neurosci* 18: 6939-6951. [Crossref]
- Eden GF, VanMeter JW, Rumsey JM, Maisog JM, Woods RP, et al. (1996). Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. *Nature* 382: 66-69. [Crossref]
- Livingstone MS, Rosen GD, Drislane FW, Galaburda AM (1991) Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc Natl Acad Sci U S A* 88: 7943-7947. [Crossref]
- Shaywitz BA, Shaywitz SE (2011) Malleability and plasticity in the neural systems for reading and dyslexia. Neuroplasticity and Rehabilitation, Guilford Press, New York, NY, USA, Pp: 149-171.
- Shelley-Tremblay JF, Syklawer S, Ramkissoon I (2011) The effect of magnoparvocellular integration training on fluency and visual evoked potentials in poor readers. *J Behav Optim* 31-37.
- Lyon GR, Shaywitz SE, Shaywitz BA (2003) Comorbidity, teachers' knowledge of language and reading: A definition of dyslexia. *Ann Dyslexia* 53: 1-14.
- Nicholson RI, Fawcett AJ (2007) Procedural learning difficulties: Reuniting the developmental disorders? *Trends Neurosci* 30: 136-141. [Crossref]
- Stanley G, Hall R (1973) Short-term visual Information processing in dyslexics. *Child Dev* 44: 841-844. [Crossref]
- Cornelissen P, Richardson A, Mason A, Fowler S, Stein J (1995) Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls. *Vision Res* 35: 1483-1494. [Crossref]
- Talcott JB, Hansen PC, Assoku EL, Stein JF (2000) Visual motion sensitivity in dyslexia: Evidence for temporal and energy integration deficits. *Neuropsychologia* 38: 935-943. [Crossref]

39. Hansen PC, Stein JF, Orde SR, Winter JL, Talcott JB (2001) Are dyslexics' visual deficits limited to measures of dorsal stream function? *Neuroreport* 12: 1527-1530. [[Crossref](#)]
40. Lawton T (2000) Methods and Apparatus For Diagnosing and Remediating Reading Disorders. United States Patent No 6,045,515, Washington, DC: U.S. Patent and Trademark Office.
41. Lawton TB (2004) Training directionally-selective motion pathways can significantly improve reading efficiency", Human Vision and Electronic Imaging IX, Ed. B. E. Rogowitz and T.N. Pappas, Proc. of SPIE-IS&T Electronic Imaging, SPIE Vol. 5292, 34-45.
42. Lawton T (2007) Training direction-discrimination sensitivity remediates a wide spectrum of reading skills. *Optometry Vision Development* 38: 37-51.
43. Lawton T (2008) Filtered text and direction discrimination training improved reading fluency for both dyslexic and normal readers. *Optometry Vision Development* 39: 114-126.
44. Lawton T (2011) Improving magnocellular function in the dorsal stream remediates reading deficits. *Optometry Vision Development* 42: 142-154.
45. Lawton T (2016) Improving dorsal stream function in dyslexics by training figure/ground motion discrimination improves attention, reading fluency, and working memory. *Front Hum Neurosci* 10: 397. [[Crossref](#)]
46. Lovegrove WJ, Bowling A, Badcock D, Blackwood M (1980) Specific reading disability: Differences in contrast sensitivity as a function of spatial frequency. *Science* 210: 439-440. [[Crossref](#)]
47. Atkinson J (1991) Review of human visual development: crowding and dyslexia. In Stein, J.F. (Edtr). *Vision and Visual Dyslexia*, CRC Press: Boston: 44-57.
48. Wilkins AJ (1995) *Visual Stress*. Oxford: Oxford University Press.
49. Vidyasagar TR, Pammer K (2010) Dyslexia: a deficit in visuo-spatial attention, not in phonological processing. *Trends Cogn Sci* 14: 57-63. [[Crossref](#)]
50. Boets B, Vandermosten M, Cornelissen P, Wouters J, Ghesquière P (2011) Coherent motion sensitivity and reading development in the transition from prereading to reading stage. *Child Dev* 82: 854-869. [[Crossref](#)]
51. Lawton T, Shelley-Tremblay J (2017) Training on movement figure-ground discrimination remediates low-level visual timing deficits in the dorsal stream, improving high-level cognitive functioning, including attention, reading fluency, and memory. *Front Hum Neurosci* 11: 236. [[Crossref](#)]
52. Vidyasagar TR (1999) A neuronal model of attentional spotlight: parietal guiding the temporal. *Brain Res Brain Res Rev* 30: 66-76. [[Crossref](#)]
53. Vidyasagar TR (2001) From attentional gating in macaque primary visual cortex to dyslexia in humans. *Prog Brain Res* 134: 297-312. [[Crossref](#)]
54. Vidyasagar TR (2012) Aetiology of Dyslexia; A visual perspective on a phonological marker, Chapter 10. In: Stein J, Kapoula Z, eds. *Visual Aspects of Dyslexia*. Oxford University Press, 151-170.
