Reader™
The Science of Dyslexia and Reader

A White Paper
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Abstract

This white paper outlines some of the current scientific theories of the dyslexia disorder and introduces some of the basic principles behind a novel technological remediation technique, Reader™. The general aim of Reader is to ameliorate reading difficulties (of slow & lazy readers), and to particularly alleviate the symptoms of dyslexia by facilitating 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 and spelling, to telling time, and/or organization. Not all problems are experienced by all dyslexics.

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.

Reader is a computer-based technological remediation developed by the founders and scientists of BrightStar Learning that is 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. Reader works, in part, by utilizing specifically designed and specifically timed computer graphics presented as part of a computer game-like exercise. The specially designed computer graphics help retrain the users' 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?

There are many definitions of dyslexia, many of which reflect their author’s theoretical orientation. One of the less theory-laden definitions suggests 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 – great difficulty in acquiring the fluent, accurate reading skills that the vast majority of the population takes for granted.

While difficulty in learning to read might be the most obvious fact about dyslexia, this difficulty stems from a wide array of 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 leading to a situation where no two dyslexics are likely to be the same in terms of the range of problems they exhibit.
The majority of dyslexia researchers now accept that the problems highlighted above stem from an underlying neurological deficit. There is a growing body of research which demonstrates that dyslexia is not a problem of motivation, teaching or socio-economic background but stems from the fact that dyslexic brains work differently from non-dyslexic brains. Though researchers have reached a consensus on the neurological basis of dyslexia the precise etiology of these neurological deficits is still the subject of intense debate.

Two Alternative Frameworks of Dyslexia

Despite decades of research, there is still a fundamental disagreement over the neurological and cognitive mechanisms which are thought to be responsible for dyslexia. At the time of writing of this white paper, there were four major theories as to the causal basis of dyslexia – Phonological Deficit, Magnocellular Deficit, Cerebellar Deficit and Auditory Temporal Processing Deficit - each of which have an impressive body of empirical support. These four theories can be divided into two competing frameworks, the Phonological Deficit theory of dyslexia standing on its own as a language based explanation, while the other three theories can be subsumed within a framework which attempts to explain dyslexia in terms of low-level sensorimotor deficits.

Phonological Deficit Theory

According to this theory, the specific reading difficulty which is characteristic of representation of speech sounds. Phonological Deficit theory suggests 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 in the retention of speech in short-term memory and in segmenting that speech into its constituent phonemes. This has led proponents of this theory to suggest that impairment in dyslexia does not affect reading directly but affects the development of the spoken language substrate that is critical in developing reading skills (Shaywitz et al, 1998).
Figure 1 above, 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. According to this model of dyslexia, reading consists of two essential processes: decoding and comprehension. 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.

There is much research evidence to demonstrate the importance of phonological ability in the reading process. Pre-school phonological aptitude is known to predict future skill at reading while training in phonological awareness significantly improves reading ability (e.g. Bradley & Bryant, 1983)\(^3\). Neural circuits which respond differently in dyslexic and non-dyslexic people while performing phonological tasks have also been found by means of fMRI scanning (e.g. Shaywitz et al, 1998)\(^2\). Short-term memory (STM) 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 the STM (McDougall et al. 1994)\(^4\).

In the face of the well documented evidence of phonological problems in dyslexia, very few researchers would deny the importance of phonological processing. Where many see a problem with this particular model, is its difficulty in explaining the numerous sensorimotor deficits associated with dyslexia which have come to light as a result of research conducted over the last decade. 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 has demonstrated that many of such deficits exist at the level of the visual magnocellular pathways, auditory analogues of the visual magnocells responsible for the processing of rapid auditory information and of the cerebellum the area of the brain responsible for timing and coordination of skilled activities as well as some aspects of the integration of various sources of sensory information. The first two of these areas have recently been subsumed into the General Magnocellular Dysfunction theory of dyslexia (e.g. Stein & Walsh, 1997). While the Cerebellar Deficit theory of dyslexia is still considered to be a separate explanation of dyslexia given the cerebellum is the target for many of the brains magnocells and its role in timing it is not inconceivable that this will also form part of a general magnocellular theory of dyslexia in the near future.

General Magnocellular Dysfunction

There is a growing body of research evidence indicating 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

The above figure shows how the general magnocellular dysfunction theory relates to the reading impairment seen in dyslexia. The darker orange 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 being caused by deficits in the visual magnocellular system. The next three sections will review some of the evidence for the existence of both temporal auditory, visual Magnocellular and cerebellar deficits and their relationship to dyslexia.

