



BrightStar

*The Science of Dyslexia and
BrightStar*

A White Paper

By the Epoch Science Team*

December 21, 2004

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Summary

This white paper outlines some of the current scientific theories of the dyslexia disorder as well as introduces some of the basic principles behind a novel technological remediation technique, BrightStar. BrightStar aims to alleviate the symptoms of dyslexia and facilitate the acquisition of improved literacy skills.

Dyslexia is a common learning disability characterized by difficulties in reading in people with seemingly normal levels of intelligence and schooling. Dyslexia is surprisingly pervasive, affecting between 30 and 45 million children and adults in the United States. Symptoms of dyslexia range from deficits in reading, writing, spelling, telling time, and/or organization, though not all problems are experienced by all with dyslexia.

While numerous explanatory theories of dyslexia exist, some theories that address specific neurological deficits in the brains of dyslexics account for many of the primary symptoms of dyslexia. Key neurological differences in the cerebellum (a brain structure critical in planning and executing movement) and in specific neural pathways (the magnocellular neurons) that carry certain types of visual information can lead to fundamental deficits in planned eye movements and the processing of visual information, two critical subcomponents of reading.

BrightStar is a computer-based technological remediation designed to improve literacy skills by targeting the key magnocellular and cerebellar deficits found in many dyslexics. It is a non-invasive, non-language based technology. BrightStar works, in part, by utilizing specifically designed and specifically timed computer graphics presented as part of a computer game. The specially designed computer graphics help retrain the dyslexics' brains in order to improve dyslexics' fundamental deficits in visual-information processing, which ultimately results in improved reading.

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What is Dyslexia?

One of the most commonly-cited definitions of dyslexia states that: “dyslexia is characterized by an unexpected difficulty in reading in children and adults who otherwise possess the intelligence, motivation, and schooling necessary for accurate and fluent reading,” (Shaywitz et al., 2001). This definition succinctly highlights the core problem of dyslexia: a great difficulty in acquiring the fluent reading skills that the vast majority of the population takes for granted. While difficulty in learning to read might be the most obvious feature of dyslexia, reading difficulties can stem from a wide array of different problems:

- **Learning Style:** May be early or late in crawling, walking or talking; appears intelligent but doesn't read, write, or spell at the level of chronological age peers; may be seen as not trying hard enough.
- **Motor Skills:** Has poor handwriting or problems with writing or copying; has poor coordination; does not do well at team sports; has difficulty with motor-oriented tasks; confuses left and right, and over and under; learns best through hands-on experiences.
- **Language and Reading Skills:** Gets dizzy and develops headaches when reading; doesn't read for pleasure; shows transpositions, additions, substitutions, or reversals in letters, numbers, and words when reading or writing; spells phonetically and inconsistently; has difficulty putting thoughts into words.
- **Numbers Skills:** Has difficulty learning to tell time or being on time; can do arithmetic but not word problems; has trouble grasping algebra or higher math; poor memory for sequences.
- **Behavior:** May be disorderly or disruptive in class; is easily frustrated about school, reading, writing, or math; shows dramatic increase in symptoms under time pressure or emotional stress.
- **Vision:** May complain of vision problems that don't show up on standard tests; may lack depth perception and peripheral vision.

Not all of these problems are experienced by all dyslexics; no two dyslexics are likely to be the same in terms of the range of problems they exhibit.

Most dyslexia researchers now accept that the problems highlighted above stem from an underlying neurological deficit. A growing body of research demonstrates that dyslexia is not a problem of motivation, teaching, or socio-economic background but stems from the fact that dyslexic brains work differently than non-dyslexic brains. Though researchers have reached a consensus on a neurological basis of dyslexia, the precise nature of these neurological deficits remains the subject of intense debate.

