The Neurological Basis of Dyslexia

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A learning disability refers to difficulty processing certain kinds of information in a way that interferes with the acquisition of basic skills, such as reading, writing, and math. Dyslexia is a type of learning disability, for which there are multiple, varying definitions. For example, Shaywitz and Shaywitz (2001) describe dyslexia as an unexpected difficulty in accurate and fluent reading experienced by children and adults who possess the necessary intelligence and motivation to read. The current author describes dyslexia as a language based learning disability that involves difficulty acquiring and using the symbols of language.

Dyslexia is perhaps more common than once thought, as the International Dyslexia Association (2008) reports that as many as 15 – 20% of all people have symptoms of dyslexia. Reading disabilities are by far the most common learning disability, as 80% of individuals with a learning disability have a reading learning disability (Shaywitz & Shaywitz, 2001). Although boys are 4 times more likely than girls to be identified by their school as having a reading problem, males and females are actually affected equally (Shaywitz & Shaywitz, 2001). A clear genetic influence on dyslexia has been established, as 23 – 65% of children of dyslexic parents, 40% of children of dyslexic siblings, and 27 – 49% of parents who have a dyslexic child have dyslexia (Shaywitz & Shaywitz, 2001).

Dyslexia, by definition, is a neurologically based disorder. The aim of the present paper is to summarize research on the neurological basis of dyslexia. As will be seen below, the research has identified brain regions associated with both phonological and visual processing as involved in dyslexia.

Shaywitz and Shaywitz (2001) propose a neural model for reading, incorporating the left inferior frontal region, parietotemporal system, involving the inferior parieta lobule and the posterior portions of the superior temporal gyrus, and the occipitotemporal region. They state that individuals without reading difficulty show an activation of the left inferior parietal lobule and posterior portion of the left superior temporal gyrus, which dyslexics do not. Conversely, dyslexics show a bilateral increase in activation of the frontal lobes, specifica in the middle and inferior frontal gyri. Shaywitz and Shaywitz argue that the inferior parietal lobule, and in particular the angular gyrus as well as the posterior portion of the superior temporal gyrus function as association areas which map the “visual percept of print onto the phonological structure of language” (p. 14). This allows visual letters to be transformed into the phonemes (meaningful units of sound) they represent. When functioning properly, this system allows for quick and efficient decoding.

Shaywitz and Shaywitz state that the occipitotemporal region allows for rapid word identification. This relationship was examined by McCrory, Mechelli, Frith, and Price (2004). McCrory et al. (2004) examined the role of the occipitotemporal region (which they define as Brodmann area 37) in confrontation and rapid naming and how this relates to reading. They state that reading and naming an object are both tasks of lexical retrieval. In each, one perceives a visual stimulus and retrieves a stored phonological code. McCrory et al., using a PET scan, compared the brain activation of 80 dyslexic adults and 10 non-dyslexic adults during reading and naming tasks. All subjects were adult, right handed males. They found that rapid naming of both pictures and digits was significantly lower in the dyslexic group. In addition, they found that non-dyslexic subjects activated in the left occipitotemporal region for both picture naming and word reading, but dyslexics did not. These results are consistent with prior research, showing that dyslexics have difficulty with rapid automatized naming (RAN) and that this dysfunction is likely related to under-activation in the left occipitotemporal region.

Reading decoding is based on both phonics and sight vocabulary. Phonics is knowledge of the sounds that letter and letter combinations make. Phonics ability allows the reader to sound out unfamiliar words. Sight vocabulary refers to words the reader recognizes instantly without having to sound them out. Having an adequate sight vocabulary is directly related to reading fluency. In absence of a sufficient sight vocabulary, the reader must sound out too many words, slowing him or her down and undermining the rhythm of decoding. Poor fluency in turn can then impair comprehension – by the time the person reaches the end of the sentence or paragraph he or she has forgotten what was at the beginning.

Rapid automatized naming is an underlying skill for sight vocabulary. Adequate RAN enables a person to quickly retrieve from memory the word associated with the letter configuration at which one is looking (e.g. being able to see "t-h-e" and instantly know that the word is "the."). Thus, dyslexics' under-activation of the occipitotemporal region is likely a factor behind their poor RAN, which impedes fluency directly and comprehension indirectly.