**Visual Magnocellular Deficits**

Visual stimuli received by the eyes are transmitted, via intermediate neural structures, to the visual cortex by retinal ganglion cells. There are two types of ganglionic cell: magnocells (M cells) and parvocells (P cells). M cells have quick acting membrane channels and heavily myelinated axons which allow these neurons to respond to stimuli & transmit information extremely quickly. M cells form about 10% of the eyes retinal ganglion cells and are located in the periphery of the retina with the remaining 90% of the retinal ganglion cells being the much smaller P cells. The M cells have large receptive fields and so respond over a large area of visual space and 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 thus keeps information about motion and color separate from information about form and transmits the segregated information in separate channels to the visual cortex.

Despite intermingling of M & P cells two distinct visual systems have been identified leaving the visual cortex (Ungerleider & Mishkin, 1982; Milner & Goodale, 1995) \(^6\)\(^7\).

A predominantly magnocellular stream of information takes a dorsal path from the visual cortex towards the parietal visual cortical areas which deal with motion, movement & navigation and spatial reasoning. A mainly 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, and the ventral parvocellular stream is known as the ‘What stream’, recognizing its important role in identifying objects. As well as the parietal visual areas the dorsal stream also projects to all areas dealing with the guidance of eye movements and also to the cerebellum.

There is now a preponderance of evidence which shows a substantial number of dyslexics has impaired development of the visual magnocellular system. Research has shown that the magnocellular layers of the LGN are more disordered in dyslexics than non-dyslexics and that the magnocells are 30% smaller (Galaburda & Livingstone, 1993; Livingstone et al, 1991)\(^8,9\). Both adult and child dyslexics have been shown to be significantly less sensitive to visual motion, a magnocellular activity, than non-dyslexics (Cornellisen et al, 1995)\(^10\). Visual motion sensitivity is also a very good predictor of a person’s reading ability no matter whether they are poor or good readers (Witton et al, 1998)\(^11\).

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 with reading accuracy while a deficit in velocity discrimination is associated with a lack of reading fluency (Wilmer et al, 2004)\(^12\). The Magnocellular system is also intimately involved in the ocular motor system which is 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)\(^13\).

**Temporal Auditory Deficits**

While 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 which specialize in tracking rapid frequency and amplitude changes in acoustic signals (Trussel, 1998)\(^14\). Galaburda et al (1994)\(^15\) found disordered large neurons within the Medial Geniculate Nucleus, a relay structure for the auditory system, in dyslexic brains similar to those found for magnocells in the LGN. Accuracy in tracking acoustic amplitude and frequency transients is necessary for phonological analysis and Stein & Talcott (1999)\(^16\) suggest that the phonological deficits which are evident in dyslexia could be due to deficits within this auditory magnocellular system. Talcott et al, 1999\(^17\) have shown that both phonological and reading ability strongly co-vary with a child’s sensitivity to modulations in auditory frequency.
One of the primary tests of phonological ability is non-word reading and Talcott et al, 2000\textsuperscript{18} 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 and is located at the rear of the brain beneath the cerebral hemispheres and on top of the brain stem.

Long thought to be solely concerned with skilled motor control, research over the last decade has shown the cerebellum to have a much wider role. Recent advances in neuro-imaging techniques have shown the cerebellum is associated with cognitive processes such as memory retrieval, control of attention, verbal fluency, and timing & synchronization of a range of neural activities and that cerebellar impairment can lead to problems on a wide range of cognitive tasks (Ivry, R.B. et al. 2001\textsuperscript{19}).

The cerebellum has also been implicated in the causation of dyslexia with a number of research studies demonstrating that dyslexic people suffer from a number of ‘soft’ cerebellar symptoms. Fawcett & Nicholson (1999)\textsuperscript{20} found 80\% of a sample of dyslexic children showed clear cerebellar symptoms as indexed by clinical tests of cerebellar dysfunction. Nicholson et al (1999)\textsuperscript{21} showed in a PET study that on a sequence learning task known to cause considerable activation in the cerebellum of non-dyslexics, only 10\% of that activation level was found in a sample of dyslexic adults. Other ‘soft’ cerebellum signs include delayed milestones in dyslexics such as crawling, walking, learning to ride a bicycle as well as the clumsiness often reported in dyslexics (Nicholson et al, 1995)\textsuperscript{22}.

**Dyslexia Frameworks – Conclusion**

The large amount of research evidence which has emerged over the last decade demonstrating that dyslexics have problems with the visual magnocellular system, the auditory temporal system and the cerebellum must cast doubt on the Phonological Deficit Theory. It is difficult to see how a purely language based disorder could lead to these 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 with 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 a difficulty in processing speech sounds of a short duration and hence to the phonological problems which are observed in dyslexics. Deficits in the visual magnocellular pathways affect reading abilities via problems with ocular motor control of rapid eye movements (i.e. saccadic suppression).

The cerebellum as the brain’s main timing device comfortably fits into such a general magnocellular system. Stein (2001)\textsuperscript{23} refers to the cerebellum as the head ganglion of the magnocellular system because of the dense inputs it receives from all the brains magnocellular systems. Bower & Parsons (2003)\textsuperscript{24} have provided evidence that the cerebellum is involved in coordinating the sensory information acquired by the brain. Thus deficient input from the brains rapid, transient pathways could well lead to cerebellar type symptoms in dyslexia.