Alternative Frameworks of Dyslexia

Despite decades of research there is still fundamental disagreement over the neurological and cognitive mechanisms thought to be responsible for dyslexia. There are currently four major etiological theories: phonological deficit, magnocellular deficit, cerebellar deficit, and auditory temporal processing deficit - each of which has an impressive body of empirical support. These four theories can be divided into two competing frameworks. The phonological-deficit theory of dyslexia stands on its own as a language-based explanation, while the other three theories can be subsumed within a framework that attempts to explain dyslexia in terms of low-level sensorimotor deficits.

Phonological-Deficit Theory

According to the phonological-deficit theory, the specific reading difficulty characteristic of dyslexia is directly caused by a deficit in the processing and representation of speech sounds. The phonological-deficit theory holds that learning an alphabetic writing system requires the brain to map letters to basic speech sounds (grapheme-phoneme conversion). Dyslexia, it is argued, is a specific problem in representing or recalling these basic sound units. Many dyslexic people have difficulty retaining speech in short-term memory and segmenting that speech into its constituent phonemes. This difficulty has led proponents of this theory to suggest that dyslexia does not impair reading directly but slows the development of the spoken language substrate that is critical in developing reading skills (Shaywitz et al., 1998).

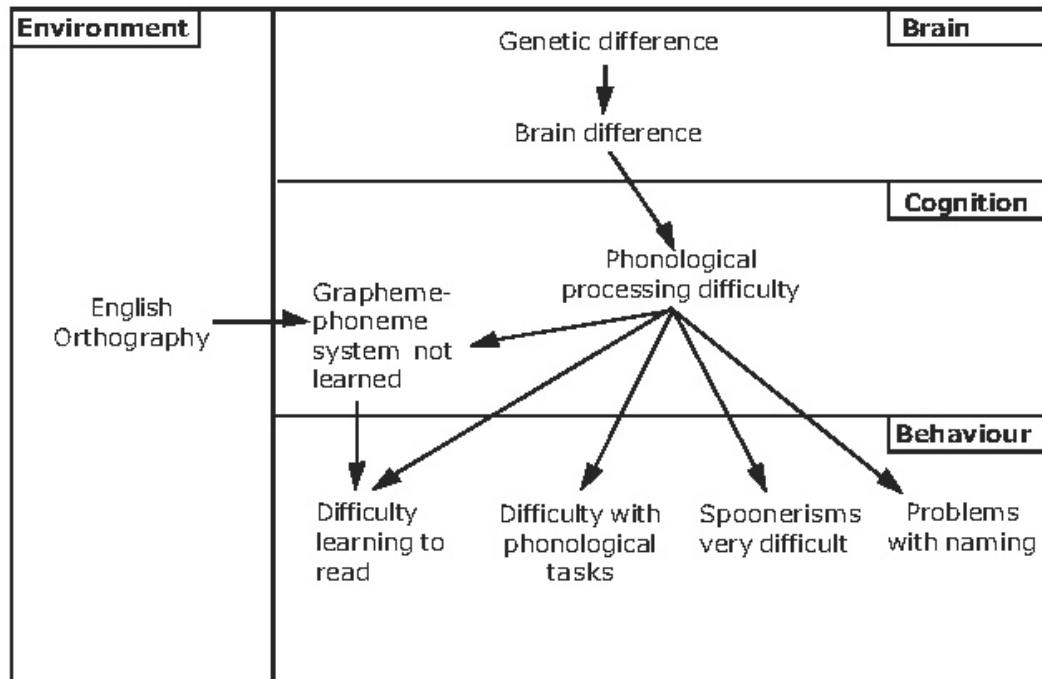


Figure 1: Phonological-deficit theory of dyslexia

Figure 1 shows the causal path assumed by the phonological-deficit model. Genetic differences lead to differences in neural structure which directly affect the ability to carry out phonological processing. Dyslexia is seen as a deficit at the level of the phonologic module which impairs the ability to segment written words into their phonological components. Higher order linguistic cognitive functions are assumed to be intact.

