Neural Correlates of Early-Stage Visual Processing Differences in Developmental Dyslexia

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ABSTRACT

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Reading requires the successful recruitment and coordination of brain networks to translate visual symbols into phonemes, which are then sequenced to match speech sounds and matched onto semantic representations. Although phonemic awareness is understood to be a core deficit associated with reading disability, neuroimaging has demonstrated an association between poor reading and disruption to various interrelated areas in the brain. This includes one of the major visual pathways, the magnocellular pathway, which contributes to the dorsal pathway in the brain and the processing of motion. For at least two decades, researchers have observed differences in motion processing, supported by the magnocellular pathway, between individuals with and without dyslexia (Eden et al., 1996; Gori et al., 2016; Livingstone et al., 1991; Wilmer, 2004). Further, psychometric studies report an association between reading ability and dorsal stream sensitivity in adults and in children before and after learning to read (Boets et al., 2011; Kevan & Pammer, 2009). Studies of the development of the major visual pathways have suggested that the magnocellular pathway follows a protracted course of development, which raises the possibility that it is vulnerable to pathological change during development and also has the potential for greater plasticity (Armstrong et al., 2002; Stevens & Neville, 2006).

To explore the potential differences in early-stage visual processing, this dissertation study investigated whether neurophysiological measures, as indexed by event-related potentials (ERP), may differ between adults with and without dyslexia to stimuli tailored to evoke a response from each of two major visual pathways: magnocellular and parvocellular. The P1 component was elicited in response to motion stimuli designed to probe magnocellular pathways,

and the N1 component was elicited in response to color stimuli designed for parvocellular processing. Group comparisons revealed statistically significant group differences in P1 amplitude for the motion/magnocellular condition, but no differences were found for N1 ERP measures for the parvocellular/color condition. Moderate to strong correlations between P1 measures in response to the magnocellular/motion condition were observed in relation to specific behavioral assessments: nonverbal reasoning and memory, orthographic choice, the word identification subtest from the Woodcock Reading Mastery Test (3rd edition: WRMT-III, Woodcock, 2011), and the sight word efficiency subtest from the Test of Word Reading Efficiency (2nd edition: TOWRE-2, Wagner, Torgesen, & Rashotte, 2011).

These results are indicative of an early-stage visual processing disruption in individuals with dyslexia observable at the level of the brain. Due to the compounding impact of even small disruptions of sensory and cognitive processing on learning, refining our knowledge of the underlying neural mechanisms of reading may permit earlier identification and potentially more focused interventions that could yield better outcomes for struggling readers. Additionally, the association of those differences with measures of word decoding will inform further research into the underlying neural mechanisms that may contribute to dyslexia and skilled reading.

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DEDICATION

"Biology gives you a brain. Life turns it into a mind."
Jeffrey Eugenides (American Pulitzer Prize-winning novelist; 1960 -)

For me, the biology started with my mom and dad, Janet E. Rosenthal and Robert E. Paul, who were also fairly influential in nurturing my mind. I am forever grateful for the time we shared and miss them every day. I am grateful to them for cultivating my curiosity, unconditional love, and emphasizing the importance of searching out the things in life that fill it with meaning.

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While I am not sure anyone in my family or my friends understands why I did this, or even exactly what I did, I dedicate this colossal amount of work to you all, because without you, I would just be a blob of biology.

1. INTRODUCTION

1.1 Developmental Dyslexia

The heterogeneous nature of dyslexia and lack of consensus regarding the underlying cause(s) have resulted in a definition of this developmental disorder that focuses on symptoms. This complicates both early identification of those with the disorder and provision of effective interventions for affected individuals. A definition of dyslexia was proposed by the Board of Directors of the International Dyslexia Association (IDA) and adopted by the National Institute of Children's Health and Human Development (NICHD) in 1994 for use in research. In 2002, it was revised and revisited in 2016 at the IDA conference (Dickmen, 2017); however, the definition from 2002 stands and states:

> Dyslexia is a specific learning disability that is neurobiological in origin. It is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction. Secondary consequences may include problems in reading comprehension and reduced reading experience that can impede growth of vocabulary and background knowledge. (https://dyslexiaida.org/definition-of-dyslexia/)

Similarly, the *Diagnostic and Statistical Manual of Mental Disorders—Fifth Edition* (*DSM-V*) (American Psychiatric Association, 2013) also indicates that the term *dyslexia* is "used to refer to a pattern of learning difficulties characterized by problems with accurate or fluent word recognition, poor decoding, and poor spelling abilities" (p. 37).

The *DSM-V* does not attribute observed symptoms of dyslexia to specific causes beyond a neurodevelopmental basis, whereas IDA and NICHD's definitions attribute the symptoms to deficits in the phonological component of language and consider the consequences of such deficits on academic skill growth. While these kinds of clinical definitions serve the primary

purpose of identification for remediation, the symptoms identified may have multiple causes. Additionally, both definitions either indirectly or directly indicate that a child must be performing below expectations or poorly relative to peers. One of the goals of identification and remediation should be the identification of at-risk children before they have experienced reading failure. This would minimize the consequences of such failures, which often include poor self-esteem and low academic confidence resulting from limited reading experience and access to curriculum (Humphrey & Mullins, 2002; Ingesson, 2007; Ridsdale, 2005; Stanovich, 1986).

Word level reading is a complex behavior, sub-served by many sensory processes and cognitive skills (see Schlaggar & McCandliss, 2007; Wandell, Rauschecker, & Yeatman, 2012). According to Peterson and Pennington (2012) and Shaywitz, Escobar, Shaywitz, Fletcher, and Makuch (1992), reading ability exists on a continuum such that individuals with dyslexia constitute the lower end of a normal distribution. To better understand reading and reading disability towards the goal of a fully literate citizenry, it is essential to continue to build our collective knowledge of the underlying neural mechanisms that support reading.

1.2 Prevalence

Prevalence rates for dyslexia vary, largely due to how the disorder is defined as well as the criteria and purposes of identification (clinical or research). It is estimated that 13-14% of students nationwide qualify for special education services; of those students, half are classified with a learning disability; and 85% of those have a primary disability related to language/reading processing (Moats & Dakin, 2008). Up to 15-20% of the U.S. population is thought to struggle with slow reading speed, poor spelling, and poor writing (Moats & Dakin, 2008). Shaywitz and Shaywitz (2005) suggest that, from an epidemiological perspective, dyslexia fits a dimensional model, such that reading ability and disability plot along a normal distribution within the

population, with the lower end of the continuum representing those who struggle to learn to read. Prevalence is, therefore, hard to measure as there is limited understanding of how individual differences interact with formal education (Butterworth & Kovas, 2013). Estimates of the prevalence of dyslexia are calculated based on a variety of sources including epidemiological studies, survey, child count, and research, often varying state to state and sometimes school to school (Interagency Committee on Learning Disability, 2011). The typical prevalence figure reported is between 5-17% (Shaywitz et al., 1998); however, without consistent criteria for identification, reported prevalence rates can be challenged.

1.3 Characteristics/Subtypes of Dyslexia

Over 20 years of research have made it clear that dyslexia is not a unitary phenomenon. Accordingly, a variety of skills are evaluated to identify children with dyslexia. These include measures of phonological ability (phonemic awareness, phonic decoding); fluency (slow, error-prone reading aloud); processing speed (rapid automatized naming); orthographic skills (misspellings, distinguishing legal and illegal letter strings); working memory (nonword repetition, memory span tasks); and language skills (vocabulary, syntax, stress, and intonation when reading aloud) (Seidenberg, 2017).

Scores on specific assessments tend to reveal patterns of discrepancies between verbal skills and reading comprehension, or difficulties in both areas, and frequently also involve challenges in focusing and attending (Kilpatrick, 2015). While phonological awareness and phonic decoding are typically amenable to intervention (e.g., Bhat, Griffin, & Sindelar, 2003; Truch, 1994), problems with rapid automatized naming and phonological working memory are not usually directly remediated and are associated with poor outcomes (Kilpatrick, 2015).

Elliott and Grigorenko (2014) pointed out that many additional symptoms or characteristics of dyslexia have been described in the research literature, including poor verbal memory, ordering/sequencing, sense of rhythm, concentration, and phonic skills. Individuals with dyslexia are frequently identified as having impairments in rapid information processing and verbal fluency as well as difficulties with speech, language, and mental calculations. Inconsistent hand preference and letter reversals are also associated with dyslexia (Elliot & Grigorenko, 2014). The research on reading and reading disability covers a range of cognitive processes that have been used to index the problems associated with dyslexia. Table 1 (following page) highlights some of this research, which seeks to understand how a number of underlying cognitive processes might contribute to dyslexia.

The heterogeneity observed in dyslexia can be explored from a top-down, hypothesis-driven perspective, where subtypes are identified through the collection of standardized assessments that compare individuals with and without dyslexia and provide support for theoretical models of reading disability (King, Giess, & Lombardino, 2007). It is also possible to take a bottom-up, data-driven approach that looks at collected data to identify clusters that account for the participants with dyslexia. Using a resampling and gap statistic, King et al. (2007) identified four subtypes of dyslexia from standardized measures of phonological awareness, rapid naming, phonological memory, word attack, word identification, spelling, passage comprehension, and verbal ability. Data were collected from 93 children serving as controls, matched on gender and age to 93 children with developmental dyslexia. Participants were 7-16 years of age and all had similar nonverbal intelligence scores. Children without developmental dyslexia did not reveal any significant patterns of performance on these assessments; however, for the children with dyslexia, almost 40% (35/93) demonstrated a pattern

Table 1

Cognitive Processes Associated With Dyslexia in the Literature

Cognitive Processes	Study Authors/Year/Findings
Attention	**Bosse, Tainturier, & Valdois (20070** Visual attention span plays a role in identification/parsing of relevant sub-lexical orthographic units; contributes to reading performance independently of phonological skills in French children in primary school.
	Stoet, Markey, & López (2007) Shifting attention impaired at the perceptual level, not at the level of central executive cognitive system.
	Lallier, Thierry, Tainturier, Donnadieu, Peyrin, Billard, & Valdois (2009) "Sluggish attentional shifting" (SAS) associated with phonological processing deficits. Regardless of age or language (French/English), individuals with dyslexia demonstrated SAS in auditory modality; SAS in visual system only observed in English adults.
Auditory and Visual Processing	**Schulte-Körne & Bruder (2010)** Review of basic auditory and visual processing differences in dyslexia evident across lifespan. Speech processing deficits observed for all ages, affecting spectral/temporal transitions in both active and passive paradigms. Comparison of studies of visual processing complicated by variability across designs. Differences observed in functioning of fast processing transient pathway—magnocellular pathway—likely contributing to visual attention/visuospatial attention.
Automatization	**Nicolson, Fawcett, Brookes, & Needle (2010)** Speeded single-word reading, long-term response learning, and overnight skill consolidation; studies reviewed concluded procedural learning takes longer in some children with dyslexia. Propose skill automatization, supported by the corticocerebellar language-based procedural learning system, is the primary neural system affected, with additional issues present in some children.
Short-term Memory	**De Jong (1998)** Working memory (WM) deficits not limited to language tasks and general WM deficits not attributable to processing efficiency or verbal memory span deficits. Individuals with dyslexia exhibit a general WM deficit for parallel processing/storage of verbal information.
	Maehler & Schuchardt (2016) Findings suggested differing working memory deficit patterns across learning disabilities: dyslexia associated with deficits in the phonological loop; dyscalculia with deficits in visual-spatial sketchpad; ADHD with deficits in the central executive. Co-occurring disorders lead to additive working memory deficits.
	Majerus & Cowan (2016) Mini-review of short-term memory (STM) deficits in dyslexia. All but one study found verbal STM deficits persist into adulthood. Serial order STM impairment appears to occur for the retention of both verbal and visuospatial sequence information. Serial order STM impairment not observed in all participants with dyslexia and is not specific to dyslexia.
Speed of Processing	**Breznitz & Meyler (2003)** Explored speed of processing (SOP) in nonlinguistic and sub-lexical linguistic auditory and visual oddball tasks using EEG. In each modality and with cross-modal tasks, visual processing was slower than auditory. Individuals with dyslexia showed slower SOP to low-probability stimuli (oddballs) than controls. SOP is most evident at the processing stage associated with working memory. Proposed deficit in SOP may affect ability to integrate information.
Rapid Naming	**Norton & Wolf (2012)** Rapid automatized naming (RAN) thought to index the ability to integral multiple neural processes. Phonological awareness and RAN are both strong predictors of reading ability and distinct constructs.

that included both phonological and rate scores one standard deviation below the mean, whereas 26% (19/93) demonstrated results that placed them either in a phonological or rate-depressed cluster or subtype (King et al., 2007). King et al. noted that standardized measures limit what subtypes can emerge and that other subtypes are very likely to exist.

Both prospective and longitudinal studies have helped to describe the phenotype associated with dyslexia—that is, the individual observable characteristics thought to be a product of interactions between genes and the environment. Pennington (2006), Menghini et al., (2010), and Di Filippo and Zoccolotti (2012) all considered a multi-cognitive deficit model to help account for factors like co-occurring disorders and individual differences. Based on their exploratory and confirmatory factor analyses of the potential underlying cognitive features of dyslexia, Tamboer, Vorst, and Oort (2016) put forward a multi-cognitive deficit model of dyslexia, suggesting such a conceptualization was more appropriate than framing dyslexia as a disorder resulting from one deficit with various behavioral outcomes, or as co-occurring disorders with similar symptoms. Tamboer et al. (2016) recruited 446 psychology students, aged 17-25 years (114 males), from Amsterdam University in the Netherlands. Based on the consistency between two independent methods of identification, a process intended to reduce selection bias, the researchers established two study groups: individuals with dyslexia (n = 63; 14%) and individuals without dyslexia (n = 345; 77%). Thirty-eight students (9%) could not be grouped due to inconsistencies between the two identification methods. Using principal component analysis, Tamboer et al. found that five factors of dyslexia (spelling, phonology, short-term memory, rhyme/confusion, and whole-word processing/complexity) accounted for 60% of the variance in scores on standardized assessments of cognitive and linguistic processing. While they were not able to identify subgroups, Tamboer et al. found that spelling deficits were

the most common problem, seen in over 78% of the individuals with dyslexia. However, the severity of dyslexia was better predicted by a combination of factors rather than just this one most frequently observed deficit (Tamboer et al., 2016).

Table 2 below highlights the signs of dyslexia by developmental phase (Rose, 2009) and indicates how the characteristics tend to change across development.

Table 2

Dyslexia by Developmental Phase

Developmental Phase	Signs of Dyslexia
Preschool	Delayed or problematic speech Poor expressive language Poor rhyming skills Little interest/difficulty learning letters
Early School Years	Poor letter-sound knowledge Poor phoneme awareness Idiosyncratic spelling Problems copying
Middle School Years	Slow reading Poor decoding skills when faced with new words Phonetic or non-phonetic spelling
Adolescence and Adulthood	Poor reading fluency Slow speed of writing Poor organization and expression in work

Adapted from the Rose Report with permission from Snowling (2008a). Developmental phases and associated characteristics of dyslexia.

Gallagher, Frith, and Snowling (2000) explored the phenotypes associated with dyslexia by recruiting 63 children from families with a history of dyslexia, and an additional 34 children from families with no history of dyslexia, for a longitudinal study that assessed participants at two time points: 3.9 and 6 years of age. Groups were matched for maternal education, SES, age, and sex. Children were grouped based on assessments at the first-time point into at-risk impaired,

at-risk unimpaired, and control groups. At-risk impaired children revealed widespread language delay (receptive/expressive language skills plus vocabulary), whereas the profiles of the at-risk unimpaired and control children were very similar to one another, with the exception of scores on a non-word repetition task that was similar between both at-risk groups. At the second time point (6 years of age), the at-risk impaired group continued to demonstrate weakness across multiple measures. While the at-risk unimpaired group performed better than the at-risk impaired group, the at-risk unimpaired group scores were weaker than the controls. A re-evaluation of the same children at a third time point, 8 years of age, revealed that 66% of the children identified as at-risk scored more than 1.5 standard deviations below the control group on reading measures but not on measures of nonverbal ability (Snowling, Gallagher, & Frith, 2003). These findings suggested an increased risk of literacy problems in children presenting with risk factors, including having at least one parent with dyslexia. That risk extends beyond phonological deficits to behavioral outcomes related to language skills (Snowling, 2000). The final phase of this study (Snowling, Muter, & Carroll, 2007) included 50 at-risk students and 20 control students, 12 to 13 years of age, all of whom had participated in the earlier studies (Snowling, 2000; Snowling et al., 2003). While all students in the at-risk group (impaired and unimpaired) made reading and spelling gains, 42% of the at-risk impaired group scored within range for reading and spelling disability, demonstrating persistent literacy problems. The observations made across studies (Snowling, 2000; Snowling et al., 2003; Snowling et al., 2007) pointed to the interrelationships between reading, language, and attention as well as between genetic and environmental influences, resulting in differing outcomes.

To understand the broader phenotype of dyslexia observed in these studies involving groups of at-risk children, Snowling (2008b) considered how relative strengths and weaknesses

of specific cognitive skills contribute to reading skill across participating individuals. Composite scores for phonological abilities, visuospatial skills, attention control, and oral language were compiled for 48 at-risk students (20 at-risk impaired/28 at-risk unimpaired) and controls. The at-risk impaired students were further defined as those with reading and spelling impairments and those with only spelling impairments. A negative correlation between literacy skills and number of deficits was observed, and the at-risk impaired group was observed to be more likely to have multiple deficits (Snowling, 2008b). The most frequently observed issues in the at-risk impaired group were phonological and attentional deficits; within this sample (n = 20), just one case presented with only a phonological deficit. Of those at-risk but unimpaired, attentional control was the most frequently observed deficit (9 out of 28 cases). Hence, while a child meeting the diagnostic threshold for dyslexia is more likely to have a phonological deficit, the severity of the disorder may be associated with more than one cognitive deficit (Snowling, 2008b).

1.4 Co-Occurring Developmental Disorders

The co-occurrence of neurodevelopmental disorders identified by the *DSM-V* is more "the rule rather than the exception" (Boada, Willcutt, & Pennington, 2012, p. 274). Studies of individuals diagnosed with attention deficit hyperactivity disorder (ADHD) have noted that 70-90% of individuals meeting criteria for ADHD will also meet criteria for an additional co-occurring disorder (Faraone, Biederman, Weber, & Russell, 1998; Willcutt, Pennington, Chhabildas, Friedman, & Alexander, 1999). The rate of co-occurrence between specific learning disability (SLD) and ADHD—and in particular dyslexia, a sub-type of SLD—is as high as 25 to 40%, which suggests co-occurrence is greater than chance (Willcutt & Pennington, 2000). Dyslexia frequently co-occurs with other disorders in addition to ADHD, such as specific

language impairment (SLI) and speech sound disorder (SSD) (Peterson & Pennington, 2012). While many theories of language impairment, speech sound disorder, and reading disability all identify phonological deficits as an underlying cause, each of these disorders varies in its phenotypes (see Peterson & Pennington, 2012). Evidence for interactions involving multiple cognitive deficits has led to the articulation of the "multiple overlapping risk factors" model (Pennington & Bishop, 2009, p. 301). This model predicts that a single cognitive deficit, such as a phonological deficit, is not sufficient to cause a disorder. Rather, risk factors both specific to and shared between disorders result in phenotypes recognized as reading disability, specific language impairment, or speech sound disorder (Pennington & Bishop, 2009).