**Dyslexia and Reader**

Reader is a computer-based intervention to alleviate dyslexic disabilities and aid in the acquisition of literacy skills. The technology behind Reader is built upon recent advances in scientific knowledge concerning the causation of dyslexia and the theoretical understanding of Reader.

**Reader Definition of Dyslexia**

Following is the definition of dyslexia which guides the research on the Reader remediation program:

A neurological condition manifested as a deficit in the brain’s temporal integration of fast information (<220ms) which is assumed to affect at least three areas of brain activity:

- Magnocellular reactivity
- Ocular motor control
- Cerebellar processing

This definition places Reader 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 impact on reading ability. Ocular motor control in terms of both saccadic eye movements, important for scanning text, and 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 in its role as responsible for the brain's central timing mechanism and integrator of sensorial 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 their timing information, then the integration of separate streams of information needed to perform such a mapping task will be seriously affected.

The above, therefore, suggests that in order to alleviate many of the problems which 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 better timing of visual events. The Reader intervention is predicated on brain's plasticity and the knowledge that it is possible to differentially stimulate neural circuits and to retrain these circuits, encouraging the development of new synaptic connections that will facilitate more efficient use of spatial and temporal information.

**How Reader Works**

Reader technology utilizes fast, transient visual stimuli that are devoid of meaning content. These stimuli are correlated in time with the individual’s statistical mean heart beat cycle value for his age group via proprietary synthetic algorithms, and consist of linear arrays of moving, flashing sprites (chains of Illuminated pixels) distributed over a computer screen. The subject’s attention is periodically directed to the center of the screen, by means of a simple game-like exercise involving hand-eye coordination, allowing the computer screen's dynamic display to stimulate the subject’s peripheral vision.

By means of the particular distribution in time and space of Reader's display (this distribution itself driven by the synthetic correlation algorithm imitating the individual’s RR interval time series intrinsic variable behavior), Reader is designed to promote the dyslexic’s attentive and perceptual processes involved in fast stimuli changes, as well as those neural processes involved with the parasympathetic side of the Autonomic Nervous System (ANS).
The spatiotemporal configuration of these visual events is based on established findings in scientific literature with extensive research having been 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.

Reader technology consists of a unique, novel animated visual display. The display generated is an optical flow field comprised of visual stimuli (sprites) where attentive resources are involuntarily allocated against a background of “orienting reflexes” or “preattentive processes." The sprites animated behavior (movement and flickering) is correlated via a synthetic algorithm to the subject’s statistical mean heart beat cycle value for his age group. The three-fold aim of this display is to promote an increase in (1) autonomic inhibitory control, (2) magnocellular reactivity and (3) visual-motor integration.

The optical flow field comprises a moving array of sprites which move with variable motion, direction, speed, size, and contrast. This variability is achieved through a synthetic algorithm that functionally imitates intrinsic RR interval time series variations of the heartbeat rhythm. This variable and fast stimulus display is designed to initiate pre-attentive processes in the subject’s brain (i.e. stimuli which contain information about timing, motion etc.) which can be extracted on a non-cognitive level.

Technologically enabling a synthetic correlation of the stimuli with the cardiac heart cycles allows the presentation of stimuli at optimum times within these cycles, during which the body’s physiological mechanisms attain a synergic correlation with attentional processes, resulting in the most efficient and rapid extraction of visual information (e.g. Sandman, et al. 1977) 25. The magnocellular system operates in the temporal domain of pre-attentive processes since its primary role is the low-level fast detection and extraction of information concerning motion and spatial position. Promotion of magnocellular reactivity occurs because the entire Reader optic flow field, its motion, color, speed, and shapes are all designed to differentially affect the magnocellular system.

Autonomic inhibitory control is achieved through the nature of the display stimuli and its synthetic correlation with the variations in a 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 University of Nottingham, UK (Liddle et al., 2005) \(^{26}\) demonstrated a significant shift toward parasympathetic activity in subjects who were exposed to the Reader stimuli technology (early Reader non-internet version) compared to control subjects experiencing a placebo display.

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

**Conclusions**

Reader is a technology that stimulates targeted transient neural circuits via a novel visual display in order to ameliorate reading difficulties (slow & reluctant readers) and alleviate the symptoms of dyslexia, thus facilitating the acquisition of literary skills. The theoretical underpinnings of this technology lie squarely within a generalized magnocellular dysfunction framework and effective allocation of attentional resources via transient covert attention against a background of “orienting reflexes” or “preattentive processes" 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 main-stream researchers as being influential factors in dyslexia. Reader, a non-language based training program, is uniquely designed to work at the level of these neural processes and take advantage of the brain's plasticity to promote retraining of these neural circuits helping slow, lazy readers and dyslexics to acquire literacy skills.
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