Much research evidence points to the importance of phonological ability in the reading process. Pre-school phonological aptitude predicts future skill at reading while training in phonological awareness significantly improves reading ability (e.g. Bradley & Bryant, 1983). Neural circuits that respond differently in dyslexic and non-dyslexic people while performing phonological tasks have also been found by means of functional magnetic resonance imaging (fMRI) scanning (e.g. Shaywitz et al., 1998). Short-term memory deficits found in many dyslexics have been shown to be due to differences in the efficiency of the speech-based rehearsal component, the phonological loop, of short-term memory (McDougall et al., 1994).

Given the well documented phonological problems in dyslexia very few researchers would deny the importance of phonological processing in this disorder. Where many see a problem with the phonological-deficit theory is its difficulty in explaining the numerous sensorimotor deficits associated with dyslexia. These deficits are not isolated within the language system or the phonologic module but are problems located at very low-level neural processes.

General Sensorimotor Deficit

There is now overwhelming evidence that the majority of dyslexics have problems with basic sensorimotor activities. Research demonstrates that such deficits exist in (1) the visual magnocellular pathways, (2) auditory analogues of the visual magnocells responsible for the processing of rapid auditory information, and (3) the cerebellum area of the brain responsible for timing and coordination of skilled activities as well as some integration of various sources of sensory information. The first two of these areas have recently been subsumed into the magnocellular-dysfunction theory of dyslexia (e.g. Stein & Walsh, 1997). While the cerebellar-deficit theory is still considered a separate explanation of dyslexia, the cerebellum's role as target for many of the brain's magnocells and its role in timing suggest it may parallel the general magnocellular theory of dyslexia.

General Magnocellular Dysfunction

A growing body of research indicates that many dyslexics have neurological deficits in those areas of the brain responsible for processing fast temporal information.

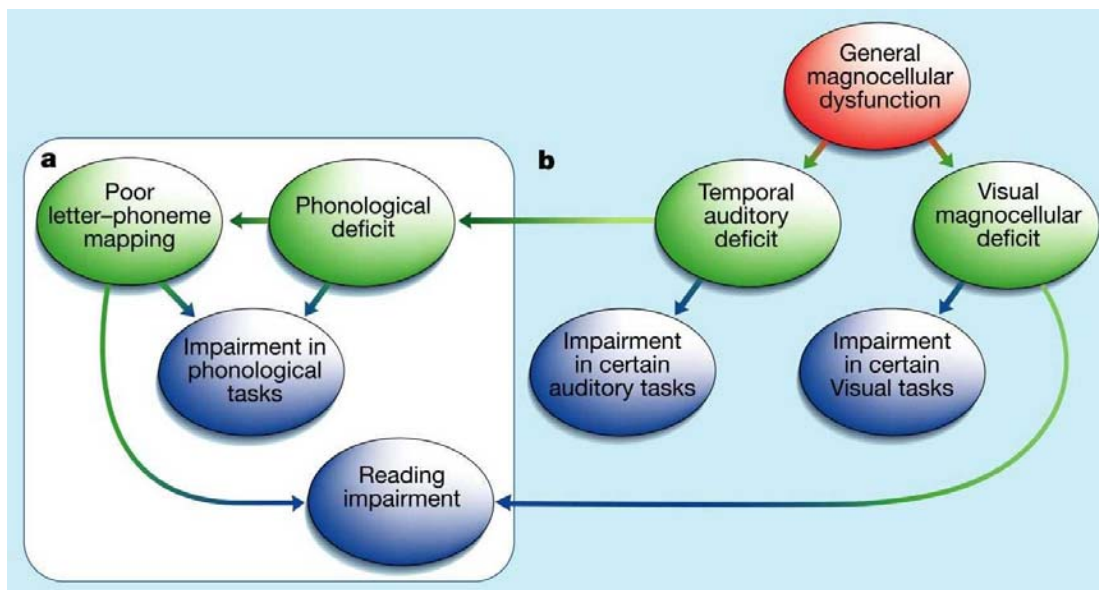


Figure 2: General magnocellular dysfunction theory of dyslexia

Figure 2 shows how the general magnocellular-dysfunction theory relates to the reading impairment seen in dyslexia. The white shaded area labeled “a” represents the phonological-deficit theory. In the area labeled “b” it can be seen that this phonological deficit is itself explained in terms of a temporal auditory deficit, with further reading impairment caused by deficits in the visual magnocellular system. The next three sections will review some of the evidence for the existence of temporal auditory, visual magnocellular, and cerebellar deficits and their relationship to dyslexia.