55. Wolf M, Bowers PG, Biddle K (2000) Naming-speed processes, timing, and reading: a conceptual review. *J Learn Disabil* 33: 387-407. [[Crossref](#)]
56. Kaplan E, Shapley RM (1986) The primate retina contains two types of ganglion cells, with high- and low-contrast sensitivity. *Proc Natl Acad Sci U S A* 83: 2755-2757. [[Crossref](#)]
57. Sclar G, Maunsell JHR, Lennie P (1990) Coding of image contrast in central visual pathways of the macaque monkey. *Vision Res* 30: 1-10. [[Crossref](#)]
58. Ridder WH, Borsting E, Banton T (2001) All developmental dyslexic subtypes display an elevated motion coherence threshold. *Optom Vis Sci* 78: 510-517. [[Crossref](#)]
59. Vidyasagar TR (2013) Reading into neuronal oscillations in the visual system: implications for developmental dyslexia. *Front Hum Neurosci* 7: 1-10. [[Crossref](#)]
60. Livingstone M, Hubel D (1988) Segregation of form, color, movement, and depth: anatomy, physiology, and perception. *Science* 240: 740-749. [[Crossref](#)]
61. Maunsell JH, Nealey TA, De Priest DD (1990) Magnocellular and parvocellular contributions to responses in the middle temporal visual area (MT) of the macaque monkey. *J Neurosci* 10: 3323-3334. [[Crossref](#)]
62. Merigan WH, Maunsell JH (1993) How parallel are the primate visual pathways? *Annu Rev Neurosci* 16: 369-402. [[Crossref](#)]
63. Felleman DJ, Van Essen DC (1991) Distributed hierarchical processing in the primate cerebral cortex. *Cereb Cortex* 1: 1-47. [[Crossref](#)]
64. Ungerleider LG, Mishkin M (1982) Two cortical visual systems. In: Ingle DJ, Goodale MA, & Mansfield RJW, editors. *Analysis of Visual Behavior*, Cambridge (UK): MIT Press, p. 549-586.
65. Kolster H, Peeters R, Orban GA (2010) The retinotopic organization of the human middle temporal area MT/V5 and its cortical neighbors. *J Neurosci* 30: 9801-9820. [[Crossref](#)]
66. Pitzalis S, Sereno MI, Committeri G, Fattori P, Galati G, et al. (2010) Human v6: the medial motion area. *Cereb Cortex* 20: 411-424. [[Crossref](#)]
67. Posner MI, Walker JA, Friedrich FJ, Rafal RD (1984) Effects of parietal injury on covert orienting of attention. *J Neurosci* 4: 1863-1874. [[Crossref](#)]
68. Battelli L, Pascual-Leone A, Cavanagh P (2007) The 'when' pathway of the right parietal lobe. *Trends Cogn Sci* 11: 204-210. [[Crossref](#)]
69. Menon V, Uddin LQ (2010) Saliency, switching, attention and control: a network model of insula function. *Brain Struct Funct* 214: 655-667. [[Crossref](#)]
70. Hupe, JM, Payne AC, Lomer BR, Girard SG, Bullier J (1998) Cortical feedback improves discrimination between figure and background by V1, V2, and V3 neurons. *Nature* 394: 784-787. [[Crossref](#)]
71. Dreher B, Fukada Y, Rodieck RW (1976) Identification, classification and anatomical segregation of cells with X-like and Y-like properties in the lateral geniculate nucleus of old-world primates. *J Physiol* 258: 433-452. [[Crossref](#)]
72. Nowak LG, Munk MH, Girard P, Bullier J (1995) Visual latencies in areas V1 and V2 of the macaque monkey. *Vis Neurosci* 12: 371-384. [[Crossref](#)]
73. Lawton T (2015) Diagnosing and Remediating Cognitive Deficits Involving Attention, Sequential Processing, Reading, Speed of Processing, and Navigation. United States Patent No. 8,979,263 B2, Washington, DC: U.S. Patent and Trademark Office.