On a side note, because dyslexic subjects showed difficulty with both picture *and* digit naming, McCrory et al. conclude that the left occipitotemporal region is not specific to orthographic decoding but instead is more generally involved in retrieving phonological codes from visual input. Thus, dyslexics' above deficit in this regard may be more accurately described as difficulty retrieving phonological codes in general as opposed to retrieving phonological codes specifically for orthographic information.

From a developmental perspective, McCrory et al. note that dyslexic children have difficulty with confrontation naming whereas dyslexic adults do not. However, dyslexic adults do show a deficit in *rapid* picture naming. This led McCrory et al. to conclude that the retrieval deficit is still present in dyslexic adults but only manifests under speeded conditions.

Frost, Landi, Mencl, Sandak, Fulbright, Tejada, Jacobsen, Grigorenko, Constable, and Pugh (2009) found that the role of the occipitotemporal region and the superior temporal gyrus in reading may be mediated by phonological awareness. As noted above, phonemes are the meaningful units of sound of which words are comprised. Phonological awareness is the ability to accurately interpret and mentally manipulate phonemes. It includes the ability to segment words into their constituent phonemes and identify and isolate individual phonemes in words. Phonological awareness is considered perhaps the single greatest predictor of reading skill.

Frost et al. examined brain activation using fMRI scans of 43, right handed subjects of mixed gender, whose ages ranged from 6 – 10 years. All subjects had Wechsler Abbreviated Scale of Intelligence Performance IQ scores of = 80. They found that when overall phonological awareness was low, the left occipitotemporal region was activated for speech, but not for print. Conversely, when overall phonological awareness ability was high, the left occipitotemporal region was activated for print, but not for speech. This lead Frost et al. to conclude that as people develop literacy skills the occipitotemporal area may become less dedicated to speech and more to print.

Frost et al. found that the left superior temporal gyrus became activated for speech regardless of whether phonological awareness was high or low. However, it became activated for print only when phonological awareness was high. is, it was not activated to print when phonological awareness was low, but was almost as activated to print as it was to speech when phonological awareness was high.

Frost et al. propose that phonological awareness may allow printed language to make contact with brain regions already set up for speech functions. The authors note that this is a logical explanation given that print represents a code of speech and phonological awareness allows one to connect a printed word to a word from one's speech lexicon.

Frost et al. also found that the left superior temporal gyrus and left inferior frontal gyrus appear to become more devoted to phonetic decoding (as opposed to sight word recognition) as phonological awareness increases. When phonological awareness was low, the left superior temporal gyrus and left inferior frontal gyrus were activated for consonant strings – that is, non-pronounceable words. As phonological awareness increased, the two areas became increasingly active for phonetic decoding and less active for non-pronounceable words. Perhaps without phonological awareness these areas cannot access the phonological structure of pseudowords and instead attempt a structural / visual analysis to decode.

Frost et al. found that the left superior temporal gyrus was particularly sensitive to the elision aspect of phonological awareness – it was more activated to print and pronounceable words the higher elision was. Elision reflects a more advanced phonological awareness skill in which one must isolate and mentally manipulate phonemes. An example elision task would be asking a person to say “tractor” without saying “or,” in which the correct answer would be “tract.” Thus, elision appears to be a particularly important predictor of the brains ability to apply phonetic decoding strategies.

Frost et al.’s findings suggest that the left superior temporal gyrus and left inferior frontal gyrus may be involved in the decoding of sight words in absence of phonological ability, and then as phonological ability develops they switch to p netic decoding. This explanation is consistent with the clinical observations of the current author. It is not an uncommon profile for a child with dyslexia to have poor phonological awareness and for his or her decoding ability to be limited to sight word recognition with little or no ability to apply phonetic decoding strategies. Such children have some sight words and read few if any pseudowords.[1] The decoding errors of these children tend to be “whole word substitution errors,” in which the reader decodes an unfamiliar target word by trying to match it to a known word based on the letters the two words share in common. For example, the child might read the word “center” as “cute,” apparently because they both begin with “c” and have a “t-e” at / towards the end of the word. Thus, such children, lacking in phonological awareness, decode based on the visual appearance of the words. With the growth of phonological awareness skills, however, children begin to apply phonetic decoding strategies.