Visual-Magnocellular Deficits

Visual stimuli received by the eyes are transduced and transmitted by retinal ganglion cells. There are two major groupings of ganglion cells: magno cells (M cells) and parvo cells (P cells). M cells have quick acting membrane channels and heavily myelinated axons that allow these neurons to respond to stimuli and transmit information extremely quickly. M cells form about 10% of the eye’s retinal ganglion cells and are located in the periphery of the retina; the remaining 90% of the retinal ganglion cells are the much smaller P cells. The M cells have large receptive fields and so respond over a large area of visual space, not to the fine details of an object which is the role of the P cells. M cells are also specialized to detect visual transients, rapid changes in illumination, and so signal when new events occur in the visual environment. P

cells with their smaller receptive fields respond to color and are best for perception of fine details. The M cells and P cells pass through the lateral geniculate nucleus (LGN), a neural structure with two distinct layers: magnocellular and parvocellular. The LGN keeps information about motion, color, and form separate and transmits them in separate channels to the visual cortex.

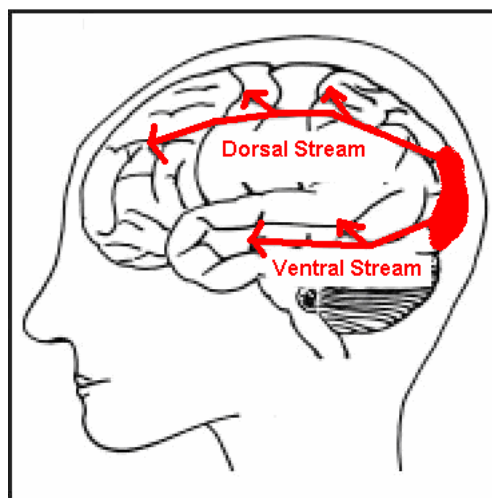


Figure 3: Separate streams of information leaving the visual cortex

Despite intermingling of M and P cells, two distinct visual systems have been identified leaving the visual cortex (Ungerleider & Mishkin, 1982; Milner & Goodale, 1995). A predominantly magnocellular stream of information takes a dorsal path from the visual cortex towards the parietal visual cortical areas that deal with motion, movement and navigation, and spatial reasoning. A parvocellular stream of information leaves the visual cortex in a ventral direction towards the temporal visual areas involved with the complex perception of patterns and forms as recognizable objects. Due to these separate functions the dorsal magnocellular stream is known as the ‘Where’ stream, as it is primarily concerned with where objects and self are, while the ventral parvocellular stream is known as the ‘What’ stream for its important role in identifying objects. In addition to the parietal visual areas, the dorsal stream also projects to all areas dealing with the guidance of eye movements, and also to the cerebellum.

A preponderance of evidence shows that many dyslexics have impaired development of the visual magnocellular system. Research reveals that the magnocellular layers of the LGN are more disordered in dyslexics than in non-dyslexics, and that the magnocells are 30% smaller

(Galaburda & Livingstone, 1993; Livingstone et al., 1991). Both adult and child dyslexics are significantly less sensitive to visual motion, a magnocellular activity, than non-dyslexics (Cornellisen et al., 1995). Visual motion sensitivity is also a very good predictor of a person's reading ability across a wide range (Witton et al., 1998). Different reading sub-skills are differentially affected by the type of motion processing deficit that exists. A deficit in detecting coherent motion is associated with problems in reading accuracy, while a deficit in velocity discrimination is associated with a lack of reading fluency (Wilmer et al., 2004). The magnocellular system is also intimately involved in the ocular motor system responsible for our eye movements during reading. Many dyslexics report that words and letters "move around on the page," producing blurred or merged visual images. Research has shown that binocular control of vergence eye movements is poor in dyslexic readers (Stein & Fowler, 1985).