1.5 Evidence Across Languages

While dyslexia exists across all languages, its characteristics are thought to differ in their impact on reading development based on the transparency[1] of the orthography (Peterson & Pennington, 2012). English is considered an opaque orthography due to the many correspondences between a single letter, or a specific letter combination, and associated sounds (grapheme/phoneme correspondence). This contrasts with transparent orthographies, such as Finnish, where letters are consistently pronounced the same across contexts. Cross-cultural findings suggested that for alphabetic languages with more transparent orthographies, such as German, difficulties with reading accuracy tend to be less severe than in opaque languages such as English (Landerl, Wimmer, & Frith, 1997). Caravolas, Volín, and Hulme (2005) compared phonemic awareness in readers of Czech, a transparent language, and English, an opaque language. Their findings suggested that in both Czech and English, alphabetic languages with

[1] The transparency of an orthography is based on the consistency (transparent) or the inconsistency (opaque) of the letter/sound mapping. For example, in English, considered to be an opaque orthography, the letter "a" can be pronounced multiple ways such as in *apple*, *was*, *made*, and *far* (Ziegler et al., 2010).

differing degrees of orthographic transparency, phonemic awareness is a strong predictor of reading ability.

Chinese orthographies use characters associated with monosyllabic morphemes. Morphemes differ from phonemes in that a morpheme represents the smallest meaningful parts of words, whereas a phoneme is simply a distinct sound in a language represented by a grapheme (character/letter). Most characters in Chinese orthography are compounds, with one component (radical) conveying the meaning of the word and the other component (phonetic) providing a cue for pronunciation (Hanley, 2005). Eighty percent of contemporary Chinese characters are compounds containing radicals and phonetics, so the majority of characters convey both lexical-semantic and phonetic information (Hanley, 2005). Research on dyslexia in Chinese students is limited, though a few studies (Chan & Seigel, 2001; Ho & Ma, 1999) have suggested that Chinese children with dyslexia have less difficulty with regular characters (consistent phonetic cues) than with irregular or low-frequency characters. However, their performance on semantic tasks is significantly worse compared with age-matched controls, yet similar to younger readers matched for reading level (Hanley, 2005). According to Chan and Siegel (2001), early reading in Chinese depends more on visual skills, whereas phonological processing becomes a crucial skill as students continue through school.

Across languages, the orthographic transparency of a script does seem to affect the rate of reading acquisition for all readers, and for those with dyslexia, irregular features present obstacles to fluent reading (Caravolas, 2005). According to Caravolas (2005), evidence points to several underlying cognitive deficits common across languages in dyslexia, including phonological awareness, verbal short-term memory, and rapid automatic naming speed (RAN).

Cross-cultural brain imaging studies have suggested that, although dyslexia may present differently across languages, the same brain regions—the left middle, inferior, and superior temporal cortex as well as the middle occipital gyrus—reveal differences between readers with and without dyslexia, regardless of orthography (Paulesu, Danelli, & Berlingeri, 2001; Silani et al., 2005). Using positron emission tomography (PET), Paulesu et al. (2001) explored the relationship between various measures of literacy-related skill in adults with and without dyslexia who were readers of opaque (English and French) and transparent (Italian) languages. They compared measures from the Wechsler intelligence test scale (WAIS) for adults and other reading and phonology tasks. Individuals with dyslexia across languages performed significantly worse than controls on subtests of the WAIS such as digit span, arithmetic, and digit symbol manipulation; however, on other measures not associated with phonological short-term memory, performance was similar between readers with and without dyslexia. On measures of phonology and reading, speakers of Italian with dyslexia outperformed speakers of English or French with dyslexia, making fewer errors on words and nonwords. All individuals with dyslexia performed significantly worse than non-dyslexic readers, regardless of language, on measures of reading and phonology. The PET findings demonstrated reduced activation in areas strongly associated with word reading, such as the perisylvian cortex, the left middle and inferior temporal gyri, and the fusiform gyrus, in individuals with dyslexia across languages as compared to controls. These findings provided evidence for a common neuroanatomical basis for dyslexia, despite differences in behavioral outcomes between transparent and opaque orthographies.

Silani et al. (2005) extended the findings from Paulesu et al. (2001) by conducting a voxel-based morphometry (VBM) analysis to determine potential differences in grey and white matter density in areas previously associated with reduced activation in individuals with dyslexia

in English, French, and Italian. Findings suggested a structural cortical disorganization associated with dyslexia, including areas with reduced grey matter (e.g., left middle temporal region) and areas with increased grey matter densities (including posterior temporal and inferior temporal cortices). Individuals with dyslexia from all three language backgrounds showed this same pattern and additionally showed a reduction in white matter density in the arcuate fasciculus. Silani et al. (2005) speculated that, while the areas observed to differ between individuals with and without dyslexia across the three languages studied are associated with phonological processing and decoding, the findings suggested a disruption to local regions and connectivity within the language network. This potentially relates to the integration of visual, phonological, and articulatory information required for reading (Silani et al., 2005).

1.6 Making Sense of Dyslexia

Stein (2001) suggested that individuals with dyslexia have atypical brains. A recent Norwegian longitudinal structural MRI study compared seven children with dyslexia and 10 children without dyslexia, following them from pre-literacy to 11 years of age, when in some instances a diagnosis of dyslexia was made (Clark et al., 2014). The study revealed neuroanatomical abnormalities in pre-readers, not in the reading network but in regions supporting auditory and visual processing (Clark et al., 2014). The Interagency Committee on Learning Disability (2011) stated there is a basic consensus that learning disabilities are attributable to atypical cognitive and linguistic processes. Where consensus breaks down is in the assessment, identification, and nature of specific learning disabilities.

Reading is a complex cognitive task, one that engages multiple sensory and cognitive systems. The current consensus in favor of the phonological deficit theory of dyslexia is the product of research undertaken during the 1980s, derived from replicated findings that children

with dyslexia struggle to process individual sounds, the constituent components of words (Nicolson, Fawcett, Brookes, & Needle, 2010). Advances in brain imaging, including electroencephalography (EEG), positron emission tomography (PET), magnetic resonance imaging (MRI), functional magnetic resonance imaging (fMRI), magnetoencephalography (MEG), diffusion tensor imaging (DTI), and near-infrared spectroscopy (NIRS), have added significantly to our understanding of the structural and functional factors contributing to the development of a functional neural network for reading as well as observed differences in the reading networks of individuals with dyslexia. However, it has proven difficult to disambiguate the underlying mechanisms that contribute to reading disability from possible factors resulting from differential reading experiences (Olulade, Napoliello, & Eden, 2013). This makes it more difficult to reach an understanding of reading disability in terms of the distinctions between potential causes, observed symptoms, and possible treatments (Nicolson et al., 2008). Furthermore, multiple neurological substrates could likely contribute to dyslexia and observed symptoms, suggesting there are potential subtypes that correspond to different abnormalities within the brain systems supporting reading. The fundamental challenge for researchers across multiple disciplines is to construct an explanatory account of the contributing sensory and cognitive systems that can contribute to reading disability and different disability profiles within a developmental neurobiological framework.

Against this background, the goals of this dissertation study can be summarized as follows: (a) to explore the neural correlates of early-stage visual processing differences in developmental dyslexia, possibly indexed by EEG event-related potential (ERP) components; and (b) to investigate whether such ERP components are associated with behavioral measures of orthographic skill as well as other key sub-skills related to reading. The intent is to contribute to

the growing body of knowledge suggesting that differences in visual information processing may contribute to reading difficulties (e.g., Lawton, 2016).

This dissertation is organized as follows: Chapter 2 provides an overview of the anatomy of the reading brain, the neurobiology of reading, and theoretical models. Chapter 3 provides the research questions and hypotheses for the dissertation study, followed by Chapter 4, which details the methodology. Chapter 5 outlines the data recording procedures, pre- and post-processing parameters, as well as the data analysis strategy. Chapter 6 summarizes results from the study. Finally, Chapter 7 discusses the findings within the context of the current research and provides thoughts on future directions as well as study limitations and delimitations.

2. THE READING BRAIN

Many factors contribute to academic skill development. Fletcher et al. (2009) provided a framework for understanding the potential interactions between factors (see Figure 1 below). This framework illustrates the complexity involved in disentangling the source(s) of observed academic skill deficits. Implicit in this framework is the dynamic nature of development. The constellation of brain systems that eventually come to support efficient reading did not evolve for the purpose of reading. Dehaene's (2009) Neuronal Recycling Hypothesis suggested that neuronal networks that evolved to accomplish other essential skills, such as language and object recognition, are co-opted through instruction to develop an additional functional system, one that recognizes written symbols in such a way that language can be perceived through a visual medium (Dehaene, 2009).

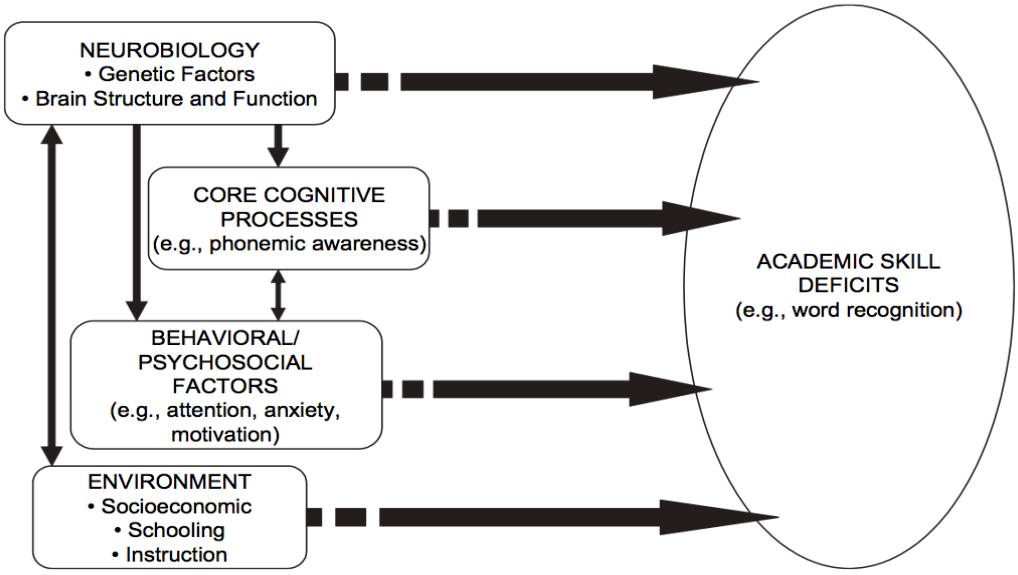

Figure 1. Influences on academic deficits
Reprinted with permission of Guilford Press (Fletcher et al., 2007, p. 3)

Each brain is the product of biological constraints, including individual genetics and how those "instructions" unfold during development in response to environmental factors, reflecting neuroplasticity (Berninger & Richards, 2002). In this section, we will consider the anatomy of the brain in relation to models of language and reading; brain development and neuroplasticity; the neurobiology and theories of dyslexia; and finally, how the vision system contributes to reading.

2.1 Anatomy of the Reading Brain

Language is typically accessed through the auditory system; reading, by contrast, typically involves accessing language through the visual system (Richardson & Price, 2009). Word recognition in skilled readers engages the inferior occipito-temporal and fusiform areas extending anteriorly in the middle and inferior temporal gyri (ventral network), the temporo-parietal area including the angular gyrus and suprmarginal gyrus (dorsal network), and posterior portions of the inferior frontal gyrus (anterior network), all primarily in the left hemisphere of the brain (Sandak, Mencl, Frost, & Pugh, 2004). These networks each play a unique role in skilled reading. The ventral system, in support of reading, develops slowly (Shaywitz et al., 2002), with the more posterior areas thought to function in a pre-semantic way (visual word form area; Cohen et al., 2002) while the more anterior parts of the ventral system (middle and inferior temporal gyri) are tuned for semantics (Sandak et al., 2004). The dorsal system is thought to be responsible for mapping language symbols (graphemes) onto the corresponding phonological and semantic components of language (Borowsky et al., 2006). More generally, its role seems to involve "attentionally controlled processing" (Sandak et al., 2004, p. 275). The anterior network, often engaged when reading low-frequency rather than

high-frequency words (Fiedback et al., 2002), is thought to support reading through phonological recoding (Sandak et al., 2004).

This overview focuses on word-level reading since this is a prerequisite for fluent reading. We begin with a brief overview of the language system on which the reading system develops.

The Broca-Wernicke-Lichtheim-Geschwind model (see Figure 2 below) was the first neurobiological model of language, based on 19th century lesion studies conducted by Carl Wernicke and Paul Broca, which provided evidence for a brain structure-function relationship (see Ben Shalom & Poeppel, 2008; Tremblay & Dick, 2016). Given the accumulation of research since, it has been argued that this "Classic model" needs to be expanded (e.g., Ardila, Bernal, & Rosselli, 2016; Ben Shalom & Poeppel, 2008; Gierhan, 2013; Poeppel, Emmorey, Hickok, & Pylkkänen, 2012; Tremblay & Dick, 2016).

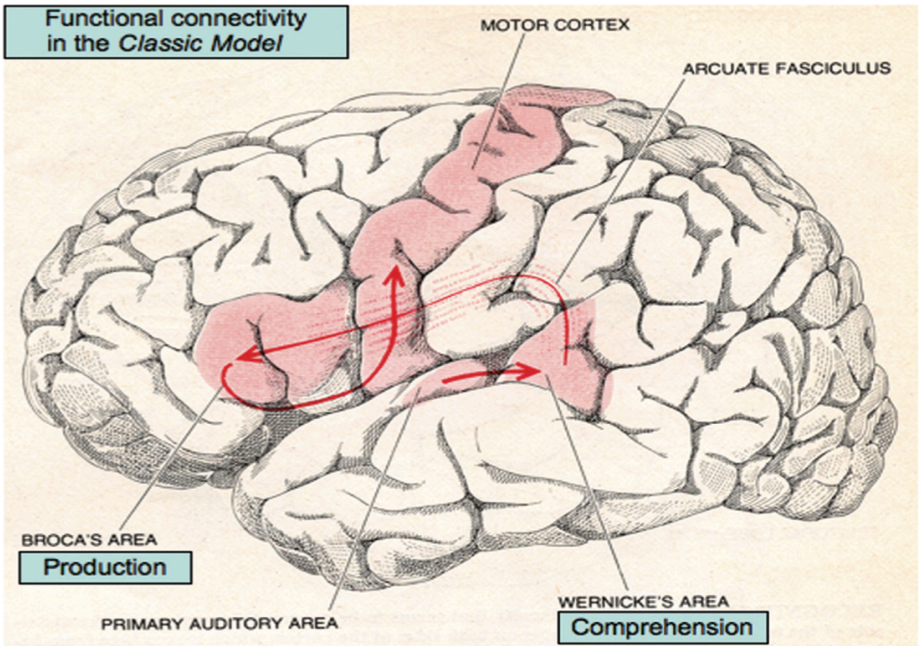

Figure 1. Classic language network model (Geschwind, 1979).
This diagram illustrates the very basic anatomical areas and functional roles of the language.
With permission from Ben Shalom & Poeppel (2008).

One approach has built on observations from sensory (specifically, visual) processing. Ungerleider and Mishkin (1982) proposed a framework, later modified by Goodale and Milner (1992), that described functional roles for visual processing that were sub-served by dorsal and ventral visual pathways. Language and reading tasks each drive different patterns of brain activation across a diffuse network of structures, a finding that has been replicated with a good degree of consistency (see Price, 2012). Brain circuitry for reading is superimposed on the brain circuitry for speech and language processing (see Figure 3 and Table 3 below for a summary overview) and includes three major circuits: left dorsal temporo-parietal, ventral occipital-temporal, and the inferior frontal circuit (e.g., Pugh et al., 2000). Within these systems, both lateralized to the left hemisphere, the phonological system has an anterior and a posterior component. The anterior component (inferior frontal gyrus/BA 44 and premotor cortex BA 6) is associated with speech production and analysis of phonological components of words (Martin, Schurz, Kronbichler, & Richlan, 2015; Seidenberg, 2017). The posterior component consists of the perisylvian region including the supramarginal gyrus (BA 40) and the angular gyrus (BA 39), part of the posterior superior temporal cortex, an area thought to support the integration of letters and sounds. The ventral circuits include the orthographic system, associated with the extrastriate area in the occipito-temporal cortex, frequently referred to as the visual word form area (VWFA), a region thought to process letter patterns as prelexical representations of words (Martin et al., 2015; Schlagger & McCandliss, 2007; Seidenberg, 2017). See Table 4 below for a summary of this classical view of reading circuits in the brain.

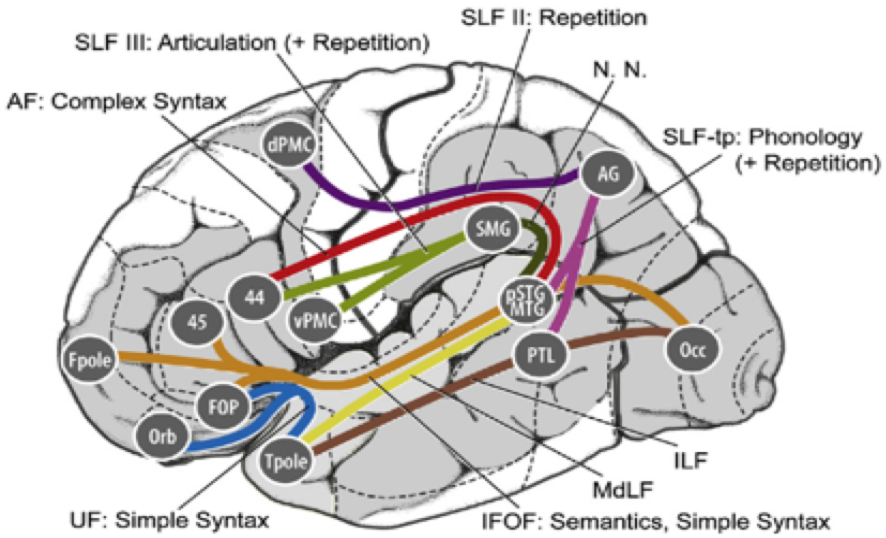

Figure 3. Neurocognitive model of neural pathways supporting auditory language in the brain, upon which networks for reading are superimposed.
With permission from Gierhan (2013). See Table 3 for explanation.