The idea that a more visually based decoding approach developmentally precedes a more phonetic decoding approach is reflected in the normative data of individualized standardized

[1] The correct decoding of pseudowords is based entirely on phonetic decoding skill since, because they are made up, the reader has not encountered the words before and thus cannot rely on memory to decode them.

achievement tests. On these instruments, for young children at an age when they are just beginning to learn how to read, a raw score of 0 on a test of real word decoding often yields a lower standard score than a raw score of 0 on a test of pseudoword reading, implying that the norm is to be able to read words by sight before being able to do so phonetically. Again, this observation is consistent with Frost et al.'s finding that upon the development of phonological awareness, certain left hemisphere regions switch from processing sight words to apparently engaging in phonetic decoding, as if one transitions from a more sight word approach to increasingly phonetic decoding (at least as far as these brain regions are concerned).

Hoeft, Ueno, Reiss, Meyler, Whitfield-Gabrieli, Glover, Keller, Kobayashi, Mazaika, Jo, Just, and Gabrieli (2007) examined positive neuroimaging predictors of later phonetic decoding skills in a study of 64 right handed children of mixed gender ranging in age from 8 – 12 years, using fMRI scanning and voxel-based morphometry (VBM). They found that increased white matter density in the left superior temporal gyrus and inferior parietal region at the beginning of the school year was associated with phonics development at the end of the school year. These findings are consistent with those cited above implicating these areas in reading, including the association between the left superior temporal gyrus and phonetic decoding skills specifically. Hoeft et al. also found left temporal lobe activation predictive of later phonics ability, however, the specific area they found associated with it was the middle temporal gyrus as opposed to the superior temporal gyrus found in above studies.

Landi, Mencl, Frost, Sandak, and Pugh (2010) argued that because similar patterns of deficits have been found for both phonological and semantic based tasks, dyslexics' difficulties may not necessarily be restricted to phonological processing, but may reflect a more broad based linguistic processing deficit. They cite previous research in which subjects with reading difficulty on semantic tasks showed reduced activation in the left angular gyrus and left temporal lobe and increased activation in the inferior frontal gyri – deficits dyslexics also show on phonological tasks. Landi et al., again summarizing past studies, also note that people with reading difficulties have been found to perform poorly on auditory perception tasks, including those requiring: temporal order judgments of rapidly presented tones, extracting auditory stimuli from noise, and categorical perception. Thus, Landi et al. conclude that dyslexia may reflect more of a broad based language problem. This argument is consistent with McCrory et al.'s assertion discussed above that dyslexics may have a more broad based difficulty, in that case with regard to retrieval, as opposed to a narrow deficit restricted to orthographic processing.

Using fMRI scans, Landi et al. examined brain activation for individuals with and without reading difficulties for semantic versus phonological tasks and for auditory versus visual stimuli, using 26 mostly right handed (i.e. all but 2) individuals, of mixed gender, ranging in age from 9 – 19 years, with an average age of 12 ½ years. All trials presented subjects with a picture and a word. In the semantic task, subjects had to decide if both the picture and the word were alive. Words were presented both aurally and visually. In the phonological condition, subjects were presented with a picture and either a visually presented real word or pseudoword and had to decide if the two words rhymed. Landi et al. found that, across tasks (semantic and phonological) and modalities (auditory and visual) indi iduals without reading difficulty showed greater activation than individuals with reading difficulty in multiple areas, including those previously implicated in dyslexia, namely the left: superior temporal gyrus, inferior frontal gyrus, and occipitotemporal region. Thus, Landi et al. conclude that dyslexia may be a manifestation of a more broad based language processing deficit, as those with reading difficulty showed decreased brain activation not only for phonological tasks but for semantic tasks as we l.

In the face of dysfunctions in the brain regions typically used for reading, it has been argued that dyslexics compensate by using other systems. Shaywitz and Shaywitz argue that the increased activation in the inferior frontal gyri shown by dyslexics reflects their ability to develop an awareness of the sound structure by forming the word with their lips, tongue, and vocal apparatus. Thus, Shaywitz and Shaywitz's model indicates that instead of using an efficient "sight to sound" system of the posterior reg sate by attempting to leverage the speech system to decode words. This explanation is consistent with the fact that the frontal lobes are involved in speech production. In addition, it coincides with the common observation that when people have difficulty decoding they often use the speech system, engaging in such behaviors from moving their lips or whispering to switching from silent to aloud reading. This is observed both in students with reading disabilities as well as normal reading individuals when they encounter a word with which they have difficulty decoding.