Temporal-Auditory Deficits

The auditory system does not have an anatomically distinct magnocellular system such as is found in the visual system. There are, nonetheless, analogous cells within the auditory system that specialize in tracking rapid frequency and amplitude changes in acoustic signals (Trussel, 1998). Galaburda et al. (1994) found disordered large neurons within the medial geniculate nucleus, a relay structure for the auditory system, in dyslexic brains; these were similar to disordered neurons found in magnocellular regions of the LGN. Accuracy in tracking acoustic amplitude and frequency transients is necessary for phonological analysis; Stein & Talcott (1999) suggest that the phonological deficits evident in dyslexia could be due to deficits within this auditory magnocellular system. Talcott et al. (1999) have shown that both phonological and reading ability strongly covary with a child's sensitivity to modulations in auditory frequency. One of the primary tests of phonological ability is non-word reading. Talcott et al. (2000) have shown that over 50% of the variance in non-word reading can be accounted for by auditory frequency modulation sensitivity.

Cerebellar Deficit

The cerebellum is the second largest structure in the brain after the cerebrum. It is located at the rear of the brain beneath the cerebral hemispheres and on top of the brain stem.

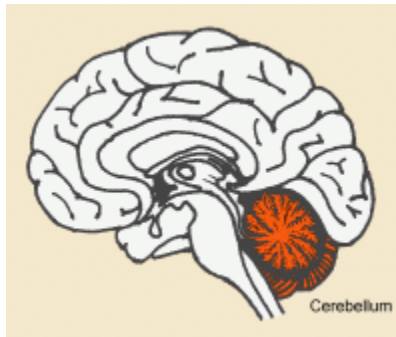


Figure 4: The location of the cerebellum (red) in an interior, side representation of a brain

Long thought to be concerned solely with skilled motor control, research over the last decade has shown the cerebellum to have a much wider role. Recent advances in functional neuroimaging have shown that the cerebellum is associated with cognitive processes such as memory retrieval, control of attention, verbal fluency, and timing and synchronization of a range of neural activities. It has also shown that cerebellar impairment can lead to problems on a wide range of cognitive tasks (Ivry, R.B. et al., 2001).

The cerebellum has been implicated in the etiology of dyslexia. A number of research studies demonstrate that dyslexics suffer from a number of ‘soft’ cerebellar symptoms. Fawcett & Nicholson (1999) found 80% of a sample of dyslexic children showed clear cerebellar symptoms as indexed by clinical tests of cerebellar dysfunction. In a PET study Nicholson et al. (1999) applied a sequence learning task known to cause considerable cerebellar activation in non-dyslexics and found only 10% of that activation level in a sample of dyslexic adults. Other ‘soft’ cerebellar symptoms include delayed milestones in dyslexics such as crawling, walking, and learning to ride a bicycle, as well as the clumsiness often reported in dyslexics (Nicholson et al., 1995).

Dyslexia Frameworks – Conclusion

The extensive research evidence over the last decade demonstrating that dyslexics have problems in visual magnocellular, auditory temporal, and cerebellar function must cast doubt on the Phonological-Deficit Theory. It is difficult to see how a purely language based disorder could lead to numerous low-level neural deficits. The most convincing explanatory framework for dyslexia would appear to be a scheme similar to that outlined in Figure 2: a problem in those low level neural circuits responsible for processing rapid, transient information from the various senses leading to the literacy problems we see in dyslexia.