74. Lehmkuhle S, Garzia RP, Turner L, Hash T, Baro JA (1993) A defective visual pathway in children with reading disability. *N Engl J Med* 328: 989-996. [[Crossref](#)]
75. Avellis FO, Darso A, Gandolfi S, Carta A (2016) Magnocellular deficit in dyslexia: A preliminary analysis of possible patterns of visual field deficit tested with frequency doubling illusion. *Translational Biomedicine* 4: 94.
76. Buchholz J, McKone E (2004) Adults with dyslexia show deficits on spatial frequency doubling and visual attention tasks. *Dyslexia* 10: 24-43. [[Crossref](#)]
77. Gori S, Cecchini P, Bigoni A, Molteni M, Facoetti A (2014). Magnocellular-dorsal pathway and sublexical route in developmental dyslexia. *Front Hum Neurosci* 8: 1-11. [[Crossref](#)]
78. Kevan A, Pammer K (2009) Predicting early reading skills from pre-reading measures of dorsal stream functioning. *Neuropsychologia* 47: 3174-3181. [[Crossref](#)]
79. Pammer K, Wheatley C (2001) Isolating the M(y)-cell response in dyslexia using the spatial frequency doubling illusion. *Vision Res* 41: 2139-2147. [[Crossref](#)]
80. Crewther SG, Crewther DP, Klistorner A, Kiely PM (1999) Development of the magnocellular VEP in children: implications for reading disability. *Electroencephalogr Clin Neurophysiol Suppl* 49: 123-128. [[Crossref](#)]
81. Lawton T, Huang MX (2015) Improving visual timing enhances cognitive functioning by altering dorsal stream and attention networks, 12th Annual World Congress of the Society for Brain Mapping and Therapeutics, Los Angeles, CA March 7.
82. Gori S, Seitz AR, Ronconi L, Franceschini S, Facoetti A (2016) Multiple causal links between magnocellular-dorsal pathway deficit and developmental dyslexia. *Cereb Cortex* 26: 4356-4369. [[Crossref](#)]
83. Talcott JB, Hansen PC, Willis-Owen C, McKinnell IW, Richardson AJ, et al. (1998) Visual magnocellular impairment in adult developmental dyslexics. *Neuro-Ophthalmol* 60:187-201.
84. Fischer B (2012) Subitizing, dynamic vision, saccade and fixation control in dyslexia, In: Stein, J. & Kapoula, Z., editors. *Visual Aspects of Dyslexia*. Oxford University Press. p. 15-43.
85. Saalman YB, Pigarev IN, Vidyasagar TR (2007) Neural mechanisms of visual attention: how top-down feedback highlights relevant locations. *Science* 316: 1612-1615. [[Crossref](#)]
86. Talcott JB, Witton C, Hebb GS, Stoodley CJ, Westwood EA, et al. (2002) On the relationship between dynamic visual and auditory processing and literacy skills; results from a large primary-school study. *Dyslexia* 8: 204-225. [[Crossref](#)]
87. Witton C, Talcott JB, Hansen PC, Richardson AJ, Griffiths TD, et al. (1998) Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexia and normal readers. *Curr Biol* 8: 791-797. [[Crossref](#)]

88. Handler SM, Fierson WM, Section on Ophthalmology; Council on Children with Disabilities; American Academy of Ophthalmology, et al. (2009) Learning disabilities, dyslexia, and vision. *Pediatrics* 127: e818-56. [\[Crossref\]](#)
89. Boden C, Giaschi D (2007) M-stream deficits and reading-related visual processes in developmental dyslexia. *Psychological Bulletin* 133: 346-366. [\[Crossref\]](#)
90. Laycock R, Crewther SG (2008) Towards an understanding of the role of the 'magnocellular advantage' in fluent reading. *Neurosci Biobehav Rev* 32: 1494-1506. [\[Crossref\]](#)
91. Georgeson MA, Scott-Samuel NE (1999) Motion contrast: a new metric for direction discrimination. *Vision Res* 39: 4393-4402.