Table 3

Neurocognitive Model based on Gierhan (2013)

Likely Pathways of Fiber Tracts Associated With Language Processing		
Pathway	**Regions and Connections**	**Associated Aspects of Language Function**
Dorsal	BA 44 (Broca's Area) connects to the posterior Superior Temporal Gyrus/Middle Temporal Gyrus via **Arcuate Fasicicle (RED)**	Complex syntax
Dorsal	Angular Gyrus connects to posterior Superior Temporal Gyrus/Middle Temporal Gyrus and Posterior Temporal Lobe via **Superior Longitudinal Fasicicle I or temporo-parietal (PINK)**	Phonology and repetition
	Angular Gyrus connects to dorsal Pre-Motor Cortex via **Superior Longitudinal Fasicicle II (PURPLE)**	Repetition
Dorsal	Supramarginal Gyrus connects to BA 44 (Broca's Area) and ventral Pre-motor cortex via **Superior Longitudinal Fasicicle III (GREEN)**	Articulation and repetition
Ventral	Orbitofrontal cortex, Frontal Operculum, Temporal Pole are connected via the **Uncinate Fascicle (BLUE)**	Simple syntax
Ventral	Occipital Cortex connects with Posterior Temporal Lobe and Temporal Pole via the **Inferior Longitudinal Fascicle (BROWN)**	
Ventral	Posterior Superior Temporal Gyrus/Middle Temporal Gyrus connects to Temporal Pole via **Middle Longitudinal Fascicle (MUSTARD)**	
Ventral	Occipital Cortex connects with BA 45, Frontal Pole, Orbitofrontal cortex, Frontal Operculum via **Inferior Fronto-occipital Fascicle (IFOF—TAN)**	Semantics, simple syntax
	Supramarginal Gyrus connects to the Posterior Superior Temporal Gyrus/Middle Temporal Gyrus via **an unnamed tract (DARK GREEN)**	

Table 4

Classical View of Reading Circuits in the Brain based on Pugh et al. (2000)

Neural Circuit – Left Hemisphere	Areas Involved	Function
Dorsal temporo-parietal circuit	Wernicke's area: ▪ Posterior superior temporal gyrus ▪ Supramarginal gyrus ▪ Angular gyrus	*Cross-modal integration – visual text and phonological structure of language.*
Ventral occipito-temporal circuit	Lateral extrastriate areas Fusiform gyrus Inferior occipito-temporal region ▪ Visual Word Form Area	*Word identification system – automatic process – late developing.*
Anterior inferior frontal circuit	Broca's area: ▪ Inferior frontal gyrus	*Speech-gestural articulatory recoding of print, naming, sequencing.*

Evidence from fMRI (e.g., Borowsky et al., 2006) has supported the existence of the functional dissociation between lexical (ventral/automatic) and sub-lexical (dorsal/non-automatic) processing streams, with both streams utilizing the lateral extrastriate (occipital lobe). However, this study also indicated that the insular cortices contribute to sub-lexical spelling-to-sound processing, as was suggested by Posner and Raichle's (1994) automaticity model. In a recent meta-analysis, Martin et al. (2015) examined fMRI reading studies to contrast reading processes in children and adults. They compiled two groups: the first included 20 fMRI studies involving children 7 to 12 years of age, and the second included 20 fMRI investigations involving adults, aged 23 to 34 years. This analysis revealed a common network for reading that included the left ventral occipito-temporal circuit, left inferior frontal gyrus, left posterior parietal cortex, and bilateral supplementary motor area. Observed differences between participant age were not related to regions, but to the consistency of activation clusters between groups. Activation was observed to be more consistent across the studies involving children for areas including the bilateral supplementary motor area (BA 6) and the left superior temporal

gyrus (BA 38), whereas for the adult studies, there was more consistency bilaterally within the cerebellum and in the left middle frontal gyrus, pericentral gyrus, and middle occipital gyrus. The review also demonstrated the importance of the left ventral occipito-temporal circuit for reading in both children and adults; however, early readers appeared to rely on greater engagement of the left anterior and middle occipito-temporal region, while skilled readers engaged the posterior occipito-temporal region (Martin et al., 2015). The consistency of observed activation levels within the studies for children, and separately for adults, led Price and Devlin (2011) to suggest that the left ventral occipito-temporal cortex supports the integration of bottom-up sensory input with top-down predictions based on prior experience. In this view, referred to as the "Interactive Account," orthographic specialization emerges without tuning to orthographic features but from regional interactions (Price & Devlin, 2011). Within the ventral occipito-temporal region, visual-sensory input activates visuospatial features that integrate with higher-level associations (actions, speech sounds, meaning). Therefore, the function of the ventral occipito-temporal region may change in relation to the regional interaction involving a hierarchy of feedforward and feedback connections—which develop based on experience (Martin et al., 2015). The Interactive Account stands in contrast to, for example, the Local Combination Detectors model of reading developed by Dehaene, Cohen, Sigman, and Vinckier (2005), which suggested that there could be a change in sensitivity to increasingly larger word components from posterior to anterior areas along the ventral visual pathway that occurs during development.

Castro-Caldas, Petersson, Reis, Stone-Elander, and Ingvar (1998) used positron emission tomography (PET) to investigate how learning to read may change the functional organization of the brain in adults. They compared measures of activation between illiterate and literate

individuals performing an auditory repetition task with words and pseudowords. Six literate and six illiterate women, all in their 60s, participated. All were from the same sociocultural environment, were right-handed, and performed within one standard deviation of one another on a short battery of qualifying assessments. The literate group was further evaluated on reading comprehension and writing skills and determined to perform typically (that is, no reading disorders were identified). The behavioral results revealed a significant difference between groups on word repetition accuracy, but a much greater difference in pseudoword repetition accuracy (84% literate group average correct repetitions; 33% illiterate group). The errors made in the pseudoword repetition task were categorized as either lexicosemantic or phonological. There were fewer lexicosemantic errors (2 made by the literate group, 53 made by the illiterate group) than phonological errors (117 made by the literate group, 475 made by the illiterate group). Patterns of brain activation observed during word vs. pseudoword repetition were similar between groups. The superior/inferior parietal regions (BA 7, 39, and 19) were activated in both groups, with the literate group demonstrating greater activation in the left inferior parietal area (BA 40) than the illiterate group. Patterns of activation for pseudowords vs. words revealed much more differentiated engagement of areas. Literate participants showed significantly greater activation bilaterally in the anterior insular (BA 14 and 15) and right frontal opercular cortices (BA 44, 45, 47, and 49), left perigenual anterior cingulate cortex (BA 24 and 32), left basal ganglia, anterior thalamus/hypothalamus, and midline cerebellum. The only significant area of greater activation during pseudoword repetition observed in the illiterate group was the middle frontal/frontopolar region (BA 10), a region associated with episodic memory tasks (Rugg, Fletcher, Frith, Frackowiak, & Dolan, 1996). These findings are meaningful because they demonstrate that unlike the literate group, who through literacy instruction appeared to have

developed a network that can support the accurate repetition of pseudowords (essentially a phonological coding task), the illiterate group seemed to rely on episodic memory rather than a phonological coding network in order to carry out the same task. Castro-Caldas et al. (1998) concluded that learning to read and write facilitates the experience-driven organization of a neural network.

Critical to learning to read is the mapping of letters (graphemes) on to speech sounds (phonemes) (e.g., Hulme, Goetz, Gooch, Adams, & Snowling, 2007). Different sensory channels contribute to this integrative process, and the brain is thought to construct a multisensory interpretation of a "letter" (Raij, Uutela, & Hari, 2000). Raij et al. (2000) explored the neural correlates of an audiovisual integration mechanism for the association of graphemes and phonemes. The stimuli consisted of capital letters that corresponded to 20 auditory letters, and altered letter parts (nonletters) presented sequentially, either on screen (capital letter), through earpieces (digital recording of Finnish phonemes), or simultaneously as an audiovisual stimulus and matched with unrecognizable symbol/sound control stimuli. Using MEG, Raij et al. identified five cortical areas that were engaged when processing integrated audiovisual linguistic stimuli: the left and right fronto-parietal and superior temporal sulci, and the right temporo-occipito-parietal junction. This finding supported the role of the superior temporal sulcus as the site of an audiovisual integration mechanism for letters. The left superior temporal sulcus response for all participants was weaker to audiovisual than control stimuli, which the authors concluded reflects a neural network that has "learned" the presented letter/sound associations.

The emergence during development of a brain network for reading brings together lower-level perceptual processes and higher-level language systems (Posner, Petersen, Fox, & Raichle, 1988). A 20-year review and synthesis of PET and fMRI studies by Price (2012) identified

multiple replicated findings that permit the demarcation of distributed patterns of activation involving multiple areas for specialized purposes—for example, the integration of visual processing, articulation, and semantics in support of orthographic processing. Studies have repeatedly demonstrated that activation of the ventral occipito-temporal cortex is associated with skilled reading (Price, 2012). This area, however, can be further parceled with posterior regions that contribute to feature extraction, and more anterior areas contributing to lexico-semantic whole word processing. Each of these regions is further thought to contribute separately to distinct pathways. One proposed pathway connects the left ventral occipito-temporal cortex and ventral inferior frontal gyrus, creating the lexico-semantic route; another connects the superior temporal and ventral inferior parietal cortices to the dorsal precentral gyrus, supporting the non-semantic phonological decoding route (Price, 2012). Over decades, and despite inconsistent findings from studies of brain activation due to subtle experimental design differences, consistent patterns can be observed for visual word form processing across individuals (Price, 2012). However, whether the left ventral occipito-temporal cortex becomes specialized specifically for visual word forms, as proposed by Cohen et al. (2000), is still debated (Price & Devlin, 2003; 2004).

The research reviewed above has suggested that a reading network emerges as a result of explicit instruction. Dehaene and Cohen (2007) contended that specific brain areas, such as the VWFA, become tuned to specific classes of stimuli such as words. While a network of brain regions important to reading, such as the VWFA (BA 37), the supramarginal gyrus (BA 40), the angular gyrus (BA 39), and the inferior frontal gyrus (BA 44) are consistently observed to activate in response to reading and reading-related tasks (Price, 2012), questions remain as to how they are functionally related. In contrast to Dehaene and Cohen (2007), others have

suggested those areas involved in reading are not dedicated to reading but rather sub-serve more general processing tasks (Price & Devlin, 2003; 2004; Vogel, Petersen, & Schlaggar, 2014).

Vogel, Petersen, and Schlagger (2012) used fMRI to explore the proposition that the left occipito-temporal area or VWFA becomes specialized for words. In a study involving 27 English speaking adults with above average IQ and reading level, participants responded to different visual stimuli including same-different word pairs, single words, phonotactically legal pseudowords, illegal nonwords, consonant strings, Amharic strings, and line drawings matched for visual complexity. Activation in the left occipito-temporal region (VWFA) was greater in response to Amharic characters than English, consistent with a more general processing performed in that region. Vogel et al. suggested that the VWFA may be most responsive to specific characteristics of visual stimuli such as complexity, spatial frequency, and contrast (e.g., Fiset, Gosselin, Blais, & Arguin, 2006; Kveraga, Boshyan, & Bar, 2007; Woodhead, Wise, Sereno, & Leech, 2011). It has further been suggested that the VWFA may be cytoarchitectonically subdivided into two regions, FG1 and FG2, both of which sub-serve visual processing for cognitive tasks (object recognition and visual attention, for example), but one of which (FG2) also serves specialized functions (Caspers et al., 2013). A connectivity analysis indicated that FG1 reflects a "transitional" area between early and higher-level processing, while FG2 shows hemispheric specialization for emotion and face processing lateralizing to the right hemisphere (fusiform face area); in the left hemisphere, FG2 is specialized for visual processing related to language (hence, the visual word form area) (Caspers et al., 2014).

In summary, the ability to read is only acquired after the brain areas associated with language and vision are recruited for solving the particular problem of associating visual symbols (graphemes) with speech sounds (phonemes), which then provide access through the

visual system to the language system. This requires training and extensive practice that ultimately results in reorganization of characteristic brain networks and activations—a process that relies on neuroplasticity.

2.2 Brain Development—Neuroplasticity

Development is a dynamic process reflecting a complex cascade of genetic and environmental factors that progress over time such that earlier-developing systems often become the precursors for later-developing sub-systems (e.g., Stiles, 2017). Connections not functionally relevant are "pruned" to minimize crosstalk and enhance the metabolic and functional efficiency of brain circuitry (Berninger & Richards, 2002). Learning from experiences is understood to result in physical changes to the brain, and such changes are thought to be a critical aspect of brain development across the lifespan (e.g., Hübener & Bonhoeffer, 2014).

Plasticity can be experience-independent, experience-expectant, and experience-dependent (Schatz, 1992). Experience-independent plasticity is a mechanism that allows the refinement of functional connections from genetic instructions driven by internal or external events, primarily during prenatal development (Kolb, Mychasiuk, Muhammad, & Gibb, 2013). The effects of experience-expectant input can be seen in studies of individuals who experience sensory deprivation in one modality during their development. For example, it has been observed that hearing-impaired individuals who experience congenital auditory deprivation, as opposed to hearing-impaired individuals who acquire American Sign Language (ASL), exhibit increased detection of peripheral motion (Neville, 1995). The possibility that the development of the dorsal visual pathway, which contributes to motion processing, is altered due to auditory deprivation was investigated by Armstrong, Neville, Hillyard, and Mitchell (2002) using EEG. Armstrong et al. developed visual stimuli that were designed to stimulate one of two major sensory input

pathways for the visual system: the magnocellular pathway, that originates in the retina and projects to the lateral geniculate nucleus, V1, and then the dorsal visual pathway in the parietal lobe; or the parvocellular pathway, that contributes to the ventral pathway in the temporal lobe (Armstrong et al., 2002). Participants included normally hearing and congenitally deaf adults, who viewed stimuli that appeared on a monitor simultaneously in four peripheral positions and one center position. For a 100-millisecond duration, the stimuli in one of the five positions would change in color—a trigger for the parvocellular pathway—or would change from a static grating to a moving grating—to evoke a response from the magnocellular pathway. Responses from the parvocellular/dorsal pathway were similar between groups; but responses recorded from the magnocellular/dorsal pathway were greater in their amplitude and had a more anterior scalp distribution for congenitally deaf participants than for normally hearing adults. This finding indicated that auditory deprivation might have a more pronounced effect on the magnocellular vs. the parvocellular pathway. It is hypothesized that within the visual system, neuronal populations supporting high acuity, for example, rely on a precision of connections dictated by a "developmental blueprint," while less precise sub-systems supporting depth perception, for example, depend more on activity-mediated interactions (Chalupa & Dreher, 1991). Stevens and Neville (2014) suggested that within the visual system, the different sub-systems display different degrees of neuroplasticity, possibly attributable to different developmental trajectories and acting to enhance or disrupt processing. Evidence has suggested the magnocellular pathway (dorsal pathway) has a longer developmental trajectory than the parvocellular (ventral pathway) (Coch, Skendzel, Grossi, & Neville, 2005; Mitchell & Neville, 2004).

One major reconceptualization in developmental neuroscience in recent years has been the acknowledgment that, in addition to local neural networks shaped by Hebbian learning

(Hebb, 1949), brain organization draws on another form of neuroplasticity that establishes connectivity with various neural partners (e.g., Anderson, 2016). Described as neuronal re-use, the learning of complex cognitive processes or behaviors requires both the modification of local general-purpose circuits and multiple neural partners that, in turn, can further refine local circuitry and reduce its availability as a general-purpose circuit (Anderson, 2016). In many ways, brain development can be considered a self-organizing process (Stiles, 2017). Johnson (2011) developed a framework to relate different levels of observation in a single explanation for the emergence of networks for cognitive functions during development. Interactive Specialization (IS) (Johnson, 2000) is a general theory for understanding human brain development. Rather than taking a specifically maturational or skill learning viewpoint, Johnson (2011) suggested that experience-dependent interactions between neuronal assemblies act over the course of development to tune local response properties so that neuronal activity in specific regions becomes specialized to support a limited set of computational abilities. The process of organizing patterns of interaction between regions underpins the emergence of networks that support cognitive functions. By way of example, Johnson pointed to converging evidence suggesting that the development of visual expertise for print likely demonstrates, as predicted by the IS framework, that more general brain processes are brought to task for word recognition in developing readers, but after instruction and practice, functional specialization emerges.

Changes to connections and patterns of activation between neuron assemblies and neural systems underlie both learning and development. Developmental disorders pose an interesting challenge in that a complex behavior such as reading is supported by multiple brain areas and the connections that link them. Inefficiencies within a network that supports a task likely contribute to the multitude of characteristics associated with disorders such as developmental dyslexia.

Reading is a potential target behavior that may reveal vulnerabilities within a network because reading requires the coordination and integration of patterns of activation emerging from visual, phonological, and articulatory processes (Silani et al., 2005).

2.3 Neurobiology of Dyslexia

The relationship between the brain and reading disability is not a straightforward one. The need to develop explicitly a text-specific network and the protracted timeline required may have implications for understanding the range of differences observed in individuals with dyslexia, for which there are likely a number of potential neurobiological causes (Goswami, 2003).

2.3.1 Structural and Functional Regional and Connectivity Differences in Dyslexia

Several studies have pointed to structural and function differences in the brain organization of non-impaired readers compared with those who have a reading disability. For example, Pugh et al. (2000) reported on converging evidence from early imaging studies that people with developmental dyslexia, compared with non-impaired readers, showed greater activation in temporo-parietal regions, thought to support phoneme/grapheme integration related to lexical-semantic features of words, and in the anterior frontal circuit, associated with articulatory recoding and silent reading; alongside reduced activation in the occipito-temporal region associated with memory-based word identification. Activation over the left hemisphere as a whole, including the dorsal (temporo-parietal) and ventral (occipito temporal) pathways, is consistently reduced in individuals with developmental dyslexia. However, anterior regions repeatedly show increased activation to tasks requiring phonological analysis, which supports the notion that deficits in phonological awareness directly contribute to difficulties with reading acquisition (Habib, 2000; Pugh et al., 2000).

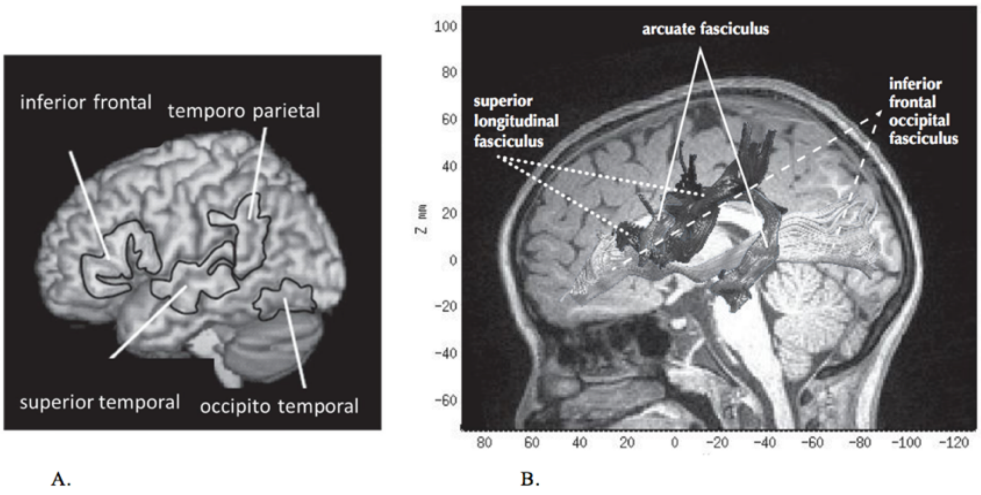

Figure 4. Cortical brain areas associated with dyslexia
With permission from Ozervoc-Palchik & Gaab (2016).
A. Cortical brain areas frequently observed to demonstrate atypical function or structure in dyslexia. B. White matter cortical pathways connecting brain regions important to reading.

Pugh et al. (2000, 2001) speculates that fluent word reading is supported by the ventral occipito-temporal region. However, the development of this area is dependent on input from dorsal temporo-parietal pathways. Attentional resources and associative learning linking phonemes and graphemes ultimately connect morphological and lexical-semantic features of words, creating an integrated representation for rapid word identification via the ventral occipito-temporal area. This symbolic representation is thought to allow words to be recognized as linguistic units rather than visuo-semantic symbols (Pugh et al., 2001). Patterns of compensatory activation observed in individuals with dyslexia, typically the anterior frontal area and corresponding dorsal and ventral areas in the right hemisphere, are thought, respectively, to engage covert pronunciation and build non-linguistic grapheme patterns that permit access to the mental lexicon (Pugh et al., 2001)(see Figure 4. For the cortical areas commonly associated with dyslexia).