Temporal auditory deficits lead to difficulty in processing speech sounds of short duration and thence to the phonological problems observed in dyslexics. Deficits in the visual magnocellular pathways affect reading abilities via impairment of ocular motor control of rapid eye movements. The cerebellum as the brain's main timing device comfortably fits into such a general magnocellular system. Stein (2001) refers to the cerebellum as the head ganglion of the magnocellular system because of the dense inputs it receives from all magnocellular systems. Bower & Parsons (2003) have provided evidence that the cerebellum is involved in coordinating the sensory information acquired by the brain. Thus, deficient input from the brain's rapid, transient pathways could well lead to cerebellar type symptoms in dyslexia.

Dyslexia and BrightStar

BrightStar is the name of a computer-based intervention to alleviate dyslexic problems and aid in the learning of literacy skills. The technology behind BrightStar is informed by recent advances in scientific knowledge and theory concerning the etiology of dyslexia.

BrightStar Definition of Dyslexia

The following is the theory-driven definition of dyslexia that guides research on the BrightStar intervention program. A neurological condition manifested as a deficit in the brain's temporal integration of fast (<250ms) information in-flow, particularly the reading of written text, and which is assumed to affect at least three areas of brain activity:

- Magnocellular reactivity
- Ocular motor control
- Cerebellar processing

This definition places BrightStar within the generalized magnocellular framework discussed in previous sections, and highlights the fact that an underlying deficit in the way the brain processes rapid, transient information leads to the major literacy difficulties experienced by dyslexics. Reduced sensitivity in the magnocellular system leads to a number of perceptual problems such as depth, brightness, and motion discrimination, all of which can impair reading ability. Ocular motor control — saccadic eye movements important for scanning text, as well as the control of eye fixations required so that a reader can fixate on text and identify letters and words — will be affected by deficits within the magnocellular system. Finally, the cerebellum's role as central timing mechanism and integrator of sensory information will be compromised by problems in the rapid, transient neural circuits. Tasks such as grapheme-phoneme mapping require precise timing and coordination of information from the auditory and visual channels. If either the auditory or visual rapid, transient circuits are not precise enough in timing information then the integration of separate streams of information needed to perform such a mapping task will be seriously affected.

The above suggests that to alleviate many of the problems that make reading such a problematic task for dyslexics, the brain's rapid, transient circuits need retraining to improve the speed of processing of visual stimuli and to better time visual events. The BrightStar intervention is predicated on brain plasticity and the knowledge that it is possible to differentially stimulate and retrain neural circuits, thereby encouraging the development of new synaptic connections. This may then facilitate more efficient use of spatial and temporal information.

How BrightStar Works

BrightStar technology utilizes fast, transient visual stimuli consisting of linear arrays of moving, flashing lights randomly distributed on a computer screen. These stimuli are synchronized with the heart beat rhythm, variation of which is controlled by the subject's autonomic nervous system (ANS). The subject's attention is periodically directed to the center of the screen by means of a simple exercise involving hand/eye coordination. This allows the computer display to stimulate the subject's peripheral vision.

The particular distribution in time and space of the presentation of the BrightStar display, this distribution itself driven by the subject's nervous system, is designed to stimulate the attentive and perceptual processes involved in fast stimuli changes, as well as those neural processes involved with the parasympathetic side of the ANS. The spatiotemporal configuration of these visual events is based on established findings in the scientific literature, particularly the extensive research carried out in the last forty years into the relationship between attention and changes in the parasympathetic component of the ANS, as well as the effects of brief, transient, visual stimuli on neural pathways and perceptual processes.

The BrightStar technology consists of a unique, novel animated visual display. The generated display is an optical flow field comprised of pre-attentive visual stimuli synchronized to an individual's periodic physiological activity. The three-fold aim of this display is to promote an increase in autonomic inhibitory control, magnocellular reactivity, and visual-motor integration.