Shaywitz and Shaywitz's model is also logical from a developmental – regression point of view. When encountering stress, individuals can regress to an earlier stage behavior. In addition, people learn to speak before they learn to read. It therefore makes sense that in the face of decoding difficulty an individual would regress to using an earlier, pre-requisite skill, in this case speech. The current author also suspects that this may be one of the causes of slow reading speed typically seen in individuals with reading disabilities. The author offers that at least one classification of slow readers are those who, even when reading "silently," are essentially reading aloud in their own minds, sub-vocally.

Numerous studies have found dyslexic individuals to activate in the right hemisphere for reading tasks typically associated with left hemisphere activation in non-dyslexic individuals. Shaywitz and Shaywitz speculate that the dyslexic, in absence of smoothly functioning left posterior systems, may compensate by using the less efficient right occipitotemporal area to perform visual analysis of words to gain recognition. The current author finds this explanation particularly interesting since a popular reading instructional technique, which is currently falling out of vogue, was for teachers to teach students to decode by examining the overall shape that is created by the letters that make up words. Perhaps it was the observation that dyslexics decode this way that prompted this technique in the first place. Shaywitz and Shaywitz's point that this represents an inefficient way of decoding implies that such a technique might actually be reinforcing the dyslexic's "bad habit" and is thus counter-indicated. Indeed, this approach to reading instruction has been sharply criticized in recent times an invalid and antiquated instructional strategy.

Consistent with Shaywitz and Shaywitz's position, Landi et al. found that when reading disabled subjects decoded there was increased activation in the right temporal and right frontal lobes. Specifically, real word decoding was associated with activation in the right inferior temporal region and phonetic decoding (i.e. the decoding of pseudowords) was associated with activation in the right middle temporal gyrus and right pre-central gyrus.

Hoeft et al. speculated that perhaps reading development involves a reduction in right hemisphere involvement and a growth of left hemisphere activation. They based this on their finding that one positive predictor of later phonics achievement was a decrease in activation of the right middle frontal gyrus and an increase in activation of the right fusiform. The former finding is broadly consistent with the novelty – routinization hypothesis by Goldberg and Costa (E. Goldberg, personal communication, 1/30/2010). They argued that the right hemisphere is critical for exploratory processing of novel cognitive situations for which no pre-existing cognitive representation is available. The left hemisphere, on the other hand, is responsible for executing well routinized cognitive strategies. Applying this notion here then, perhaps for skilled readers, reading is no longer a novel task and hence they are able to use more routinized strategies. With regard to the latter finding, Hoeft et al. postulated that perhaps reading is transiently dependent on the right fusiform before reading skills become reliant on the left hemisphere. Thus, dyslexics show an increase in activation in the right hemisphere and perhaps this reflects the fact that their brains have not been able to shift from right to more efficient left hemisphere structures.

Once thought to be purely a visual perceptual problem, clinicians and researchers now emphasize the role of phonological processing in dyslexia, which has been the focus of the paper thus far. However, in the zeal to relate phonological processing to reading it is important not to neglect the role that visual processing has in reading as well. For example, Graves, Frerichs, and Cook (1999) in their review of the research indicate multiple visual processing deficits associated with impaired reading. They state that dyslexic individuals have been shown to have prolonged visual persistence, decreased luminance contrast sensitivity, and lower flicker fusion thresholds. Dyslexics have also been shown to have abnormalities in the lateral geniculate nucleus on post-mortem. Additional studies cited by Graves et al. have found that: bilateral inferior parietal lobe lesions impair reading due to an inability to redirect eye direction, less skilled young readers are less accurate at localizing serial position of briefly flashed letters, and poor readers are less accurate in localizing briefly flashed letters or shapes especially the further they are from the fixation point. The finding that bilateral lesions of the inferior parietal lobe negative impact reading for visual processing reasons is particularly interesting since other researchers have discussed this area in the context of phonological processing and reading (e.g. Shaywitz and Shaywitz's notion that this area plays a role in translating grapheme into phoneme).

Graves et al. point out that visual input is predominantly from the magnocellular system, in which visual input travels from the retina through the Later Geniculate Nucleus to the cortex, including a significant feed into the parietal lobe. The magnocellular system is involved in visual – spatial localization. Accordingly, lesions in this area cause visual-spatial neglect, impaired visual attention, mislocalization of seen objects, and the inability to direct hands or eyes towards seen objects.

In a series of two experiments, using 12 non-reading impaired and 5 dyslexic and 9 non-reading impaired and 9 dyslexics, respectively, Graves et al. found that dyslexics were significantly more impaired at localizing visual stimuli in their peripheral field of vision compared to the non-reading impaired group. The difference was large, as they found effect sizes of approximately 1 standard deviation.