2.3.1.1 Post-mortem studies. Early imaging studies were more associative than explanatory but added to the post-mortem studies of the 1970s, 1980s, and 1990s, which helped to establish that dyslexia has a neurobiological origin. In one post-mortem study, Galaburda and Kemper (1979) examined the morphological markers in the brain of a (male) person with documented developmental dyslexia, whose family members exhibited symptoms as well. While no gross abnormalities were observed, at the microscopic level, the left hemisphere, including the parietal, occipital, and temporal lobes as well as limbic, primary, and association areas, all showed mild dysplasias and focal polymicrogyrias.[1] Greater concentrations of abnormalities were found in the left planum temporale and posterior aspects of the auditory regions. In another single brain study of a male with documented developmental dyslexia, Galaburda and Eidelberg (1982) found polymicrogyrias in the left posterior temporo-parietal area and ectopic[2] cell collections in language areas as well as disruptions to the posterior thalamus. Galaburda and Livingstone (1993) conducted a post-mortem evaluation of the differences in the lateral and medial geniculate nuclei (LGN and MGN) in the brains of five adults with dyslexia (four male) with a mean age of 34 and five adults without dyslexia (all male) with a mean age of 40. The parvocellular layers of the LGN showed no differences, but there was more variability in magnocellular layers, which were on average 27% smaller in the brains of adults with dyslexia. In the MGN, observed differences did not reach significance in this study, though it appeared that individuals with dyslexia had fewer large cells and more small cells (Galaburda & Livingstone, 1993). Differences in the MGN were more conclusively demonstrated by

[1] Dysplasias and polymicrogyrias are malformations of cortical cytoarchitecture that occur during cortical development, typically during the late stage of neuronal migration or early in the stage of cortical organization (Barkovich, 2010).

[2] Ectopias are disorganized groupings of neurons located in layer I of cortex due to breaks in the pial-glial limiting laminar membrane, thought to be a result of abnormal neuronal migration (Hyde et al., 2001).

Galaburda, Menard, and Rosen (1994), when comparing the post-mortem brains of five individuals with dyslexia and seven comparison brains. The brains of individuals with dyslexia showed MGN asymmetries, with more small neurons than large neurons in the left hemisphere compared to the right; the authors speculated that this could contribute to phonological deficits observed in dyslexia (Galaburda et al., 1994).

Humphreys, Kaufmann, and Galaburda (1990) reported the presence of ectopias and dysplasias in three female brains with developmental dyslexia, and also showed the presence of glial scars in temporal cortical vasculature. In one case, the scars were observed more broadly in anterior, middle, and posterior vascular areas. A re-examination of three previously studied male brains revealed such scarring in just one brain that also presented with vascular ectopias (Humphreys et al., 1990). Glial scarring is thought to occur only if disruption occurs before myelination is complete; therefore, observed scarring in the cortex and corpus striatum likely happens before or just after birth (Humphreys et al., 1990). Dysplasias and ectopias are related to neuronal migration and form around the 6th month in utero (e.g., Habib, 2000). These studies collectively suggested that microscopic cortical malformations observed in the brains of individuals with dyslexia differ from controls, pointing to atypical cortical development (Habib, 2000).

Post-mortem studies of both male and female brains have also shown that typical asymmetry of the planum temporale, with the left hemisphere structure larger relative to the right, can be absent in those with developmental dyslexia (Galaburda & Kemper, 1979; Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Humphreys et al., 1990). Approximately 90% of those with dyslexia are thought to present with a left planum temporale volume ≤ the right planum temporale volume; however, symmetry of the plana temporale also

occurs among individuals without dyslexia (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990).

2.3.1.2 Structural and functional imaging studies. Numerous imaging studies have identified differences between adults and children with and without dyslexia across the brain regions thought to support reading. For example, Brambati et al. (2004) identified gray matter volume differences between individuals with and without dyslexia across a wide age range (13 to 57 years of age), bilaterally in the plana temporale, inferior temporal cortex, and cerebellar nuclei. Silani et al. (2005) observed activation differences in regions such as the left middle and inferior temporal gyri as well as the left arcuate fasciculus, which were associated with altered density measures of gray and white matter. Hoeft et al. (2007) explored activation and brain volume differences in children with dyslexia and comparison children matched for age or reading ability. Relative to both comparison groups, the left parietal region was the only area observed to have reduced gray matter volume in children with dyslexia. Children with dyslexia exhibited hypoactivation in the left parieto-temporal cortex and the left fusiform gyrus and hyperactivation in the left inferior frontal cortex. Hoeft et al. concluded that regions of hyperactivation in children with dyslexia likely reflect processing differences related specifically to reading ability, whereas regions of hypoactivation reflect functional differences related to dyslexia. Steinbrink et al. (2008) found reductions in fractional anisotropy (FA) measures for white matter tracts (inferior and superior longitudinal fasciculus) traveling between the bilateral fronto-temporal and left temporo-parietal regions in individuals with dyslexia. The relationship between these FA measures was highly correlated with pseudoword reading ability. Gray matter volume differences were also observed in the superior temporal gyri of both hemispheres.

Cutting et al. (2013) explored the question of whether distinct brain "signatures" are associated with different types of reading difficulties. Fifty-one English-speaking adolescents ranging in age from 10 to 14 years (mean age 12.06; SD 1.26) were assessed and split into three groups: those with dyslexia (DYS group), those with specific reading comprehension deficits (S-RCD group), and typically developing (TD group). Based on the lexical quality hypothesis (Perfetti et al., 2007) which attributes efficient word reading to strong phonological, orthographic, and semantic representation of words, a lexical decision task was used with fMRI to determine how word processing differed across groups. No significant differences in reaction time were observed between the TD and S-RCD groups, indicating that the S-RCD group's word recognition responses were not disrupted. However, the S-RCD and DYS groups showed patterns of activation that deviated from the TD group. The DYS group revealed a significant covariance of activity between the VWFA and bilateral medial frontal gyri in response to low-frequency words, demonstrating a context-dependent functional interaction anomaly in the IFG. The S-RCD group, on the other hand, revealed a significant covariance of activity in hippocampal and parahippocampal regions and the left IFG, compared to TD. For pseudowords, the DYS group showed greater connectivity than the TD group between left AG and areas in the right hemisphere mirroring the left hemisphere language circuit. For the S-RCD group, reduced functional connectivity was observed in areas between the right AG and anterior and posterior cingulate regions. These findings suggested that the reading difficulties experienced by the S-RCD group, who did not reveal the characteristic activation and connectivity changes observed in the DYS group, were related to neurobiological differences that affect the access of lexical-semantic representations of words during word identification—a difference that could be a cause or consequence of an altered reading experience (Cutting et al., 2013).

Xia, Hoeft, Zhang, and Shu (2016) used voxel-based morphometry (VBM) to investigate influences of maturation and atypical development in individuals with dyslexia. Four groups of Chinese students were included: older and younger students with dyslexia (DYS Older mean age 14.1; SD .45; DYS Younger mean age 11; SD .6); an older typically developing group age-matched to the DYS Older group (TD Older); and a TD Younger group age-matched to the DYS Younger group and to the DYS Older group reading level. VBM analysis revealed reduced gray matter volume for the DYS groups in the left temporo-parietal cortex (Heschl's gyrus, planum temporale, and supramarginal gyrus), left middle frontal gyrus, and left superior occipital gyrus; white matter reductions were seen in the bilateral parietal-occipital regions, the left cuneus and right precuneus. Xia et al. concluded that disorder-by-maturation effects could be observed in the dorsal part of the left pars opercularis, the genu of the corpus callosum and left ventral occipital temporal cortex, and these could contribute to disruptions in higher-order brain functions.

Vandermosten et al. (2015) examined connectivity differences (FA) between pre-reading Dutch children with and without familial risk of dyslexia. Regions of interest were three white matter tracts associated with reading: the ventral inferior fronto-occipital fasciculus (IFOF), and the dorsal arcuate fasciculus subdivided to into AF1 (connecting frontal regions with the inferior parietal and temporal regions) and AF2 (connecting posterior temporal and posterior parietal regions). Measures of phonological awareness, rapid automatized naming and letter knowledge, along with FA measures, were used to compare groups. Findings revealed that FA measures of AF1 correlated with phonological awareness in pre-readers; however, the left IFOF demonstrated white matter anomalies in pre-readers with familial risk of dyslexia. The IFOF tract connects the VWFA with frontal areas such as the pars triangularis. These results were unexpected and the

authors suggested they may reflect a sample of children without phonological problems (Vandermosten et al., 2015).

Clark et al. (2014) conducted a longitudinal MRI and behavioral study, with the aim of identifying early neurobiological changes associated with dyslexia. Fifty-two Norwegian children with and without familial risk of dyslexia (n = 26 in each group) were observed at four time points: 5-6 years (behavioral data only), 1st grade (ages 6-7 years), 3rd grade (ages 8-9 years), and 6th grade (ages 11-12 years). Thirteen participants received a diagnosis of dyslexia in 6th grade. Retrospective analysis indicated that these children showed thinner cortex in regions associated with low-level auditory, visual and executive function, as well as other parts of the reading network: left hemisphere Heschl's lingual, medial frontal, and middle cingulate gyri, and the right orbitofrontal cortex. Areas such as Heschl's gyrus correspond to regions identified by Galaburda et al. (1985) as loci of microscopic disruptions in post-mortem studies; the other regions are thought to contribute to low-level visual processing and executive functioning, respectively (Clark et al., 2014).

Clark et al. (2014) also evaluated cortical thickness based on MRI scans taken at ages 11-12 years. Children with dyslexia showed a significantly thinner cortex in left orbito-frontal, anterior superior temporal, and adjacent middle temporal cortical regions. This contrast was more pronounced in male children, who also showed greater differences when regions of interest were expanded to the traditional reading network (temporo-parietal region, visual word form area, and the inferior frontal gyrus), as well as right hemisphere regions including anterior cingulate and Heschl's gyrus extending to the insular cortex. Interestingly, when only female participants were compared, this extended network of reduced cortical thickness was not observed. Mean cortical thickness over time was also analyzed. Findings suggested that for

children with dyslexia, several areas increased in thickness during development, while the lingual gyrus in typically developing children became thinner over time. These changes obscured between-group differences by the end of the study, leaving Heschl's gyrus as the only area still significantly thinner in children with dyslexia.

Clark et al.'s (2014) findings, consistent with other studies, suggested that familial risk of dyslexia increases the likelihood of behavioral and neuroanatomical differences associated with a variety of potentially contributing factors, which points to a genetic contributor. The one persistent region of difference in this study, Heschl's gyrus, has been implicated in prior studies (e.g., Altarelli et al., 2014) and suggests a deficit in low-level auditory processing (Clark et al., 2014; Goswami, 2003). Additionally, pre-reading differences in thickness of the lingual gyrus (thought to support low-level visual processing) potentially indicate differences in plasticity. Children without dyslexia showed a reduction in thickness attributed to experience-driven changes, as opposed to children with dyslexia, where lingual cortical thickness remained more or less constant across development.

2.3.1.3 Event-related potential studies. Electroencephalography (EEG) has been used extensively to study language as well as other sensory and cognitive processes and provides insights into brain-behavior developmental issues. Event-related potentials (ERP) relate specific time-locked stimuli to measures of brain activity recorded in the continuous EEG signal. Several studies have indicated disruptions to speech processing very early in development for children who are later identified as having reading disabilities.

Using recorded speech and non-speech sounds, Molfese (2000) collected ERP data from 48 infants within 36 hours of birth. ERP responses to speech and non-speech sounds were compared to reading and IQ measures obtained from the same children at age 8. Neonatal ERP

measures predicted reading difficulties across three groups of children (poor readers, children with dyslexia, and controls) with 81.25 % accuracy. Molfese speculated that early differences in sensory functioning might contribute to later reading ability.

Another ERP study explored speech sound processing in Dutch babies aged 2 months using an oddball passive listening paradigm (van Zuijen, Plakas, Maasen, Maurits, & van der Leij, 2013). ERP responses were examined for 26 babies with familial risk of dyslexia (17 boys) and 12 controls (8 boys). When participants reached 2nd grade (age 7), reading fluency scores were used to form three groups: fluent-control (12 children), fluent with familial risk (16 children), and non-fluent with familial risk (10 children). ERP measures at 2 months were found to differentiate between these groups. While both fluent groups showed an ERP mismatch response (MMR), controls presented a right anterior positivity while fluent at-risk readers presented a right parietal positivity. Non-fluent readers showed no MMR, possibly indicating a basic auditory processing deficit. Van Zuijen et al. (2013) speculated that combinations of risk and protective factors predict which children with familial risk become fluent or non-fluent readers.

ERP has also been used to explore possible differences in the visual tuning for print between children with and without dyslexia. Maurer et al. (2007), using visually presented words, pseudowords, symbols, and line pictures, asked participants to detect repetitions of the stimuli, which were presented for 700 ms with 2050 ms intervals. Participants included 15 (6 male) children with, and 22 (11 male) children without, familial risk of dyslexia; all participated in the experiment in kindergarten (mean age 6.5) and again in 2nd grade (mean age 8.25). Children falling below the 10th percentile on reading and spelling assessments were identified with dyslexia (13 children from the familial risk group and 2 from the no-risk group).

While performance accuracy improved from kindergarten to 2nd grade, ERP data confirmed that children with dyslexia showed an attenuated difference in brain responses to words vs. pseudowords. These distinctions were thought to represent differences in visual tuning to print and were also found to correlate with differences in reading speed.

2.3.2 Subcortical Differences

The cerebellum contributes to linguistic processing as well as other cognitive (Desmond, Gabrieli, & Glover, 1998; Mariën et al., 2014) and sensory processes (Fulbright et al., 1999). Finch, Nicolson, and Fawcett (2002) compared cerebellar Purkinje cell density and size in the post-mortem brains of four adults with dyslexia (mean age 21.4) and four adults without (mean age 57.25). They observed that Purkinje cells were larger in the posterior cerebellar cortex for the dyslexic group; the same pattern was observed in anterior regions but did not reach significance. An abundance of larger cells and fewer smaller cells was also noted, and cell size differences could not be accounted for by age-related atrophy effects.

Using probabilistic tractography, Fernandez et al. (2016) sought to determine whether previous findings documenting bilateral volume differences in the anterior lobe of the cerebellum in children with impaired single-word decoding (Fernandez, Stuebing, Juranek, & Fletcher, 2013) might be explained by disruptions in pathways to areas associated with reading, including the temporo-parietal, occipito-temporal, and inferior frontal regions. Greater bilateral fractional anisotropy (FA) was observed in children with dyslexia compared to typically developing children, between the anterior lobe of the cerebellum and the temporo-parietal region, as well as the inferior frontal region. Connectivity patterns between the cerebellum and occipito-temporal region also differed with respect to age. Younger children with dyslexia showed reduced FA whereas older children with dyslexia showed increased FA, both in comparison to children

without dyslexia. Overall, children with dyslexia showed increased FA, regardless of age. Fernandez et al. speculated that this increase in fractional anisotropy reflects the cerebellum serving a compensatory role in coordinating processes that support skilled automatic reading.

Feng et al. (2016) explored levels of activation in the cerebellum and connectivity with other regions of interest in Chinese children with (mean age 10.19, SD 0.68) and without (mean age 10.31, SD 1.0) dyslexia. Children with dyslexia showed greater bilateral cerebellar activation in an orthographic task; greater severity of dyslexia was associated with greater activation. Differences in functional connectivity measures were found to be task-dependent. For the orthographic task, greater connectivity between the right cerebellum V1 lobule and the left fusiform gyrus was observed in children with dyslexia, whereas a phonological task elicited greater connectivity between the left cerebellum V1 lobule and the left supramarginal gyrus. While this pattern of differences points to the cerebellum's compensatory role in reading deficits, Feng et al. discussed re-evaluating the role of the cerebellum in light of accumulating evidence for reciprocal networks of cortico-cerebellar pathways contributing to a range of cognitive processes, including multiple linguistic functions (Mariën et al., 2014). The collective evidence suggested that observed differences in cerebellar structure and functional connectivity reflect the complex nature of development in relation to skill acquisition (see Stoodley & Stein, 2011).

Anatomical and functional differences in the thalamus have also been associated with dyslexia. Giraldo-Chica, Hegarty, and Schneider (2015) examined volumetric differences in the lateral geniculate nucleus (LGN) between adults with (mean age 24.08, SD .54) and without (mean age 23.46, SD .37) dyslexia (n = 13 in each group). The findings revealed that the left LGN was significantly smaller for adults with dyslexia and the left LGN volume was positively and significantly correlated with spelling ability.

Fan, Davis, Anderson, and Cutting (2014) used diffusion tractography-based thalamo-cortical connectivity analyses to investigate differences between children with and without developmental dyslexia. Compared to typical readers, children with dyslexia showed greater connectivity between the thalamus and sensorimotor as well as lateral prefrontal cortices. The thalamic-sensorimotor connectivity correlated significantly with reading scores. Fan et al. speculated that this could reflect an immature subcortical sensorimotor system, required to integrate visual and motor processes that contribute to the neural basis of language and support reading. The observed increase in connectivity between the thalamus and the lateral prefrontal cortex might represent compensatory mechanisms, as this pathway is thought to contribute to working memory processes (Braver et al., 1997). These observed thalamo-cortical differences between children with and without dyslexia suggested that the thalamus may mediate task-specific functional connectivity (Fan et al., 2014).

2.3.3 Heritability and the Genetics of Reading Differences

Morgan (1896) was one of the first to identify "congenital word-blindness" as a familial trait, reporting a case involving one family, in which over two generations and six members presented with a deficit relating to the visual representations of words. Today, it is widely acknowledged that developmental dyslexia is both familial and heritable (Berninger & Richards, 2010; Gilger, Pennington, & DeFries, 1991; Paracchini, Diaz, & Stein, 2016; Pennington, 1995).

The question of why children differ in reading ability has often taken an environmental focus, as reading is a learned skill requiring formal instruction (Olson, Keenan, Byrne, & Samuelsson, 2014). Based on twin studies, it is thought that genetic risk factors may change as curricular demands across academic years also change (Berninger & Richards, 2010). Olson et al. (2014) reviewed a number of longitudinal studies in the United States, Australia, Scandinavia,

and the United Kingdom to provide cross-language and cross-cultural perspectives on reading disability. They suggested that after the early grades, when it seems schools can reduce environmental variance with formal instruction, discrepancies between those with and without reading disability are strongly influenced by genetics (Olson et al., 2014). Environmental influences such as maternal antibodies (Vincent et al., 2002), fatty-acid deficiencies (Cyhlarova et al., 2007; Taylor et al., 2000), and prenatal testosterone (Geschwind & Galaburda, 1985) may contribute to the nature/nurture interaction. Environmental factors such as parenting style, relationships with peers, as well as access to medical care, proper nutrition, and education can affect development. The influence of such factors on the expression of genes, in turn affecting the plasticity and development of the nervous system, has yet to be unraveled, but is thought to contribute to the nature-nurture interaction (Institute of Medicine (US) Forum on Neuroscience and Nervous System Disorders, 2008).

2.3.3.1 Heritability.[3] Reading ability is thought to be normally distributed (Fisher & DeFries, 2002; Shaywitz, & Shaywitz, 2005), but family studies of dyslexia have reported heritability estimates of 58% and higher (Friend, DeFries, & Olson, 2008; Kirkpatrick, Legrand, Iacono, & McGue, 2011; Pennington & Olson, 2007). Between 23 and 65% of all children with dyslexia have a parent with dyslexia (Scarborough, 1990) and that risk increases to 76-78% if both parents have dyslexia (Gilger, Hanebuth, Smith, & Pennington, 1996). Approximately 20-33% of the siblings of an individual with dyslexia will also have dyslexia even without affected parents (Gilger et al., 1996). Estimates can be lower when obtained earlier in development (Byrne et al., 2009). Reading disability can result from limited or poor instruction; in such cases, the environment will influence reading ability separately from genetic endowment

[3] Heritability provides a statistical description of the proportion of variance in a trait or characteristic thought to be associated with genetic rather than environmental influences (Fisher & DeFries, 2002).