92. Borsting E, Ridder WH, Dudeck K, Kelley C, Matsui L, et al. (1996) The presence of a magnocellular defect depends on the type of dyslexia. *Vision Res* 36: 1047-1053. [\[Crossref\]](#)
93. Barlow HB, Levick WR (1965) The mechanism of directionally selective units in rabbit's retina. *J Physiol* 178: 477-504. [\[Crossref\]](#)
94. Sperling AJ, Lu Z, Manis FR, Seindenberg MS (2006) Motion-perception deficits and reading impairment: It's the noise, not the motion. *Psychol Sci* 17: 1047-1053. [\[Crossref\]](#)
95. Benassi M, Simonelli L, Giovagnoli S, Bolzani R (2010) Coherence motion perception in developmental dyslexia: a meta-analysis of behavioral studies. *Dyslexia* 16: 341-357. [\[Crossref\]](#)
96. Lawton T (1985) Spatial Frequency Spectrum of Patterns Changes The Visibility Of Spatial-Phase Differences. *J Opt Soc Am A* 2: 1140-1152. [\[Crossref\]](#)
97. Lawton T (1989) Outputs of paired Gabor filters summed across background frame of reference predicts direction of movement. *IEEE Trans Biomed Eng* 36: 130-139. [\[Crossref\]](#)
98. Callaway EM (1998) Local circuits in primary visual cortex of the macaque monkey. *Annu Rev Neurosci* 21: 47-74. [\[Crossref\]](#)
99. Callaway EM (2005) Structure and function of parallel pathways in the primate early visual system. *J Physiol* 566: 13-19. [\[Crossref\]](#)
100. Nassi JJ, Lyon DC, Callaway EM (2006) The parvocellular LGN provides a robust disynaptic input to the visual motion area MT. *Neuron* 50: 319-327. [\[Crossref\]](#)
101. Allman J, Miezin F, McGuinness E (1985) Stimulus specific responses from beyond the classical receptive field: Neurophysiological mechanisms for local-global comparisons in visual neurons. *Annu Rev Neurosci* 8: 407-430. [\[Crossref\]](#)
102. De Valois RL, Cottaris NP, Mahon LE, Elfar SD, Wilson JA (2000) Spatial and temporal receptive fields of geniculate and cortical cells and directional selectivity. *Vision Res* 40: 3685-36702. [\[Crossref\]](#)
103. Braddick OJ, O'Brien JM, Wattam-Bell J, Atkinson J, Hartley T, et al. (2001) Brain areas sensitive to coherent visual motion. *Perception* 30: 61-72. [\[Crossref\]](#)
104. Zohary E, Celebrini S, Britten KH, Newsome WT (1994) Neuronal plasticity that underlies improvement in perceptual performance. *Science* 263: 1289-1292. [\[Crossref\]](#)
105. Solan HA, Shelley-Tremblay J, Hansen PC, Silverman M, Larson S, et al. (2004). M-cell deficit and reading disability: a preliminary study of the effects of temporal vision-processing therapy. *Optometry* 75: 640-650. [\[Crossref\]](#)
106. Amitay S, Ben-Yehudah G, Banai K, Ahissar M (2002) Disabled readers suffer from visual and auditory impairments but not from a specific magnocellular deficit. *Brain* 125: 2272-2285. [\[Crossref\]](#)
107. Skottun BC (2000) The magnocellular deficit theory of dyslexia: the evidence from contrast sensitivity. *Vision Res* 40: 111-127. [\[Crossref\]](#)
108. Williams MJ, Stuart GW, Castles A, McAnally KI (2003) Contrast sensitivity in subgroups of developmental dyslexia. *Vision Res* 43: 467-477. [\[Crossref\]](#)
109. Baker CL (1988) Spatial and temporal frequency selectivity as a basis for velocity preference in cat striate cortex neurons. *Vis Neurosci* 4: 101-113. [\[Crossref\]](#)
110. Galuschka K, Ise E, Krick K, Schulte-Korne G (2014) Effectiveness of treatment approaches for children and adolescents with reading disabilities: A meta-analysis of randomized controlled trials. *PLOS One* 9: e105843. [\[Crossref\]](#)
111. Thatcher RW, Walker RA, Giudice S (1987) Human cerebral hemispheres develop at different rates and ages. *Science* 236: 1110-1113. [\[Crossref\]](#)