The optical flow field comprises a moving array of rectilinear shapes that move with variable direction, speed, size, and contrast. This variability is achieved through synchronization with the variation in the heartbeat of an individual. This variable stimulus display is designed to initiate pre-attentive processes in the individual, i.e., stimuli that contain information about timing, motion, etc which the brain can extract on a non-cognitive level. Synchronization of the stimuli with the cardiac heart cycle allows presentation of stimuli at optimum points within this cycle, during which the body's physiological mechanisms allow the most efficient extraction of visual information (e.g. Sandman, et al., 1977).

The magnocellular system operates in the domain of pre-attentive processes since its primary role is the low-level detection and extraction of information concerning motion and spatial position. Promotion of magnocellular reactivity occurs because the entire BrightStar optic flow field — its motion, color, speed, and shapes — is designed to differentially stimulate the magnocellular system.

Autonomic inhibitory control is achieved by the nature of the display stimuli and by its synchronization with the subject's heartbeat to promote parasympathetic activity within the autonomic nervous system. Inhibitory control is important for a number of ocular motor activities. Saccadic eye movements and smooth eye tracking greatly benefit from parasympathetic inhibition which aids these ocular motor activities to proceed in a smooth and efficient fashion. Independent research at Nottingham University (Liddle et al., *in press*) demonstrated a significant shift toward parasympathetic activity in subjects who were exposed to the BrightStar stimuli compared to control subjects experiencing a placebo display.

Visual-motor integration is promoted via the magnocellular properties of the BrightStar display in conjunction with the performance of a sensory-motor task periodically throughout a BrightStar session while still receiving peripheral magnocellular stimulation. The aim is to stimulate those cerebellar-magnocellular circuits important for acquiring timing information from sensory stimuli.

Conclusions

BrightStar is a non-invasive technology that works by stimulating targeted neural circuits via a visual display in order to alleviate the symptoms of dyslexia and facilitate the acquisition of literary skills. The theoretical underpinnings of this technology lie squarely within a generalized magnocellular dysfunction framework for which there is an ever growing body of research support. Low-level deficits in the neural processing of rapid, transient information are now accepted by many mainstream researchers as being the most important factors in dyslexia. BrightStar, a non-language based intervention, is uniquely designed to work at the level of these neural processes. It takes advantage of brain plasticity to promote retraining of these neural circuits, to help the dyslexic individual acquire literacy skills.

References

- Bower, J.M., and Parsons, L.M. (2003). Rethinking the lesser brain. *Scientific American*, 289, 50-57.
- Bradley, L., Bryant, P. (1983) Categorizing sounds and learning to read: a causal connection. *Nature*, 301, 419.
- Cornellisen, P.L., Richardson, A.R., Mason, A., Fowler, M.S., and Stein, J.F. (1995). Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls. *Vision Research*, 35, 1483-1494.
- Fawcett, A.J. and Nicholson, R.I. (1999). Performance of dyslexic children on cognitive and cerebellar tests. *Journal of Motor Behaviour*, 31, 68-79.
- Galaburda, A.M., and Livingstone, M. (1993). Evidence for a Magnocellular defect in developmental dyslexia. *Annals of the New York Academy of Sciences*, 682, 70-82.
- Galaburda, A.M, Menard, T., and Rosen, G.D. (1994). Evidence for aberrant auditory anatomy in developmental dyslexia. *Proceedings of the National Academy of Science of the USA*, 91, 8010-8013.
- Ivry, R.B., Justus, T.C., and Middleton, C. (2001). The cerebellum, timing, and language: Implications for the study of dyslexia. In Wolf, M. (Ed), *Dyslexia, Fluency, and the Brain*, pp. 189-211. Timonium, MD: York Press.
- Liddle, E., Jackson, G and Jackson, SR. (in press). An evaluation of a visual biofeedback intervention in dyslexic adults. Accepted for publication in *Dyslexia*.
- Livingstone, M.S., Rosen, G.D., Drislane, F.W., and Galaburda, A.M. (1991). Physiological and anatomical evidence for a Magnocellular defect in developmental dyslexia. *Proceedings of the National Academy of Sciences*, 88, 7943-7.
- McDougall, S., Hulme, C., Ellis, A.W., and Monk, A. (1994). Learning to read: The role of short-term memory and phonological skills. *Journal of Experimental Child Psychology*, 58, 112-133.
- Milner, A.D, and Goodale, M.A. (1995). *The Visual Brain in Action*. Oxford; Oxford University Press.
- Nicholson, R.I., Fawcett, A.J., Berry, E.L., Jenkins, I.H., Dean, P., and Brooks, D.J. (1999). Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. *Lancet*, 353, 1662-7.