(Paracchini et al., 2016). As such, in families at risk for dyslexia, higher parental education levels correspond to greater heritability estimates (Friend et al., 2008; Peterson & Pennington, 2012). However, additional environmental factors that may contribute to reading disability in at-risk children are presently unknown (Elliot & Grigorenko, 2014).

The heterogeneous nature of dyslexia has prompted some researchers to use standardized psychometric measures such as orthographic processing, phoneme awareness, and rapid automatized naming as a proxy of different endophenotypes, potentially providing evidence of a causal link to underlying genetic factors (Carrion-Castillo, Franke, & Fisher, 2013). Strong heritability estimates have been reported for a number of factors, including reading performance (41-74%) (Grigorenko, 2004); semantic processing and reading comprehension (60-67%) (Betjemann et al., 2008); phonological processing (50-80%) (Byrne et al., 2009); orthographic processing (60-87%) (Gayán & Olson, 2003); and spelling (90%) (Grigorenko, 2004). Keenan, Betjemann, Wadsworth, DeFries, and Olson (2006) found that distinct genetic influences on word recognition and listening comprehension were shared with genetic factors contributing to reading comprehension, together accounting for all the genetic influence on reading comprehension. To address inconsistent findings among studies, Betjemann, Keenan, Olson, and DeFries (2011) investigated whether selection of a particular behavioral measurement contributes to discrepancies across studies. They found that differential patterns of covariance for separate measures of reading comprehension were similar in overall genetic influence, but that distinct genetic factors correlated with decoding and listening comprehension separately.

While heritability estimates indicate the percentage of individual difference in a phenotypic characteristic that may be attributable to genetic individual difference, these estimates do not specify what genes contribute to observed reading difficulties. Accumulated

family data suggested that inheritance is multifactorial and not the result of a single gene transmitted from generation to generation in a Mendelian manner (Rutter & Maughan, 2005). Plomin and Kovas (2005), reviewing the quantitative research, suggested that susceptibility genes likely have both specific and more general influences on learning disability.

2.3.3.2 Genetics. There are many approaches to identifying genetic loci and candidate genes for dyslexia (see Fisher & DeFries, 2002). Early studies focused on mapping of potential risk loci, identification of gene variants associated with dyslexia, and gene function in animal models (Carrion-Castillo et al., 2013). Nine chromosomal regions are associated with risk of reading disability, including DYX1-DYX9 (Gabel, Gibson, Gruen, & LoTurco, 2010) and 14 candidate genes, although some findings have not been replicated (Poelmans, Buitelaar, Pauls, & Franke, 2011). Evidence for multiple risk loci suggested that identified loci contribute to both typical and atypical reading development, such that individuals with dyslexia have less advantageous alleles and/or increased environmental risk factors (Fisher & DeFries, 2002; Pennington & Olson, 2007). Intermediary factors may also influence downstream behavior, potentially resulting in alterations of gene and protein expression (Eicher & Gruen, 2013).

Many susceptibility genes for dyslexia, such as ROBO1, DCDC2, DYX1, and KIAA0319, are associated with variant functions involving brain development, especially neuronal migration (Ozernov-Palchik, Yu, Wang, & Gaab, 2016; Paracchini, Scerri, & Monaco, 2007). Galaburda, LoTurco, Ramus, Fitch, and Rosen (2006) proposed that the variant functions of susceptibility genes involved in early brain development could result in cortical disruptions during neural migration and axonal growth. These disruptions could subsequently alter cortico-cortico and cortico-thalamic circuits associated with frequently observed cognitive, perceptual, and sensorimotor dysfunctions reported in dyslexia. Giraud and Ramus (2013) proposed that

such cascades might alter brain oscillations[4] and disrupt auditory signal sampling, contributing to difficulties in phonological processing.

One susceptibility gene, ROBO1, is thought to influence axonal guidance and has been implicated in corpus callosum development (Sun et al., 2017). The ROBO1-callosum relationship was explored in Chinese readers grouped by alleles and behavioral reading measures (Sun et al., 2017). Two ROBO1 polymorphisms were investigated that showed significant effects on word list reading, but not on a Chinese character recognition task. DTI and MR data revealed that the gene-to-brain mechanism was specific to the genu and splenum (anterior and posterior regions of the corpus callosum respectively)—specifically relating to axonal diameter and density in the area of the genu and axonal myelination in the splenum.

Eicher et al. (2016) used structural MRI and DTI data from the Pediatrics Imaging Neurocognition Genetics (PING) study to investigate the relationship of risk variants DYX2 and DYX3, associated with IQ, language, and reading, with cortical thickness and volumetric measures in children aged 3 to 20 years. They found associations of KIAA0319 and cortical thickness in the left orbito-frontal region and FAM65B with global fractional anisotropy for the DYX2 locus. They also found suggestive associations between temporal region cortical thickness and volume measures for the DYX3 markers. These findings provided a basic pathway for establishing specific risk markers which may impact behavioral phenotypes.

Gori et al. (2015) considered the possible role of the DCDC2 intron[5] 2 deletion in developmental dyslexia. DCDC2 is associated with developmental dyslexia (Paracchini et al.,

[4] Neural Oscillations—rhythmic fluctuations of neural excitability that can be synchronized across large numbers of neurons, present as periodic waves in EEG. Oscillations are quantified in terms of frequency bands, phase, and amplitude/spectral power (Calderone, Lakatos, Butler, & Castellanos, 2014).

[5] An intron is a segment of DNA or RNA that does not code for protein but is involved in splicing, mRNA transport, and expression regulation. They are thought to provide selective advantages to cells to be evolutionarily maintained, however at a high energetic cost (see Jo & Choi, 2015).

2016), and with typical reading ability (Scerri at al., 2011). Imaging studies have linked DCDC2 to fiber tracts connecting the left middle temporal gyrus with other cortical areas via the angular and supramarginal gyri as well as the superior longitudinal fasciculus and corpus callosum (Darki, Peyrard-Janvid, Matsson, Kere, & Klingberg, 2012). The magnocellular/dorsal stream, associated with attention and motion perception (Schneider & Kastner, 2009) and with reading disability (Franceschini, Gori, Ruffino, Pedrolli, & Facoetti, 2012; Kevan & Pammer, 2009), is implicated in these connections. Gori et al. set out to demonstrate first that developmental dyslexia is associated with a magnocellular/dorsal pathway deficit and then show that the DCDC2 intron deletion is selectively associated with deficits in this pathway. Groups of Italian-speaking children, with dyslexia and without, were grouped on the presence vs. absence of DCDC2 intron deletion. A rotating-tilt line illusion task and an accordion grating illusion task (AGI) were used to evaluate magnocellular/dorsal functioning, and a grating orientation identification task was used to evaluate parvocellular/ventral functioning. Children with dyslexia required higher contrast than either the chronological-age comparison group or reading-level comparison group to perceive the illusion for the magnocellular tasks, but there were no group differences for the parvocellular/ventral task. There were significant response differences between children with and without the DCDC2 intron deletion on the magnocellular tasks. For the parvocellular task, however, there was no effect of DCD2 intron deletion for children with dyslexia. Finally, the authors extended the study to include typical adult readers with and without DCDC2 intron deletions. Once again, presence of the deletion was associated with increased mean contrast thresholds for magnocellular tasks, again suggesting that this genetic risk variant might contribute to disruption of the magnocellular/dorsal stream, preserving the

parvocellular/ventral stream. The magnocellular/dorsal pathway is thought to contribute to sequencing letters, and attention; thus, these findings suggested ways in which reading acquisition could be influenced by gene-brain-behavior pathways.

The magnocellular theory of developmental dyslexia posits that across auditory, visual, somatosensory, and motor systems, larger (magno) cells specialized for temporal processing are disrupted in individuals with dyslexia (Stein, 2001). Approximately 10% of all neurons in the brain can be categorized as magnocellular, and they are observed in many brain areas including cortex, thalamus, and cerebellum (Hockfield & McKay, 1983). Identified gene variants cannot be said to cause dyslexia, but they point to potential vulnerabilities and suggest ways in which genetic pathways, influenced by factors such as epigenetic modifications and environmental effects, can be disrupted during development (Stein, 2017). Ozernov-Palchik and colleagues (2016) updated the multidimensional etiology model put forth by Pennington (2006), expanded by Van Bergen, van der Leij, and de Jong (2014), to begin to specify factors and mechanisms that contribute to developmental reading disability (see Figure 5). This figure emphasizes the complexity of developmental dyslexia and the importance of interdisciplinary approaches to the genetic, neural, cognitive, and environmental influences in reading differences.

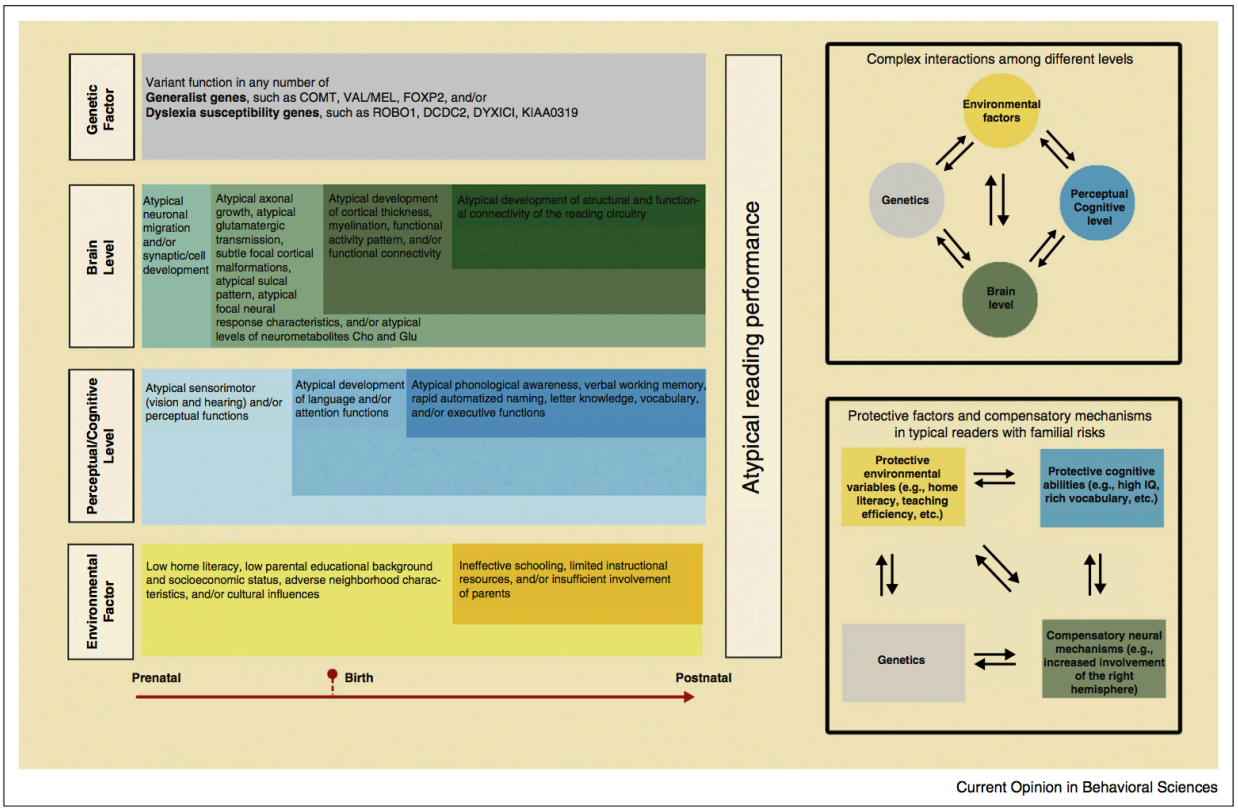

Figure 5. Prenatal to postnatal developmental timeline that summarizes the multiple levels of risk facts thought to contribute to atypical reading performance. With permission from Ozernov-Palchik, Yu, Wang, & Gaab (2016). Right panels illustrate interactions among the levels of risk factors and bottom right highlights the protective factors at each level that potentially contribute more successful compensation.

2.4 Theories of Dyslexia

Multiple theories have emerged to explain the heterogeneous characteristics associated with dyslexia. The theories are likely not mutually exclusive (Ramus et al., 2003) because they differ in nature (cognitive, biological) and level (descriptive, explanatory, causal). The most common deficit specifically related to reading involves the representation, storing, and retrieval of phonemes, which when mapped to graphemes permit access to oral language via text. The most widely-supported theory attempting to account for this deficit is the phonological deficit theory (Fowler, Brady, & Shankweiler, 1991; Peterson & Pennington, 2015; Snowling, 1981).

However, Paracchini et al. (2016) pointed out that the phonological deficit theory does little more than describe a difficulty experienced by individuals with dyslexia, failing to provide an explanation. Most theories of dyslexia likely account for subsets of neural and cognitive profiles associated with dyslexia resulting from complex interactions between multiple genetic and environmental factors (Scerri & Schulte-Körne, 2010). One of the challenges to understanding causality is the knowledge that reading experience contributes to mastery; this is difficult to unravel from the neurobiological structural and functional differences seen between individuals with and without dyslexia (Olulade et al., 2013).

The three most prevalent theories of dyslexia—the phonological deficit theory, the temporal information processing deficit theory, and the magnocellular theory—are highlighted in this section.

2.4.1 The Phonological Deficit Theory of Dyslexia

Beginning in the early 1970s, accounts of the etiology of dyslexia shifted from perceptual deficits to linguistic explanations, based on difficulties associating linguistic and visual complements (Vellutino, 1979a). Since reading is a linguistic skill, it was presumed that reading difficulties result from deficits in aspects of linguistic processing (Vellutino, 1979b). Shankweiler and Liberman (1972) were the first to take a linguistic approach to understanding reading disability. Their approach built on ideas advanced by Chomsky (e.g., Chomsky, 1986) and Fodor (e.g., Fodor, 1983) who considered language to be independent of fundamental sensory processing (Stein, 2001).

The phonological deficit theory suggests that dyslexia involves multiple issues with language-related processes, specifically poor phonological awareness, reduced verbal short-term memory, and slow lexical retrieval (Wagner & Torgesen, 1987). Many studies have provided

evidence for a core phonological deficit among children and adults with dyslexia (e.g., Elbro, Nielson, & Peterson, 1994; Fowler et al., 1991; Snowling, 1981; Stanovich, 1988; Wagner & Torgesen, 1987). Weak phonological representations are thought to compromise a developing reader's ability to learn grapheme/phoneme correspondences—that is, the relationships between letter symbols and speech sounds (Ramus et al., 2003). Regardless of compensation strategies, the phonological deficit is thought to persist across the lifespan and individual differences in the severity of the phonological difficulties correspond to reading abilities (Nation & Snowling, 1998).

The phonological theory of dyslexia also postulates that automatization of letter-to-sound correspondences is disrupted due to an inability to attend to and manipulate the sounds of one's language (e.g., Peterson & Pennington, 2015). Ramus and Szenkovits (2008) conducted a series of experiments probing the nature of the phonological deficit in dyslexia and, based on their findings, they concluded that phonological access, rather than phonological representation or processing, is disrupted in dyslexia. Potential supporting evidence for this view comes from neuroimaging; for example, Boets et al. (2013) demonstrated that while connectivity between the auditory cortex and left inferior frontal gyrus was reduced in adults with dyslexia, brain responses to speech sound contrasts in the primary and secondary auditory cortex were similar to typical adult readers. However, while there continues to be consensus among many researchers that a phonological deficit is core to dyslexia, there is little consensus on the nature of that deficit (Nicolson et al., 2010; Ramus et al., 2003).

In short, the phonological deficit theory proposes a straightforward link between a cognitive deficit and reading disability. At the neurological level, it is reasoned that disruptions to the structure and/or function of the left-hemisphere perisylvian regions are responsible for the

poor integration of phonological and orthographic representations in individuals with dyslexia. However, given the breadth of observed differences between people with dyslexia across other domains (sensory, motor, cognitive), the phonological theory seems insufficient to explain the whole phenomenon. While there is significant evidentiary support for this theory, other theories have been actively pursued.

2.4.2 Temporal Information Processing Deficit Theory

The temporal information processing deficit theory attributes reading disability to a sensory dysfunction in the processing of rapid (transient) and sequential auditory signals (Tallal, 2004). Early in this line of research, Hirsh (1959) showed that various temporal and sequencing tasks were dependent on more basic perceptual processes. Tallal and Piercy (1973, 1974) were the first to propose that a primary cause of language disorders could be a disruption in the low-level processing of rapid auditory transitions. Tallal (1980) later linked the putative temporal auditory processing deficit to reading by suggesting that such a deficit would impact the phonological processing necessary for reading acquisition. Temporal processing deficits have also been observed in the visual system (Lovegrove, 1993) and motor system (Wolff, 1993).

Farmer and Klein (1995) conducted a review of evidence for a temporal processing deficit as the causal factor in a number of cases of dyslexia. They concluded that temporal processing could be an issue for a subset of individuals with dyslexia, but speculated that the deficit could be speech-specific or result from other limiting factors involving attention, perception, and/or memory. They further noted that not only could dyslexia result from an auditory temporal processing deficit but from a visual temporal processing deficit as well. In the latter case, disruption to the transient (magnocellular system) as opposed to the sustained (parvocellular system) channels in the visual system might result in the persistence of

orthographic representations from fixation to fixation and alter the availability of information in the parafoveal area. A subsequent study by Farmer and Klein (1995) suggested that the observed temporal processing deficits could be more general in nature, but that the impact on reading is most strongly related to deficits in auditory processing.

Waber (2001) summarized research on a number of timing measures across modalities collected from children aged 7-11 years, who did and did not have a learning disability diagnosis. While poor readers performed worse than controls on low-level tests of temporal information processing, deficits in temporal information processing tasks, such as rapid automatized naming, were observed in children with learning impairments—some of whom did not have a reading disability. Waber did not clarify whether a temporal processing deficit contributes causally to deficits in higher cognitive functions. It could be that such a deficit merely reflects an effect of poor general processing, presenting as differences in rate, timing, or perceptual accuracy—an indicator of an underlying processing problem rather than the cause of it. Finally, Waber suggested that timing task performance may reveal temporal processing deficits that can co-occur with reading disability and interact with instruction.

Talcott et al. (2000) investigated associations between sensory sensitivity and reading ability. Measures of sensory psychophysics and children's cognitive, reading, and spelling abilities were correlated. Findings suggested that low-level visual and auditory function contributes to reading ability, supporting Tallal's view that poor phonological processing abilities could be attributed to disrupted discrimination of rapidly changing acoustic signals. Hence, this theory posits that a phonological deficit is secondary to a deficit in rapid auditory processing (Ramus et al. 2003). However, the results of studies of temporal auditory processing

have been mixed, providing little clarity about differences in performance between individuals with and without dyslexia (Ramus & Ahissar, 2012).