112. Buzsaki G (2006) *The Rhythms of the Brain*. Oxford University Press.
113. Lawton T (1984) The effect of phase structures on spatial phase discrimination. *Vision Research*, 24, 139-148. [\[Crossref\]](#)
114. Watanabe T, Sasaki Y, Miyauchi S, Putz B, Fujimaki N, et al. (1998) Attention-regulated activity in human primary visual cortex. *J Neurophysiol* 79: 2218-2221. [\[Crossref\]](#)
115. Martinez A, Anillo-Vento L, Sereno MI, Frank LR, Buxton RB, et al. (1999) Involvement of striate and extrastriate cortical areas in spatial attention. *Nat Neurosci*. 2: 364-369. [\[Crossref\]](#)
116. Somers DC, Dale AM, Seiffert AE, Tootell RB (1999). Functional MRI reveals spatially specific attentional modulations in human primary visual cortex. *Proc Natl Acad Sci U S A* 96: 1665-1668. [\[Crossref\]](#)
117. Saalman YB, Pinsk MA, Wang L, Li X, Kastner S (2012) The pulvinar regulates information transmission between cortical areas based on attention demands. *Science* 337: 753-756. [\[Crossref\]](#)
118. Pammer K, Hansen P, Holliday I, Cornelissen P (2006) Attentional shifting and the role of the dorsal pathway in visual word recognition. *Neuropsychologia* 44: 2926-2936. [\[Crossref\]](#)
119. Facoetti A, Zorzi M, Cestnick L, Lorusso ML, Molteni M, et al. (2006). The relationship between visuo-spatial attention and nonword reading in developmental dyslexia. *Cogn Neuropsychol* 23: 841-855. [\[Crossref\]](#)
120. Solan HA, Larson S, Shelley-Tremblay J, Ficarra A, Silverman M (2001) Role of visual attention on cognitive control of oculomotor readiness in students with reading disabilities. *J Learn Disabil* 34: 107-18. [\[Crossref\]](#)
121. Stuart GW, McAnally KI, Castles A (2001) Can contrast sensitivity functions in dyslexia be explained by inattention rather than a magnocellular deficit? *Vision Res* 41: 3205-3211. [\[Crossref\]](#)
122. Goswami U (2011) A temporal sampling framework for developmental dyslexia. *Trends Cogn Sci* 15: 3-10. [\[Crossref\]](#)
123. Lawton T, Huang MX (2019) Dynamic Cognitive Remediation for Traumatic Brain Injuries Significantly Improves Attention, Working Memory, Processing Speed, and Reading Fluency. *Restor Neurol Neurosci* 37: 71-86. [\[Crossref\]](#)
124. Posner MI, Petersen SE (1990) The attention system of the human brain. *Annu Rev Neurosci* 13: 25-42. [\[Crossref\]](#)
125. Supekar K, Menon V (2012) Developmental maturation of dynamic causal control signals in higher-order cognition: A neurocognitive network model. *PLoS Comput Biol* 8: e1002374. [\[Crossref\]](#)
126. Silver MA, Ress D, Heeger DJ (2005) Topographic maps of visual spatial attention in human parietal cortex. *J Neurophysiol* 94: 1358-1371. [\[Crossref\]](#)
127. Somers DC (2014) Attentional "Spotlight" in Early Visual Cortex, in *New Visual Neurosciences*, Werner JS & Chalupa LM, editors, MIT Press p. 1097-1106.
128. Farah MJ, Wong AB, Monheit MA, Morrow LA (1989) Parietal lobe mechanisms of spatial attention: modality-specific or supramodal? *Neuropsychologia* 27: 461-470. [\[Crossref\]](#)
129. Strong G, Torgerson CJ, Torgerson D, Hulme C (2011) A systematic meta-analytic review of evidence for the effectiveness of the 'FastForWord' language intervention program. *J Child Psychol Psychiatry* 52: 224-235. [\[Crossref\]](#)
130. Slaghuis WL, Ryan JF (1999) Spatio-temporal contrast sensitivity, coherent motion, and visible persistence in developmental dyslexia. *Vision Res* 39: 651-668. [\[Crossref\]](#)
131. Slaghuis WL, Ryan JF (2006) Directional motion contrast sensitivity in developmental dyslexia. *Vision Res* 46: 3291-3303. [\[Crossref\]](#)
132. Lawton T, Stephey D (2009) Training direction discrimination improves usable field of view, short term memory, and navigation in older adults. *Optometry Vision Development* 40: 82-93.

Copyright: ©2019 Lawton T. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.