- Nicholson, R.I., Fawcett, A.J., and Dean, P. (1995). Time estimation deficits in developmental dyslexia: evidence of cerebellar involvement. *Proceedings of the Royal Society of London series B*, 259, 43-7.
- Sandman, C.A., McCanne, T.R., Kaiser, D.N., and Diamond B. (1977). Heart rate and cardiac phase influences on visual perception. *Journal of Comparative and Physiological Psychology*, 91, 1, 189-202.
- Shaywitz, S.E., Shaywitz, B.A., Pugh, K.R., Fulbright, R.K., Constable, R.T., Mencl, W.E., Shankweiler, D.P., Liberman, A.M., Skudlarski, P., Fletcher, J.M., Katz, L., Marchione, K.E., Lacadie, C., Gatenby, C., and Gore, J.C. (1998). Functional disruption in the organization of the brain for reading in dyslexia. *Proceedings of the National Academy of Sciences*, 95, 2636-2641.
- Shaywitz, B.A., Shaywitz, S.E., Pugh, K.R., Fulbright, R.K., Mencl, W.E., R. Constable, T., Skudlarski, P., Fletcher, J.M., Lyon, G.R. and Gore, J.C. (2001). The neurobiology of dyslexia. *Clinical Neuroscience Research*, 1, 291-299.
- Stein, J.F., and Fowler, M.S. (1985). Effect of monocular occlusion on visuomotor perception and reading in dyslexic children. *Lancet*, 337, 69-73.
- Stein, J.F. and Talcott, J. (1999). Impaired neuronal timing in developmental dyslexia – the Magnocellular Hypothesis. *Dyslexia*, 5, 59-77.
- Stein, J.F. and Walsh, V. (1997). To see but not to read; the Magnocellular theory of dyslexia. *Trends in Neuroscience*, 20, 147-152.
- Talcott, J.B., Witton, C., McClean, M., Hansen, P.C., Rees, A., Green, G.G.R., and Stein, J.F. (1999). Can sensitivity to auditory frequency modulation predict children's phonological and reading skills? *NeuroReport*, 10, 2045-2050.
- Talcott, J.B., Witton, C., McClean, M., Hansen, P.C., Rees, A., Green, G.G.R., and Stein, J.F. (2000). Visual and auditory transient sensitivity determines word decoding skills. *Proceedings of the National Academy of Sciences*, 97, 2952.
- Trussel, L.O. (1998). Cellular mechanisms for preservation of timing in central auditory pathways. *Current Opinion in Neurobiology*, 7, 487-492.
- Ungerleider, L.G. and Mishkin, M. (1982). Two cortical visual systems. In D.J, Ingle, M.A, Goodale and R.J.W, Mansfield (Eds), *The Analysis of Visual Behaviour*, pp. 549-586. Cambridge, MA; MIT Press.
- Wilmer, J.B., Richardson, A.J., Yue Chen, and Stein J.F. (2004). Two visual motion processing deficits in developmental dyslexia associated with different reading skills deficits. *Journal of Cognitive Neuroscience*, 16:4, 528-540.

Witton, C., Talcott, J.B., Hansen, P.C., Richardson, A.J., Griffiths, T.D., Rees, A., Stein, J.F., and Green, G.G.R. (1998). Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexic and normal readers. *Current Biology*, 8, 791-797.