A variant of the temporal processing deficit approach is the temporal sampling framework proposed by Goswami (2011), which recognizes the parallel processing of sensory input at different timescales (oscillatory frequencies) and across different neuronal groupings. Buzsáki (2006) hypothesized that neuronal oscillations play a critical role in processing sensory information and could be the mechanism by which the brain organizes and integrates information. According to the temporal sampling framework, oscillatory mechanisms may be impaired in dyslexia such that the sampling of the speech stream by the auditory cortical network, which operates at different oscillatory frequencies (delta, theta, gamma), inefficiently encodes the linguistic information in speech (Goswami, 2011). This approach relates to the multi-time resolution model (MTRM) of speech, which suggests that the right-lateralized "theta sampling network" facilitates temporal integration at the level of the syllable. The theta band (3-6 Hz) segments auditory input into syllable packets that reset in response to speech dynamics ultimately thought to reflect the encoding of the amplitude envelope (multiple frequency bands in the speech signal that differ in amplitude over time). According to MTRM, temporal integration at the phonetic level is accomplished by the "gamma sampling network" (28-40 Hz) operating bilaterally. The different oscillatory networks sample different aspects of the speech stream at the level of the syllable and phoneme, which can then be integrated with the mental lexicon (Goswami, 2011). Goswami's work builds on Poeppel's (2003) Asymmetric Sampling in Time (AST) hypothesis which posits that a speech signal includes information on multiple time scales (format transitions, intonation contours, and syllabicity, for example), each of which is

mediated by left and right auditory cortices in the form of a symmetric bilateral neural representation. These representations are elaborated asymmetrically within the time domain, such that the left hemisphere processes rapidly changing signals (20-40 ms), whereas the right hemisphere samples signals within a longer integration window (150-250 ms). This integration provides a framework for constraining temporally evolving information, reflected as oscillatory neural activity in the gamma and theta ranges (Poeppel, 2003). Giraud and Poeppel (2012) further elaborated on this granularity in the bottom-up temporal analysis of speech, suggesting that oscillations segregate and track information related to speech dynamics. The importance of temporal information would predict that dysfunctional oscillatory processes, resulting from ion channel formation and functioning, for example, may influence oscillatory neuronal behavior and indicate a neural mechanism behind neurodevelopmental disorders such as dyslexia (Giraud & Poeppel, 2012). Susceptibility genes associated with dyslexia, such as KIAA0319 or DCDC2, when knocked out in animal models, disrupt both superficial and subcortical layers, the generators of oscillations (Peschansky et al., 2009; Wang et al., 2011).

Lehongre et al. (2011) compared auditory cortex gamma oscillations in adults with and without dyslexia measuring auditory steady state response (ASSR) using magnetoencephal-ography (MEG). Based on the AST approach, the authors hypothesized that oscillatory patterns in the low-gamma band (25 to 35 Hz) are optimal for phonemic sampling and that slower or faster rates would, respectively, affect phonemic discrimination or overwhelm the auditory system with spectrotemporal information. Their data suggested that in the left hemisphere, individuals with dyslexia had a reduced bias to the optimal frequency (30 Hz) for phonemic analysis, such that left auditory cortex oversamples phonemic information.

Vidyasagar (2013) speculated that oscillatory activity might influence visual processing during reading by co-opting the neural processes critical to recognizing a target in a crowd scene. Synchronized oscillation is thought to support visual attention (Gregoriou, Gotts, Zhou, & Desimone, 2009; Miller & Buschman, 2013; Saalmann, Pigarev, & Vidyasagar, 2007) and spatiotemporal sampling of visual input for letter and word identification is likely facilitated by a top-down serial search mediated by synchronized neuronal oscillations (Saalman et al., 2007). A top-down visual attentional mechanism is thought to emerge from afferent magnocellular pathways or the dorsal cortical stream. Since words vary in length, Vidyasagar (2013) suggested that graphemes are sampled in V1 because graphemes do not vary much in the cortical space occupied. In Figure 6, Vidyasagar illustrated how lower gamma frequency ranges facilitate top-down attentional mechanisms, synchronizing posterior parietal cortex (PPT) and middle temporal (MT) areas with V1 and sequentially processing visual input within V1 across the retinotopic map. It is therefore plausible that temporal processing differences may cause phonological deficits as well as affect the visuo-spatial processing of input to the visual system (Vidyasagar, 2013). Goswami (2011) also speculated that such inefficiencies in neuronal entrainment to auditory signals could account for apparent phonological deficits as well as impacting attention and auditory-visual integration.

Theoretical perspectives encompassing a temporal mechanism attributed to reading disability offer a deeper level of explanation than does the phonological deficit. However, such approaches are only beginning to explain how multiple processes may be affected.

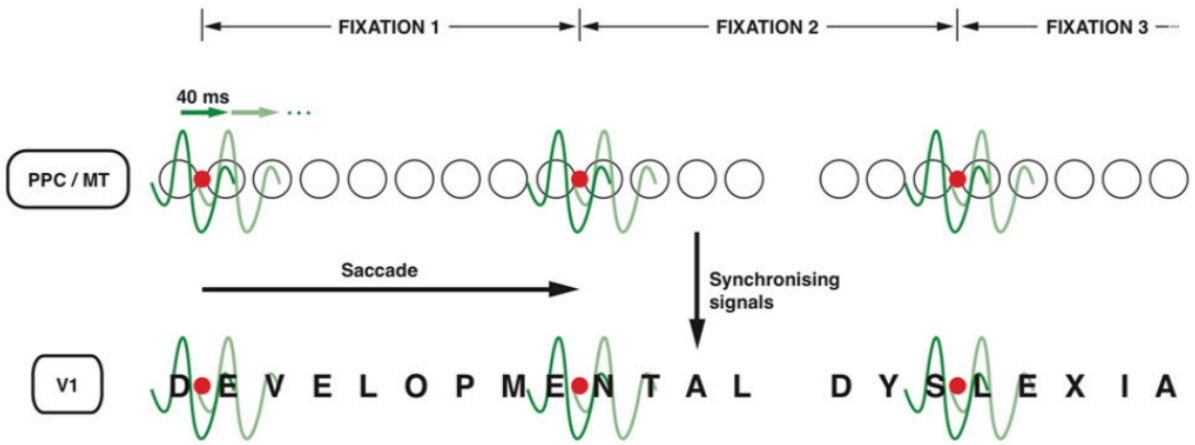

Figure 6. How lower gamma frequency ranges facilitate top-down attentional mechanisms
Reproduced from Vidyasagar (2013),
permitted under the Creative Commons Attribution License (CC BY).
Depicts how synchronized neuronal oscillations between posterior parietal cortex (PPC) and the middle temporal (area MT) might facilitate sequential letter processing in V1 during reading. The top row indicates fixation periods of 250 to 300 ms. The middle row consists of empty circles indicating lateral intraparietal cortex and MT encoding the locations of letters in the text, with red dots representing the point of fixation. V1 responses are depicted in the bottom row as bottom-up sensory signals representing the visual world as gamma frequency wavelets sequentially sample the retinotopic map that, in this instance, captures the form of letters.

2.4.3 The Magnocellular Theory of Dyslexia

While it is undisputed that the visual system is involved in reading, the general assumption is that dyslexia is rooted in a linguistic but not a visual problem. Therefore, the view that the etiology of dyslexia is related to a visual deficit of some nature is controversial. Lovegrove, Martin, Blackwood, and Badcock (1980) first demonstrated reduced visual sensitivity thresholds in individuals with dyslexia in response to low-contrast, low-spatial, and/or high-temporal frequency stimuli—the kinds of stimuli to which magnocellular layers of the LGN are most sensitive (Shapley, 1990). As discussed above, various studies have shown abnormalities affecting magnocellular neurons in the brains of people with dyslexia (e.g.,

Galaburda & Livingstone, 1993; Giraldo-Chica, Hegarty, & Schneider, 2015; Livingstone, Rosen, Drislane, & Galaburda, 1991).

The observation that the magnocellular pathway and not the parvocellular pathway is disrupted in dyslexia (Livingstone et al., 1991; Lovegrove, Martin, & Slaghuis, 1986; Stein & Talcott, 1999; Stein & Walsh, 1997) has inspired decades of debate (see Skottun, 2000; Skottun & Parke, 1999; Skottun & Skoyles, 2006, 2008; Stein, Talcott, & Walsh, 2000) and further research. The most consistent findings supporting a deficit in the magnocellular pathway in individuals with dyslexia have come from studies of coherent motion tasks, such as random dot kinetograms (e.g., Conlon, Sanders, & Wright, 2009; Conlon, Sanders & Zapart, 2004; Cornelissen, Hansen, Hutton, Evangelinou, & Stein, 1998; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Olulade et al., 2013). Across multiple psychophysical studies, a large proportion of individuals with dyslexia have shown reduced sensitivity to coherent motion perception as well as other speed discrimination tasks (Demb, Boynton, Best, & Heeger, 1998; Hansen, Stein, Orde, Winter, & Talcott, 2001; Witton et al., 1998), even when matched for age and IQ (Cornelissen et al., 1995, 1998; Talcott et al., 1999, 2000).

The magnocellular pathway primarily projects to the dorsal stream including the middle temporal (MT), medial superior temporal (MST), and parietal cortical regions (Ungerleider & Mishkin, 1982). Functional MRI studies have revealed reduced or no activity in the MT during motion coherence tasks (Demb et al., 1997; Eden et al., 1996; Heim et al., 2010). Stein (2001) argued that reduced sensitivity to motion indicates a reduced sensitivity of the magnocellular pathway generally, which contributes to dorsal stream functioning including visual guidance of movement such as eye movements. The magnocellular system is also thought to support visual attention and, therefore, visual search as well; both are worse in people with dyslexia (Everatt,

1999; Iles, Walsh, & Richardson, 2000). Benassi, Simonelli, Giovagnoli, and Bolzani (2010) conducted a meta-analysis investigating between-group differences in motion processing between individuals with and without dyslexia, revealing a large effect. Further research is required to understand better whether higher- or lower-order functions within the dorsal stream of the visual system contribute to observed reading difficulties (Benassi et al., 2010).

In the auditory system, the large (magno) cells are not anatomically distinct from other neurons. However, magno cells are consistently specialized for analyzing transient signals across the sensory system (Stein & Walsh, 1997). The magnocellular theory posits that magnocellular temporal processing deficits result in disruption to both basic visual and auditory processing (Stein, 2001). However, studies have also implicated a low-level auditory impairment in dyslexia traceable to brainstem nuclei (McAnally & Stein, 1996), where neural discharges have been seen to be phase-locked to stimulus characteristics such as onsets and offsets.

Investigators have explored possible relationships between magnocellular deficits and impaired reading, attributing difficulties to factors such as abnormal saccade suppression (Breitmeyer & Ganz, 1976), or impairments in binocular fixation and rapid eye movements (Stein & Talcott, 1999). Others have suggested that a magnocellular deficit reflects comorbid mechanisms such as attentional (Stuart, McAnally, & Castles, 2001; Willcutt & Pennington, 2000) or perceptual speed deficits (McLean, Stuart, Coltheart, & Castles 2011). While the specific factor is still to be determined, Boets, Vandermosten, Cornelissen, Wouters, and Ghesquière (2011), based on longitudinal data collected from Dutch-speaking pre-readers followed through 3rd grade, provided evidence that coherent motion sensitivity can serve as an index of magnocellular/dorsal stream integrity and can be used to predict reading and spelling problems in 1st and 3rd grades. Threshold differences were greater between pre-reading children

later diagnosed with dyslexia (11 out of 31 from a group of children with familial risk and 5 out of 31 from a group with no family history) and typically developing readers. This finding suggests that magnocellular/dorsal stream integrity facilitates proficient reading.

It remains unclear whether a visual magnocellular deficit is causal or is a by-product of dyslexia. Reading experience may indirectly improve the efficiency of the visual system for motion detection. A magnocellular deficit may simply co-occur and not contribute to reading disability. Alternatively, the magnocellular pathway, which is thought to have a more protracted developmental trajectory compared to the parvocellular pathway, may be more vulnerable to disruption and/or amenable to intervention (Coch et al., 2005; Mitchell & Neville, 2004). However, Gori et al. (2015; Gori, Seitz, Ronconi, Franceschini, & Facoetti, 2016) strongly suggested a causal role for magnocellular-dorsal pathway deficits in developmental dyslexia, also reporting that such a deficit could be associated with a genetic deficit, DCDC2-Intron 2 deletion, in individuals with and without dyslexia.

2.4.4 Overview of Cognitive and Biological Explanations

Cognitive and biological explanations of dyslexia, while relevant, all have limitations. Regardless of the nature of the explanation, there is consensus in the scientific community that causal factors of dyslexia are inherent to the individual (Vellutino, Scanlon, & Sipay, 1997). Of the theories that have persisted, each seems insufficient independently to explain the whole of dyslexia, leading many researchers to consider that dyslexia is a multifactorial behavioral disorder involving both risk and protective factors (Pennington, 2006; Pennington & Bishop, 2009). The multiple deficits hypothesis (see Catts, McIlraith, Bridges, & Nielsen, 2017) posits that while a phonological deficit may be the primary causal factor, to reach diagnostic criteria across a reading continuum, other biological and environmental factors must co-occur. In

addition to finding support for different theoretical frameworks, reviews have supported a multi-componential model of dyslexia that considers how genetic, environmental, and cognitive factors interact as risk and protective factors (Snowling & Melby-Lervåg, 2016; Vandermosten, Hoeft, & Norton, 2016). These multiple theoretical perspectives provide tentative explanations for the likely subgroups that exist under the umbrella of dyslexia and the many possible reasons for experiencing difficulty in learning to read and attaining proficient reading fluency.

2.5 Reading and the Visual System

Early research pointed to the visual system as the source of disruption in both acquired and developmental dyslexia. Referred to as "wordblindness" (Tomkins, 1894), early cases of acquired dyslexia were attributed to the visual system and, later, related to the developmental disorder. Orton (1964) attributed deficits to a failure of the brain to represent print. More recently, a joint statement issued by the American Academy of Pediatrics (2009) declared that while vision problems such as strabismus, amblyopia, refractive errors, convergence, and/or focusing deficiencies can disrupt learning, "vision problems are not the cause of primary dyslexia or learning disabilities" (p. 837). Furthermore, the statement discourages diagnostic and treatment approaches that include eye exercises, behavioral vision therapy, or tinted lenses in the treatment of reading disabilities due to a lack of scientific evidence. Lack (2010) offered a rebuttal to the Academy's statement, arguing, for example, that a review of the literature (Vidyasagar & Pammer, 2010) provides emerging evidence that poor visual coding contributes to phonological problems and reading impairment. Specifically, a processing disruption within the dorsal visual stream receiving the majority of its input from the magnocellular pathway may affect the attentional mechanisms thought to contribute to serial scanning of letters. Such a disruption could result in any number of effects, including impeding the visual processing of

graphemes and their translation to phonemes—processes essential for the development of phonemic awareness (Lack, 2010).

Laycock and Crewther (2008) described how reading fluency is achieved when mapping from orthographic symbols to phonemes becomes automatic. Fluency frees cognitive resources, otherwise dedicated to decoding, to achieve the goal of reading comprehension (Fuchs, Fuchs, & Maxwell, 1988). While fluency requires the coordination of eye movements, accommodation, and fixation, those coordinated processes allow for the rapid spatiotemporal processing of the visual input (Laycock & Crewther, 2008).

The relationship between neuronal structure/sensi;ivity and perception is not straightforward. Due to the inconsistency of the signal and an inherently noisy sensory system, it appears that response patterns emerge in the visual system through trial and error, supporting behavior based on probabilistic inferences drawn from experience (Purves, 2010). DeYoe and Van Essen (1988) proposed a framework for aspects of visual perception that considers patterns of connectivity and physiological specializations of the cortical pathways. They suggested that retinal images are represented as intensity distributions, derived from functions of position, wavelength, time, and eye. Information is economically represented in early processing stages, then inputs are integrated and later-stage neurons show sensitivity to spatial, temporal, and/or chromatic aspects of the retinal image. Inconsistencies in the spatial or temporal distribution of luminance or spectral properties form the basis of more complex features such as spatial contrast, velocity, disparity, and orientation. From these features, many properties of the visual scene can be inferred and translated into attributes such as shape, size, color, texture, spatial relationships, and movement.

The retina and lateral geniculate nucleus (LGN) consist primarily of neurons that constitute the magnocellular and parvocellular visual pathways, each with distinct physiological response preferences (Shapley & Perry, 1986; Yoonessi & Yoonessi, 2011). Ungerlieider and Mishkin (1982) proposed that each pathway might process different visual features: the dorsal "where" pathway handles spatially-related information while the ventral "what" pathway deals with object-related features (see Figure 7). It has been observed that neurological symptoms resulting from temporal lobe lesions in the ventral "what" stream contribute to selective impairments such as loss of color perception, loss of object recognition, and inability to recognize faces with maintained object recognition (Livingstone, 2002). Parietal lobe lesions affecting the dorsal "where" stream are associated with problems perceiving motion or depth, telling right and left, seeing complex objects as whole, and the spatial perception of motion (Livingstone, 2002).

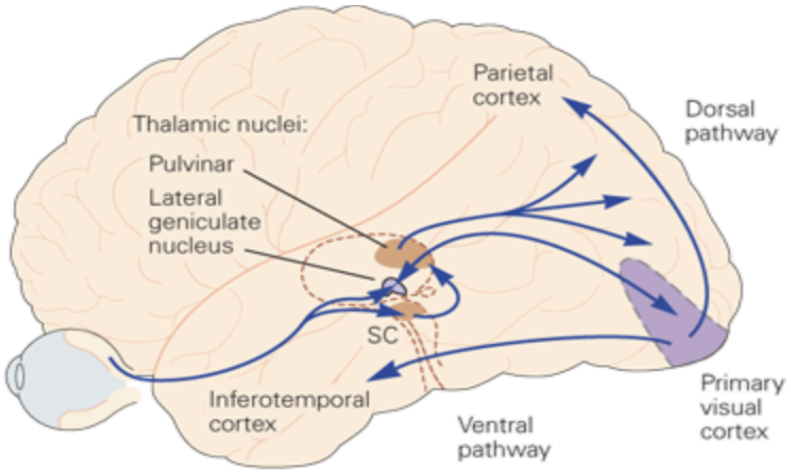

Figure 7. Visual processing
Kandel et al. (2012), Copyright © 2018 McGraw-Hill Education.
The magnocellular and parvocellular pathways project information from the eyes to the thalamus which includes the lateral geniculate nucleus and the pulvinar. From there, information travels to the primary visual cortex and then the magnocellular pathway travels primarily to the dorsal pathway extending to the parietal cortex. This area serves motion detection and visually guided movement. The parvocellular pathway travels primarily to the ventral pathway in the inferotemporal cortex. This area is associated with object recognition.

However, findings are beginning to support the notion that accurate perception and action require engaging both the ventral and dorsal streams in an integrative manner (Braddick & Atkinson, 2011). Building on the work of Fenske, Aminoff, Gronau, and Bar (2006), Breitmeyer (2014) proposed a model that incorporates a neural network approach to object recognition, with input from the fast dorsal stream that modulates early stage visual processing. Under this model, a coarse low-frequency image is transmitted from V1 via the dorsal stream to the prefrontal cortex (PFC), where the representation triggers probable corresponding object representations that facilitate top-down processing in inferior temporal cortex. This coarse representation is complemented by higher-resolution representations from the slow ventral pathway, derived from bottom-up perceptual processing. In this way, feed-forward and feed-back activation loops in the ventral stream are modulated by top-down input from the PFC and dorsal stream (Breitmeyer, 2014; see Figure 8). Other studies (Cloutman, 2013; Freud, Plaut, & Behrmann, 2016; Giese & Poggio, 2003; Gilaie-Doton, 2016; Himmelbach & Karnath, 2005; Keizer, Colzato, & Hommel, 2008) supported the view that there is cross-talk between dorsal and ventral processing streams in areas such as the superior temporal sulcus and fusiform face and in low-level visual processing.

Johnston, Pitchford, Roach, and Ledgeway (2016) investigated whether tasks developed to explore dorsal and ventral stream processing differed between university students across reading abilities. Participants had to judge the direction of motion in a global motion task and the orientation of stimuli in a global form task. Findings revealed that global motion and global form processing mechanisms are not entirely separate, as the authors found a strong positive correlation between thresholds for the form and motion tasks. Additionally, for both poor readers and individuals with reading disability, performance differences did not seem to be related to motion processing but to temporal information processing.