Graves et al. argue that their experimental condition simulates rapid reading where saccadic movements cause text to be briefly presented in periphery to which the visual system must automatically, unconsciously, and continually reorient. Specifically, the experimental conditions simulated localizing text 3 inches into one's periphery. Difficulty engaging in this operation, Graves et al. suggest, may cause dyslexics to have difficulty correctly registering words as they read. Graves et al. do not explicitly indicate the types of reading errors one would expect in such a case, but the current author believes the implication is that it could cause the omission of words, word sequencing errors, and possibly letter inversions (e.g. reading “flee” as “feel”).

Graves et al. argue that the dyslexics’ difficulty on this task implies involvement of the magnocellular system since this systems processes information that is presented in the periphery for brief periods and the parietal lobe, since this area performs visual-spatial localization based on input from the magnocellular system. Thus, the Graves et al. study, although somewhat old at this point, nonetheless is an example of how the role of visual processing in reading and reading disabilities needs to be acknowledged.

More recently, Richards, Berninger, Winn, Stock, Wagner, Muse, and Maravilla (2007), using fMRI scans, compared brain activation of 10 good readers with 20 dyslexic readers, and found that dyslexic readers were under-activated in the occipital lobe in Brodmann Area 19. Subjects in one condition received 8, 3 hour sessions of non-phonological based treatment, consisting of performing problem solving exercises in a virtual reality setting in which subjects had to find “Luna the Whale.” As is implied, the treatment was heavily visually based. Following treatment, these subjects showed an increase in activation in Brodmann Area 19, activating in this area up to the level shown by control subjects.

Richards et al. argue that this finding may reflect the influence of the magnocellular system in dyslexia, the former of which processes rapidly changing visual information and integrates visual information necessary to move around in the environment. With regard to reading, they state that the magnocellular system may be involved in the "serial application of attention focus to individual letter positions, identities, and groupings within written words," a deficit of which may interfere with the "transformation of letters into phonemes…that is, orthographic-phonological mapping" (p. 739).

A recent example of behavioral research on the role of visual processing in reading comes from Badian (2005). Badian describes "visual dyslexia" which is characterized by letter reversals, letter order confusion, weak orthographic imagery, and difficulty reading "exception words." Exception words are words that do not follow normal phonetic patterns (e.g. the word "said") and hence require memorization to correctly decode.

Badian examined 208 children, aged 8 – 10 years, two-thirds of whom were male. She found that visual orthographic skill, operationalized here as the ability to discern letter and number reversals, accounted for significant independent variance in decoding of real words, decoding of pseudowords, and reading comprehension, with it accounting for the largest amount of variance in reading comprehension, presumably because committing reversals slowed down reading interfering with comprehension.

The existence of a sub-type of dyslexia involving primarily visual perceptual deficits is consistent with the authors own clinical experience. He has found it not uncommon to encounter children with marked decoding and phonics impairment in the presence of significant visual perceptual deficits, yet adequate phonological processing skills. For example, a recent client of the author's fit the above pattern and his decoding errors reflect a visual decoding approach, marred by visual perceptual dysfunction. For example, he read the nonsense word “ep” as “bit.” In this case he appeared to rotate and reverse the “p,” perceiving it as a “b,” read right to left, and inserted a word he knew began with “b.” One can infer the strong influence of visual processing in his decoding, since his decoding error is largely the byproduct of visual perceptual errors and the word he produced (“bit”) does not share any of the phonemes as the target word “ep.” Of note, as alluded to above, this child has mastered the alphabet and performed in the average range on multiple phonological processing tasks, namely tasks of phonological memory, sound blending, and elision. As another example, he read “as” as “say,” in which he again read right to left, inverting “a – s” into “s – a” and then produced a word he knew that begins with “s – a” (i.e. “say”). Again, we can infer a visual decoding approach since the word “say” was in part a reflection of a visual perceptual error of inverting the letters and the word “say” does not share any of the same phonemes as “as” (note that the “s” in “as” produces a /z/ and not an /s/ sound). Thus, the current author’s own clinical experience is consistent with research showing the role of the visual processing system in certain dyslexic children.