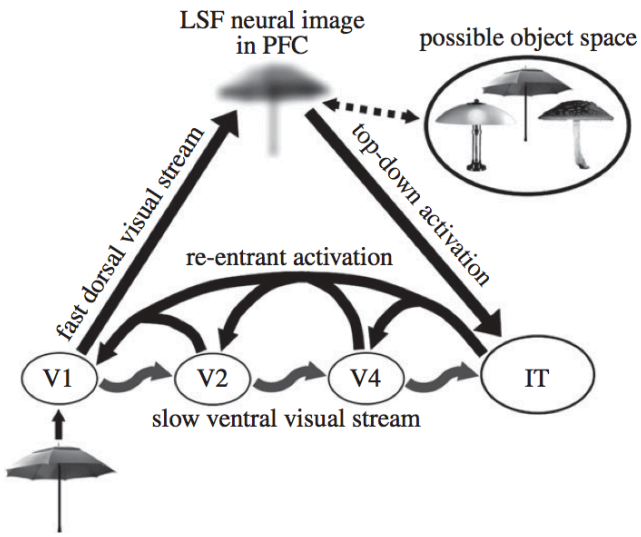

Figure 8. Interactive Model
Adapted from Fenske et al. (2006) by Breitmeyer (2014); reproduced with permission.
Interactive model of the contributions of top-down (fast, dorsal) and
bottom-up (slow, ventral) visual processing streams to object recognition.

Other theoretical perspectives on the kinds of visual and cognitive processes involved in reading have also been proposed. Dien (2009) reviewed EEG literature with a view to evaluating the utility of the cognitive dual-route cascade (DRC) model of visual word recognition. In the DRC model, two pathways are available to transform printed text to semantic access. A low-level perceptual analysis that supports letter identification is proposed to precede processing in the lexical or phonological pathways. Orthographic analysis of a percept involves identifying the orthographic code within the lexicon and then linking this to meaning. Should the percept not be identified as a known orthographic code, the less efficient pathway implementing a rule system for converting grapheme to phonemes enables the generation of a phonological representation. The brain region associated with the orthographic route (posterior fusiform gyrus) is also involved in object recognition more generally, and this region may act as an interface between visual information and higher-order processes necessary for drawing meaning from visual

stimuli (Devlin, Jamison, Gonnerman, & Matthews, 2006). Dien (2009) concluded from existing EEG literature that the pathways proposed under the DRC model are coordinated in a "convergence of processes during the initial information burst and resonance processes during an extended harmonization process that follows" (p. 19).

Grainger, Dufau, and Ziegler (2016) expanded on work by Ehri (1992) and Share (1995) to propose a theoretical framework for visual and orthographic processing involved in reading, suggesting that spatial attention facilitates the development of a specialized system. On this view, skilled readers develop a mechanism for computing letter identity alongside location relative to fixation and position within a word. To access lexical level information from print efficiently, beginning readers engage in a slow sequential process of phonological decoding, which is dependent on whole-word phonological representations previously linked to meaning. Unfamiliar words require knowledge of both the symbol system and grapheme-phoneme correspondences. With practice, letter-level representations can bypass phonology and provide direct access to associated meaning. With mastery, readers increase their use of "flexible orthographic representations" (Grainger et al., 2016, p. 176).

Most brain imaging reading paradigms have required the serial visual presentation of words to minimize movement artifact, consequently putting a greater focus on the ventral pathway (Zhou et al., 2016). Zhou et al. (2016) used fMRI to investigate the functional connectivity of the reading network during text reading in 16 Chinese students by simultaneously presenting multiple Chinese characters. Results indicated connectivity between the left middle frontal gyrus (MFG), the left intraparietal sulcus (IPS), and the visual word form area (VWFA) involving top-down effects from the MFG to the left IPS and VWFA as well as the IPS to the VWFA. Additionally, resting state data indicated that dorsal connections were more strongly

associated with reading fluency than with lexical decision. Skeide at al. (2017), in a study of illiterate adults learning to read Devangari script, found that after only 6 months of training, the functional connectivity between the occipital lobe and subcortical areas in midbrain (right superior colliculus) and the thalamus had increased; individual rates of connectivity were strongly correlated with decoding skill gains. While the Zhou et al. study provided evidence for particular connectivity patterns and their functional relevance, the Skeide et al. study is of particular importance because it demonstrates the reorganization of subcortical connections, at least for literacy training in adults. This finding called into question whether thalamic disruption could be a causal factor in developmental dyslexia and/or evidence of altered reading experience (Skeide et al., 2017). However, more research is needed to evaluate whether the observed functional changes constitute a response to necessary encoding and/or skill consolidation.

Phonemic awareness is understood to be a core deficit associated with reading disability, and even adults with dyslexia find it difficult to process and manipulate the sounds of their language. Neuroimaging has demonstrated an association between poor reading and disruption to various interrelated brain regions. In a recent meta-analysis of PET and fMRI studies, Paulesu et al. (2014) suggested that, in addition to the expected differences in the left hemispheric reading network (including the left inferior frontal, premotor, supramarginal cortices, and left infero-temporal and fusiform regions), reduced activation in the occipito-temporal cortex in dyslexia was associated with reduced reading and reading-like behaviors, including visuo-phonological mapping. Specifically, there was consensus among studies that individuals with dyslexia showed differences in motor and attentional systems recruited for reading, which seem to be associated with the dorsal left fronto-parietal cortex (Paulesu et al., 2014).

Such observations have prompted the suggestion that reduced activation in the VWFA might reflect a consequence of a reading impairment rather than a cause (e.g., Boros et al., 2016). To better understand orthographic processing in developmental dyslexia, Boros et al. (2016) conducted two experiments with French-speaking children with and without dyslexia (mean age 11.5 years). In the first experiment, participants searched for letters, digits, and symbols within five element strings. In the second experiment, participants passively viewed pseudowords and false font strings. Group differences were observed in the VWFA as well as the middle occipital gyrus, an area associated with visuospatial processing and thought to be necessary for the ordering of symbols in unfamiliar strings. The authors speculated that observed differences in the VWFA are secondary to a deficit associated with the middle occipital gyrus, which is outside of the ventral pathway. Further, Boros et al. suggested that stimuli that cannot be processed automatically will engage the dorsal stream.

For at least two decades, researchers have observed differences in motion processing between individuals with and without dyslexia (e.g., Eden et al., 1996; Gori et al, 2016; Livingstone et al., 1991; Wilmer, Richardson, Chen, & Stein, 2004). How such a disruption would contribute to reading difficulties is not well understood. However, some research has demonstrated an association between reading ability and dorsal stream sensitivity in adults and in children before and after learning to read (Boets et al., 2011; Kevan & Pammer, 2009). Pammer and Vidyasagar (2005) suggested that dorsal pathway disruption is related to reading failure because of its role in visuospatial attention.

Research has shown that magnocellular visual functioning is disrupted in children and adults with reading disability, using coherent motion detection tasks (random-dot kinematograms: Cornelissen et al., 1995; Demb, Boynton, & Heeger, 1998; Edwards et al., 2004;

Raymond & Sorensen, 1998; Talcott et al., 1998). Yet it was not well understood how degraded information processing in areas receiving input from the magnocellular pathway would disrupt reading, though it was speculated that position encoding of letter features could be involved (Cornelissen et al., 1995). Scheuerpflug et al. (2004) used electrophysiology to explore visual processing differences between German children with and without dyslexia to a motion-onset paradigm and coherent-motion condition. For the motion-onset paradigm, results supported previous findings that motion detection is less developed in children with dyslexia, measured as a reduction in neural activity thought to be influenced by the magnocellular system. As for the coherent-motion paradigm, amplitude measures increased as coherence increased with no difference between groups. Scheuerpflug et al. concluded that group differences in electrophysiological responses to moving stimuli are dependent on the stimulus condition and strongly suggest specific disruptions to visual processing.

Vidyasagar (1999) proposed that the motion deficit observed in individuals with dyslexia implicated the magnocellular pathway, speculating that the magnocellular pathway may be critical to directing sequential attention during reading. Cheng, Eysel, and Vidyasagar (2004) manipulated stimulus luminance values in a visual search task. Because luminance can only be detected by the magnocellular cells, longer reaction times in response to isoluminant stimuli suggest the importance of this pathway in serial search. However, in a feature search task, luminance had no effect. These findings suggested that the magnocellular pathway provides a fast track for visual input to the parietal cortex used to deploy attentional resources within the slower parvocellular pathway. Slow parvocellular input is therefore employed as an attentional or processing mechanism (Kveraga et al., 2007; Laycock & Crewther, 2008; McLean et al., 2011; Vidyasagar, 2013).

Lawton (2016) proposed a mechanism whereby disrupted magnocellular processing may hinder developing readers (see Figure 9). This working hypothesis highlights how disruptions in timing due to "sluggish" magnocellular cells can minimize the effectiveness of attentional influences on parvocellular cells in sequential processing.

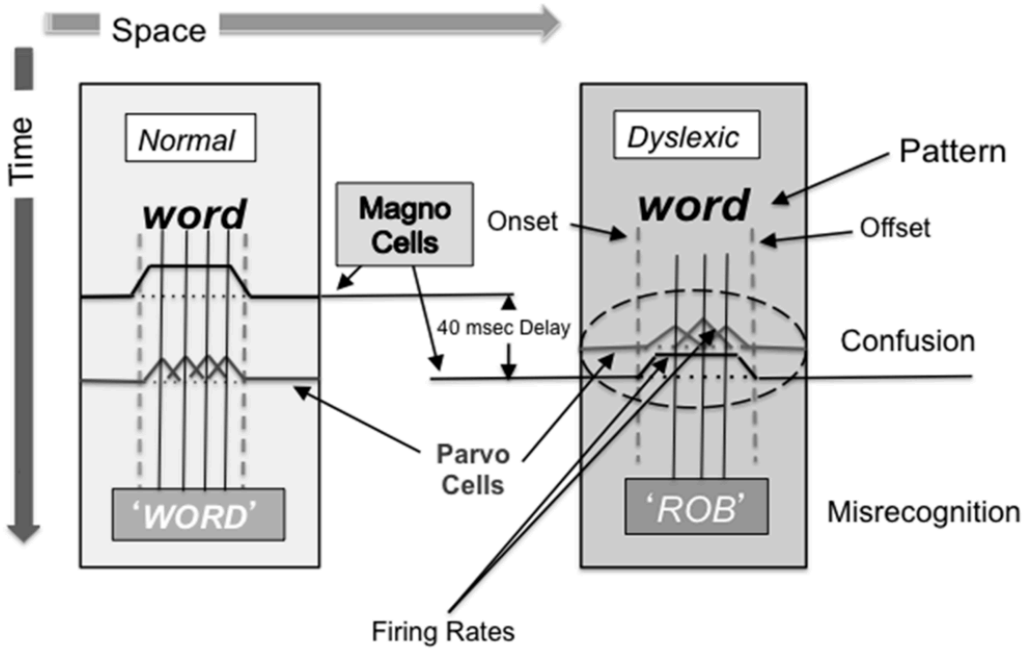

Figure 9. Disrupted magnocellular processing
Reproduced from Lawton (2016), permitted under
the Creative Commons Attribution License (CC BY).
This illustration demonstrates how magnocellular neurons that are slowed by 20 to 40 ms might cause confusion during word identification across space and time.
It is hypothesized that the dorsal stream rapidly processes visual input to establish a "frame of reference" that the ventral stream sequentially analyzes.

Disruptions to common networks that manifest as subtle behavioral variations may have more complex effects when interacting with other systems in complex behaviors such as reading. Neuronal activity, behavior, and abilities across different people are influenced by genetic and environmental variability, and the direct and indirect interactions between multiple pathways and factors (Institute of Medicine, 2008). Reading is a behavior that relies on multiple sensory and

cognitive networks, requiring a survey of genetic, brain, perceptual/cognitive, and environmental levels of explanation to understand the effects of experience-driven learning.

This study sought to add to our understanding of magnocellular and parvocellular visual processing in adults with and without dyslexia and to gain insights into the relationships between pathway functioning and the sub-skills that contribute to reading fluency.

3. STUDY RATIONALE, RESEARCH QUESTIONS, HYPOTHESES, AND PREDICTIONS

This dissertation study evaluated the magnocellular theory of dyslexia by investigating the response characteristics of the magnocellular and parvocellular pathways specific to the LGN in individuals with and without dyslexia using ERP measures as indices of early visual processing, as well as to explore possible relationships between ERP and behavioral measures.

Visual stimuli, developed to preferentially bias each of the two pathways, were used to obtain amplitude and latency measures of early visual ERP components (specifically the P1, N1, P2 components). Earlier studies have dissociated the two pathways using stimuli manipulated across spatial, temporal, luminance, and chromatic parameters (Coch et al., 2005; Denison, Vu, Yacoub, Feinberg, & Silver, 2014; Mitchell & Neville, 2004). Magnocellular neurons at the level of the LGN are most sensitive to high temporal resolution, low contrast, and low spatial resolution (Lennie, Trevarthen, Van Essen, & Wässle, 1990; Yamasaki et al., 2014), and parvocellular neurons are most sensitive to high spatial resolution, color, high contrast, and low temporal resolution (Lennie et al., 1990; Tobimatsu & Celesia, 2006).

Luminance, the physical intensity of light, is perceived by humans as brightness in chromatic and achromatic images. The perception of brightness is dependent on the sensitivity of an individual's eyes to each wavelength of light (Livingstone, 2002), and it is therefore a subjective experience. Although color contains luminance (more commonly referred to as the *value* of color), luminance is processed by the magnocellular pathway that contributes to the dorsal "where" system (Livingstone, 2002). However, this pathway can be blinded to an object by removing luminance-contrast information, such as when a color image is equiluminant (Livingstone, 2002). Therefore, to use color to preferentially bias the parvocellular pathway, a

color stimulus that minimizes luminance contrast was used. Isoluminance, or equiluminance, means that colors vary in chromatic contrast only (hue) but are equal in their luminance value. To accommodate for differences in the perception of brightness among individuals, a task was included that matched the brightness of one color to a paired color so that color pairs included in the color stimulus were perceived as equiluminant. One method for matching luminance values is the Motion Null Technique (see Appendix A for explanation); this technique was used in a luminance-matching task and completed by each participant prior to setting specific values for luminance of experimental stimuli.

Electroencephalography (EEG) was used to measure the neurophysiological responses to the stimulus parameters that preferentially bias the parvo- and magnocellular pathways. This provided an online recording of electrical field potentials generated by neuronal activity. Continuous EEG recordings were then segmented and averaged together to provide Event-Related Potentials (ERPs). As an index of early visual sensory processing, the ERP components of interest were the P1, N1, and P2 components, each of which has clearly delineated differences in timing, amplitude, and scalp distributions (all discussed further in Chapter 4 below). A differentiation between ERP latencies and/or amplitudes between individuals with and without dyslexia in response to motion stimuli (biasing the magnocellular pathway) but not to color stimuli (biasing the parvocellular pathway) would support the magnocellular theory of developmental dyslexia. However, if there were observed differences in response to both stimuli between groups, or no differences in response to both stimuli between groups, then the motion coherence and speed detection differences observed in other studies of sensory differences in dyslexia would not be well explained by the magnocellular theory.

Finally, differences in task performance on selected behavioral measures, including components of the Comprehensive Test of Phonological Processing (CTOPP-2) (Wagner, Torgensen, & Rashotte, 1999); the Woodcock Reading Mastery Test-Revised (WRMT-R/NU) (Woodcock, 1998); the Test of Word Reading Efficiency—Second Edition (TOWRE-2) (Torgesen, Rashotte, & Wagner, 1999); the Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV) (2009); as well as measures of orthographic processing and nonverbal visual reasoning/memory, were correlated with the findings from the ERP study.

3.1 Research Question One

Are there differences in early visual responses as measured by event-related potential methodology to stimuli developed to separately and preferentially bias the magnocellular or parvocellular visual pathways between groups of adults with and without dyslexia as would be expected by the Magnocellular Theory of Dyslexia?

Hypothesis: *The magnocellular-mediated aspects of early visual processing are affected in dyslexia in such a way that amplitude and latency measures of the P1, N1, and P2 ERP components are altered in response to motion stimuli, while parvocellular-mediated component measures are unaffected.*

Predictions: Based on this hypothesis, longer latencies and attenuated amplitude measures are predicted for all of the ERP components measured in response to motion stimuli in individuals with dyslexia compared to individuals without dyslexia. Such differences are thought to be reflective of a disruption to the processing of input that biases the magnocellular pathway (high temporal, low contrast, and low spatial resolution). In addition, there will be no differences between the group of adults with and without dyslexia in response to the color stimulus (biasing

the parvocellular pathway) in the ERP latency or amplitude measures. Such findings would support the Magnocellular Theory of Dyslexia.

3.2 Research Question Two

Do behavioral instruments that measure orthographic skill, phonological ability, and processing speed correlate with neurophysiological measures of early visual processing in the magnocellular and/or parvocellular pathway?

Hypothesis: *The contribution of visual sensory processing to specific reading skills, as measured by amplitude and/or latency values for the P1, N1, and P2 components obtained in response to the motion stimulus (biasing the magnocellular pathway) and the color stimulus (biasing the parvocellular pathway), will be reflected in the relationship between neurophysiological measures and measures of reading skill.*

Predictions: Early visual processing, indexed by latency and amplitude measures for the P1, N1, and P2 components in response to both motion and color stimuli, are predicted to vary by participant. Latency and amplitude values observed in response to the motion stimulus are predicted to correlate positively with behavioral measures of *orthographic processing* such as the Orthographic and Homophone Choice Tasks and the assessment of Rapid Automatized Naming and Word Identification, but not with behavioral measures of *phonological processing* such as Phonological Awareness and Word Attack. No relationship is predicted between any behavioral measures and the ERP latency and/or amplitude measures in response to the color stimuli.

The following chapter provides detailed information about the measures and methods used in this study.

4. RESEARCH METHODS AND DESIGN

Electroencephalography (EEG) provides a non-invasive means for measuring electrical activity generated by neuronal assemblies in the brain. This activity can be measured from outside the brain only when the alignment of these neuronal populations allows for the summation of excitatory and inhibitory postsynaptic potentials. This applies principally to cortical pyramidal cells, which are the primary generators of recorded activity because of their parallel and orthogonal alignment in the cortex (Woodman, 2010). Synchronous activation or deactivation, typically from apical dendrites of pyramidal cells in the upper layer of the cortex, produces voltage fluctuations (typically 5-10 microvolts [μV]) that conduct to the surface of the scalp (Luck, 2005, 2014; Molfese, Molfese, & Kelly, 2001), where they can then be recorded by electrodes positioned across the head. The recorded electrical signal is amplified and digitally recorded.

Event-related potentials (ERPs) are the wave-pattern components of a continuous EEG recording that are brought about by an experimental stimulus, which can be a sensory, motor, or cognitive event (Luck, 2014). ERPs are derived from an EEG recording through time-locking to stimulus events, segmentation, and averaging (Luck, 2014). Averaged waveforms associated with each trial type make it possible to link the neuroelectrical response to a stimulus presentation—hence, event-related—because activity related to other sensory, cognitive, and biological processes can be averaged out of the waveform, improving the signal-to-noise ratio (Molfese et al., 2001).

An ERP component can be characterized by a sequence of voltage deflections, positive or negative, and their order of occurrence or the latency of the deflection peak (Picton et al., 2000). For example, an N1 component describes the first negative deflection present in the waveform;

an N100 describes a negative deflection occurring approximately 100 milliseconds after the stimulus is presented. Components are interpreted within the context of the research question to provide insights relating to perception, cognition, and motor functions (Handy, 2005).