Lastly, although not given as much attention as other brain regions, research suggests that the cerebellum has a role in reading. Frost et al. found the cerebellum to be active when subjects were processing speech and print and Landi et al. found that subjects with a reading disability showed less activation in the cerebellum compared to control subjects. The exact role the cerebellum plays is not known, but the current author speculates that perhaps it plays a role in coordinating the eye movements necessary for reading or is somehow involved in the timing necessary to read with fluency. In terms of the latter, fluent readers decode by grouping the words into phrases and reading phrase by phrase (called "scoo in a rhythmic fashion. Perhaps the cerebellum helps with the timing needed to do that.

In conclusion, although research has identified many brain areas associated with dyslexia, several primary areas have emerged as particularly salient. Dyslexics show an under-activation in the left temporal lobe, particularly the superior temporal gyrus, an area important for applying phonetic decoding strategies. Research suggests that the left superior temporal gyrus and left inferior frontal gyrus are involved in decoding sight words when phonological processing skill is low, but as phonological processing skill develops they switch to processing phonetic decoding. The superior temporal gyrus's transition to phonetic decoding is especially sensitive to the elision aspect of phonological processing.

Dyslexics also demonstrate an under-activation in the left occipitotemporal region, an area responsible for rapid word recognition, or "sight words." Dyslexics appear to compensate for these difficulties, one manifestation of which is an increased activation in the frontal lobes, especially the left inferior frontal gyrus. This may reflect a reliance on the speech system to decode in the absence of properly functioning posterior regions. Dyslexics also show increased activation of right hemisphere regions, including those in the right temporal lobes. It may be the case that reading temporarily relies on right hemisphere regions in normal development before switching to the more efficient left hemisphere regions. Dyslexics' hyper-activation of the right hemisphere regions during reading may thus reflect a lower stage of development, as their brains have not switched over to the right hemisphere for reading functions.

Research has shown dyslexics to have abnormal brain activation for semantic tasks as well as phonological tasks. This has lead to the proposition that dyslexia may reflect a more broad based linguistic processing deficit.

Although the emphasis in the literature has been on the role of phonological processing in reading, the research also suggests the involvement of visual processing systems as well. In particular a dysfunction in the magnocellular system may interfere with dyslexics' ability to properly orient and sequence letters and words.

Lastly, although not given as much attention in the literature as the above areas, the cerebellum has been identified as possibly playing a role in reading. Future research should examine its possible contribution to dyslexia.

References

Badian, N. A. (2005). Does a visual-orthographic deficit contribute to reading disability? *Annals of Dyslexia, 55 (1)*, 28-52.

Frost, S. J., Landi, N., Mencl, W. E., Sandak, R., Fulbright, R. K., Tejada, E. T., Jacobsen, L., Grigorenko, E. L., Constable, R. T., & Pugh, K. R. (20 . Phonological awareness predicts activation patterns for print and speech. *Annals of Dyslexia, 59*, 78-97.

Graves, R. E., Frerichs, R. J., & Cook, J. A. (1999). Visual localization in dyslexia. *Neuropsychology, 13 (4)*. 575-581.

Hoeft, F., Ueno, T., Reiss, A. L., Meyler, A., Whitfield-Gabrieli, S., Glover, G. H., Keller, T. A., Kobayashi, N., Mazaika, P., Jo, B., Just, M. A., & Gabrieli, J. D. E. (2007). Prediction of children's reading skills using behavioral, functional, and structural neuroimaging measures. *Behavioral Neuroscience, 121 (3)*. 602-613.

International Dyslexia Association. (2008). *Dyslexia basics.* [Fact Sheet]. Baltimore, MD: Author.

Landi, N., Mencl, W. E., Frost, S. J., Sandak, R., Pugh, K. R. (2010). An fMRI study of multimodal semantic and phonological processing in reading disabled adolescents. *Annals of Dyslexia, 60 (1)*. Retrieved 2/17/2010, from www.springerlink.com.

McCrory, E. J., Mechelli, A., Frith, U., & Price, C. J. (2004). More than words: a common neural basis for reading and naming deficits in developmental dyslexia? *Brain, 128 (2),* 261-267.

Richards, T., Berninger, V., Winn, W., Stock, P., Wagner, R., Muse, A., & Maravilla, K. (2007). *Neuropsychology, 21 (6),* 732-741.

Shaywitz, S. E. & Shaywitz, B. A. (2001). The neurobiology of reading and dyslexia. *Focus on Basics Connecting Research and Practice 5 (A),* 11-15.