Data are re-referenced offline to the average reference. During recording, a reference electrode location is selected (typically the vertex) and then during data processing, the calculated mean voltage of all electrodes is subtracted from that of each individual electrode (Dien, 1998; Picton, Lins, & Scherg, 1995; Picton et al., 2000). The average reference is close to the theoretically desirable zero voltage (Bertrand, Perrin, & Pernier, 1985). It is generally agreed that sampling from high-density electrode nets provides an acceptable approximation to zero (Dien, 1998; Luck, 2005, 2014).

This study used the ERP technique to measure neurophysiological differences between individuals with dyslexia and without dyslexia, in response to stimuli designed to separately bias the magnocellular and parvocellular visual pathways. The following sections discuss the experimental design, participants, recruitment procedures, sample size, measures, instruments, equipment, and procedures used in this study.

4.1 Experimental Design

Group differences were explored using a mixed repeated measures analysis of variance comparing adaptive mean amplitude and peak latency measures for each ERP component of interest. ERP measures were recorded in response to stimuli designed to preferentially bias each of the dominant visual pathways (magnocellular and parvocellular) in separate stimulus conditions (Motion/Magnocellular and Color/Parvocellular). For each condition, group differences were explored in adults, ages 18 to 28 years, between individuals with dyslexia (experimental group) and individuals without dyslexia (comparison group) (see Table 5).

Table 5

Planned Group Comparisons

Condition/ ERP Measure	Experimental Group Individual With Dyslexia			Comparison Group Individual Without Dyslexia		
Motion/MAG Amplitude	P1	N1	P2	P1	N1	P2
Motion/MAG Latency	P1	N1	P2	P1	N1	P2
Color/PAR Amplitude	P1	N1	P2	P1	N1	P2
Color/PAR Latency	P1	N1	P2	P1	N1	P2

Independent variables: Group—Experimental Group/Individuals with dyslexia and Comparison Group/Individuals without dyslexia; Condition—Motion/MAG (= stimuli designed to bias the magnocellular pathway) vs. Color/PAR (= stimuli designed to bias the parvocellular pathway).

Dependent variables: ERP components—Adaptive mean amplitude and mean peak latency for the P1, N1, P2 for each condition. For planned comparisons, refer to condition/measure and the color-coded rows (P1/blue columns, N1/gray columns, and P2/yellow columns).

Time windows and scalp distribution of electrodes for each of the components (P1, N1, P2) were determined based on industry practices for capturing early visual components. The adaptive mean amplitude (in microvolts, μV) was derived from the electrodes of interest for each time window. The data were evaluated for outliers, normality, and homogeneity. If any of these assumptions were violated, appropriate constraining procedures were applied to the statistical analyses. The data analysis strategy, pre- and post-processing parameters, data analysis procedures, and analysis for this study are reviewed in Chapter 6, Data Processing and Analysis.

4.2 Participants

This study involved two groups of participants. Both groups included adults 18 to 28 years of age: one group with documented dyslexia; the other, a comparison group consisting of individuals without dyslexia. To detect any visual sensory deficiencies that could interfere with

the processing of visual stimuli, all participants were screened for normal or corrected-to-normal vision, normal color vision, and contrast sensitivity. To be eligible as a comparison participant (one without a documented reading disability), an individual had to report no history of learning disability, language disorder, or brain damage. Exclusion criteria for all participants included the following: uncorrected deficiency in visual acuity, colorblindness, poor contrast sensitivity, or history of seizure. Additionally, English was the primary language of all participants.

4.2.1 Recruitment and Informed Consent

Recruitment of participants included informal channels such as through contacts of the principal investigator, libraries, advocacy groups, Neurocognition of Language Lab Facebook page, and the Teachers College online bulletin. Participants received gift cards or cash remuneration for taking part in the experiment. All recruitment, consent, and other study procedures were carried out under the approval of the Teachers College Institutional Review Board for the Protection of Human Subjects (see Appendix B).

Before arriving at the lab, participants received an email providing an overview of the visit, including duration of screening, types of assessments, and procedures involved in EEG recording (Appendix C). Upon arrival, participants were given a tour of the lab and were again apprised of the schedule. Participants were provided a consent form (Appendix D) to review and sign, after which the baseline screenings were conducted. All participants were encouraged to ask questions, and it was emphasized that participants could withdraw from participation in the experiment at any time during the course of the visit without penalty.

4.2.1.1 Verification of developmental dyslexia diagnosis. Adult participants with developmental dyslexia were asked to verify that a qualified professional had provided a formal diagnosis of developmental dyslexia or specific learning disability with impairment in reading. The

latter is the more typical diagnostic term (American Psychiatric Association, 2013). However, "dyslexia is an alternative term used to refer to a pattern of learning difficulties characterized by problems with accurate or fluent word recognition, poor decoding, and poor spelling abilities" (p. 67). Therefore, either diagnosis was considered acceptable as an inclusion criterion. The consent form included a statement of diagnosis and required signature (Appendix C).

4.2.1.2 Statistical power and sample size. Many factors contribute to the small sample sizes of reported EEG studies and the difficulty in generating power estimations. Using statistics to analyze EEG and ERP data has its own issues (Picton et al., 2000; also see Keil et al., 2014 for an overview of some of the issues involved in statistical approaches to analyzing EEG and ERP data). EEG researchers generally consult previous studies for guidance on which study parameters can control factors that might contribute to Type II errors. A review of five EEG studies, chosen for their population and focus exploring early visual components related to development (Campbell & Sharma, 2016; Charollais et al., 2016; Coch et al., 2005; Doucet, Gosselin, Lassonde, Guillemot, Lepore, 2005; Mitchell & Neville, 2004), revealed a rather tight range of participant group sizes (Table 6), with an average of 15.39 participants per group. ERP studies typically involve 10-20 participants per group (Luck, 2014), which was the aim of the recruitment efforts. Despite broad recruitment efforts, seven individuals with dyslexia and 16 individuals without dyslexia were recruited. Statistical power can be increased by other factors, such as having more trials, artifact detection thresholds, trial averaging, and baseline correction (Luck & Gaspelin, 2017). ERP components are considered small or large, depending on the number of necessary trials for reliable detection. The signal-to-noise ratio—that is, the relative size of the signal (the ERP) in contrast to the size of the noise (the background EEG)—can be greatly influenced by the number of trials. The components of interest in this study—P1, N1, and

Table 6

Sample of Group Size in Related Studies

Study	Method	Study Focus	Sample Size
Charollis et al. (2016)	ERP	ERP variability as related to reading ability	2 groups (adults/children); Average per group: 18
Campbell & Sharma (2016)	ERP	Visual cortical development	3 groups (children); Average per group: 13.6
Coch et al. (2005)	ERP	Visual pathway development (magnocellular & parvocellular)	4 groups (adults/children); Average per group: 20
Doucet et al. (2005)	ERP	Visual maturation	6 groups (adults/children); Average per group: 10.3
Michelle & Neville (2004)	ERP	Visual pathway development (magnocellular & parvocellular)	3 groups (adults/children); Average per group: 15

P2—are considered small because they can require 300-1000 trials per condition to measure reliably (Luck, 2014; Woodman, 2010). However, Mitchell and Neville (2004) and Coch et al. (2005) investigated the development of visual pathways and established that 288 trials per condition were adequate for capturing this study's components of interest. Observations from earlier stimulus testing indicated that the recording time involved in delivering 288 trials was tolerable for participants, yet provided a number of usable trials that was within the range of prior published studies.

The raw data collected measures of voltages recorded at a rate of 500 samples per second for each of 128 electrodes on each participant's scalp. Eight minutes of recording yielded 500 samples/second x 60 seconds/minute x 8 minutes/session, or 240,000 data points for each of the 128 sensors for each participant. Within these time-series data, there are two sources of variability of interest: within-participant time course variability (fluctuations from one time point to another) and within-participant experimental variability (variation in the effectiveness of the

experimental manipulations in producing a percentage signal change). Within-participant variability was minimized by ensuring trial-by-trial consistency, artifact detection, baseline correction, and averaging (Handy, 2005; Luck, 2005), and experimental design parameters were set to reduce variability where possible and hence, noise within the data.

4.3 Measures

4.3.1 Screenings

All participants underwent a pre-experimental screening to ensure that they met criteria for study inclusion. Screening procedures included testing for visual acuity, color vision, contrast sensitivity, and the Edinburgh Handedness Inventory, as detailed below.

4.3.1.1 Snellen chart. The Snellen chart presents a series of letters, with the type sizes getting progressively smaller from the top row to the bottom row. Participants stood 20 feet from the chart and read down the chart until they were unable to discern the letters. If the individual being tested could read the row labeled "20/20," that individual was deemed to have normal acuity. For those participants who wear glasses for distance, this screening ensured that a participant's best-corrected visual acuity is at least 20/20. This test of acuity measures how accurately the cornea and lens focus light on the retina at a distance of 20 feet. For the purposes of this study, measures of 20/25 were deemed acceptable given the more intermediate acuity requirements of the task.

4.3.1.2 Ishihara Color Blindness Test. This is the most common test to assess color vision deficiencies, of which red/green is the most common. It consists of 16 images filled with colored dots in different shades. Some of these dots are arranged to depict numbers. Individuals who have great difficulty detecting the hidden number are said to have a color vision deficiency for that combination of shades. The first 14 images assess red/green deficiencies. Recognition of

fewer than eight of those images by an individual would categorize that person as having a color vision deficiency and render the person ineligible to participate. There are also two screening images for yellow and blue deficiency, which is very rare. Failure to recognize both of these images would disqualify the person from study participation.

4.3.1.3 Contrast Sensitivity Function Test. The Contrast Sensitivity Function (CSF) is the visual system's threshold for detecting contrast for a range of sine wave spatial frequencies. CSF is assessed using the Pelli-Robson test (Pelli & Robson, 1988), in which the person is positioned 40 inches from a wall-mounted chart that is illuminated at approximately 85 cd/m^2. The chart follows the luminance, font, and letter spacing recommended by the National Research Council Committee on Vision (National Academy of Sciences, 1980). The chart is arranged in groups of three letters decreasing in contrast. Participants read through the chart, and their score was determined by the lowest contrast level at which they could read at least two letters. A score of 2.0 indicates normal contrast sensitivity (100%) and a score below 1.5 suggests a sensitivity impairment (Mäntyjärvi & Laitinen, 2001). Although contrast sensitivity varies by age (Mäntyjärvi & Laitinen, 2001), this study would have excluded anyone who did not score above 1.5.

4.3.2 Qualitative Measures

4.3.2.1 Edinburgh Handedness Inventory. This is a set of questions designed to quantify a participant's preferential hand, made available through BrainMapping's shared software (http://www.brainmapping.org/shared/Edinburgh.php). Those taking the assessment indicate right hand, left hand, or no preferred hand for a variety of activities. Responses result in an assessment of handedness as measured by a laterality index and decile score (Oldfield, 1971). Handedness was not an exclusionary criterion for this study; however, it provides suggestive

information about cerebral laterality and contributes potential insights into the interrelationships between the items inventoried and possible ERP differences.

4.3.2.2 Participant questionnaire. A questionnaire was completed by all participants. It was developed to probe issues with spelling, memory, organization, sequencing, and the general reading experience (see Appendix E). Some of the questions were adapted from the checklist featured on the Bristol Dyslexia Centre website (http://www.dyslexiacentre.co.uk/signs-of-dyslexia/) as well as the International Dyslexia Association (http://www.interdys.org/AreYou Dyslexic_AdultTest.htm). This information was collected to provide a qualitative description of the participants' self-reported background and strengths and weaknesses.

4.3.3 Quantitative Measures

4.3.3.1 Standardized assessments. Components of the Comprehensive Test of Phonological Processing (CTOPP-2; Wagner, Torgensen & Rashotte, 1999); the Woodcock Reading Mastery Test-Revised (WRMT-R/NU; Woodcock, 1998); Test of Word Reading Efficiency—Second Edition (TOWRE-2) (Torgesen et al., 1999); and Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV) (2009) were administered to all participants to provide behavioral data for group and correlation analyses. As some of the participants fell outside of the normed age range, for the CTOPP and TOWRE-2 subtests, raw scores were used for purposes of group comparison. For the correlation analysis, raw scores from all assessments were correlated with neurophysiological measures.

4.3.3.1.1 Comprehensive Test of Phonological Processing (CTOPP; Wagner, Torgensen, & Rashotte, 1999). The subtests administered from the CTOPP were elision, word blending, phoneme isolation, rapid letter naming, and rapid digit naming.

4.3.3.1.2 Woodcock Reading Mastery Test-Revised (WRMT-R; Woodcock, 1998). From the WRMT-R, the word identification and word attack subtests were used. These tests were selected to quantify participants' reading fluency.

4.3.3.1.3 Test of Word Reading Efficiency—Second Edition (TOWRE-2) (Torgesen et al., 1999). The *Sight Word Efficiency (SWE)* and *Phonemic Decoding Efficiency (PDE)* tests were administered to provide timed measures.

4.3.3.1.4 Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV) (2009). Delivered via the Q-interactive® Web-based Administration and Scoring platform, only the Digit Span subtest was administered which included *Digit Span Forward*, *Digit Span Backward*, and *Digit Span Sequencing* assessments. Collectively, this subtest measures working memory, mental manipulation, cognitive flexibility, rote memory and learning, attention, and encoding.

4.3.3.2 Non-standardized measures.

4.3.3.2.1 ***Direction discrimination task.*** The stimulus parameters specified in the motion null task as well as for the EEG motion condition (see Table 6) may not be optimal for all participants. For example, studies have suggested that different aspects of human visual function development, such as contrast sensitivity, spatial frequency discrimination, and temporal frequency discrimination, occur at different rates (Ellemberg, Lewis, Liu, & Maurer, 1999; Gordon & McCulloch, 1999; Lewis & Maurer, 2005). Additionally, sensitivity thresholds for such aspects of vision as spatial frequency are typically established based on measures obtained from adults with normal vision rather than from children or special populations. Therefore, this behavioral task assessed a participant's ability to perceive and judge the direction of a drifting luminance-defined Gabor-like patch (moving grating inside a static window with smooth edge)

at varying spatial and temporal frequencies as well as at two contrast levels. This direction discrimination task established that all participants perceive motion consistent with the parameters of the motion null task and the EEG motion condition; additionally, it provided some insights into task performance (reaction times) differences between groups.

Stimuli for the direction discrimination task were generated using Psykinematix software (KyberVision, Sendai, Japan, psykinematix.com, 2016) and presented on a LCD monitor. Participants were seated 46" from the monitor and viewed a 2° static circular window with smooth Gaussian edges enveloping a grating of varying spatial and temporal frequencies (see Figure 10) presented center screen against a consistent gray background (mean luminance of 85 cd/m^2). Participants indicated whether the randomly presented conditions appeared to be traversing leftwards or rightwards using arrow keys on a standard computer keyboard. Both accuracy and response time data were collected to confirm that each participant's accuracy rate was above 80% and to determine whether groups varied significantly in their response times to particular sets of parameters related to the motion null task and/or the EEG motion condition. The task included five different sets of parameters (see Table 7), the first of which simulated the parameters used in the motion null task. Three other sets of parameters explored different combinations of spatial frequencies (6 cpd and 4 cpd) and temporal frequencies (10 cps, used in the EEG motion condition, and 2.5 cps used in motion null task). The first four sets of parameters were all presented at 20% contrast, the initial contrast-level setting in step one of the motion null task. The final set of parameters replicated the EEG motion condition parameters (1 cpd, 8% contrast, 10 cps).

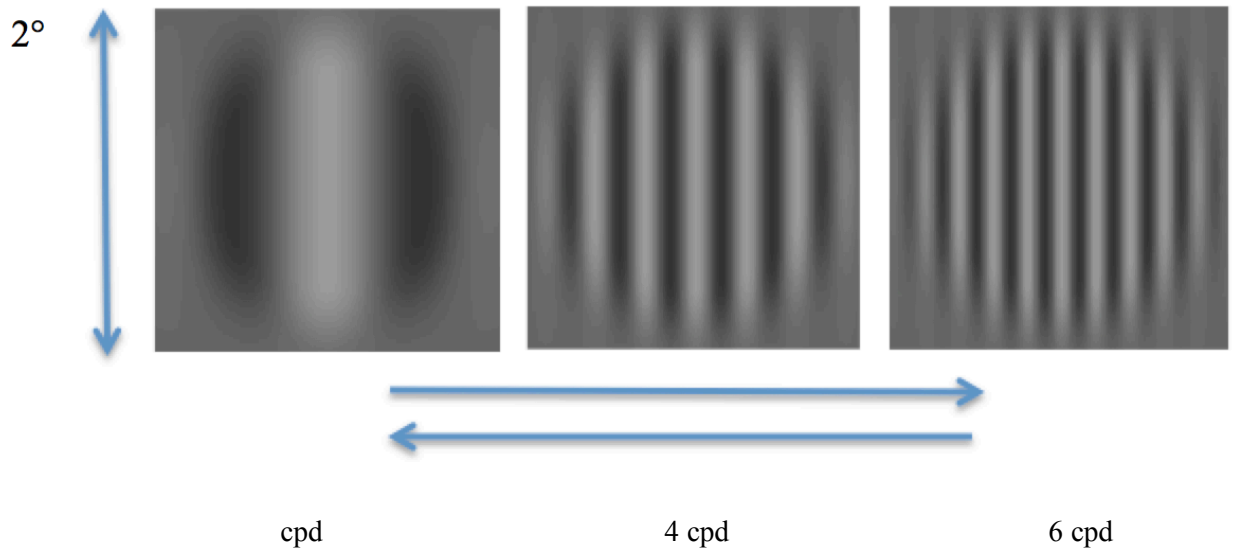

Figure 10. Direction discrimination task
Smooth Gaussian edge, presents at three different spatial frequencies (cpd—cycles per degree). Image to traverse left to right or right to left at three different cycles per second (cps).

Table 7

Parameters for the Direction Discrimination Task

Direction Discrimination Parameters	Cycles per degree (cpd)	Contrast	Cycles per second (cps)
1 – Motion Null Task Parameters	6 cpd	20%	2.5 cps
2 – Same SF as Motion Null/Higher TF	6 cpd	20%	10 cps
3 – Lower SF/Same TF as Motion Null	4 cpd	20%	2.5 cps
4 – Lower SF/Higher TF	4 cpd	20%	10 cps
5 – EEG Motion Condition	1 cpd	8%	10 cps

Accuracy and reaction times will be collected for each set of parameters. Parameters differ in their spatial frequencies (SF) indicated by cycles per degree (cpd) and temporal frequencies (TF) indicated by cycles per second (cps). Parameters 1 and 5 mimic the parameters of the motion null task and EEG motion condition respectively. Conditions 2-4 test a range of parameters that may contribute to performance on the motion null task.

Each of the conditions was presented randomly in one block 40 times (total 200 presentations), with left/right motion randomly selected for a total of 20 left trials and 20 right trials for each block. This task took approximately 5 minutes to complete.

4.3.3.2.2 Orthographic and homophone choice tasks. Two tasks were used to evaluate orthographic processing, or coding, while minimizing the use of phonological processing in response generation. Standardized measures of orthographic skills typically provide a comparative measure of the integration of orthographic and phonological skills. However, the *Orthographic Choice Task* (Olson, Wise, Conners, Rack, & Fulker, 1989; Sperling, 2004; Sperling, Lu, Manis, & Seidenberg, 2006;) and *Homophone Choice Task* (Olson et al., 1989; Sperling et al., 2006) are non-standardized tasks. The Orthographic Choice Task requires participants to make lexical decisions to 64 pairs of phonetically matched pseudo- and real words (e.g., tight/tite [exception]; sheep/sheap [regular]). For the Homophone Choice Task, participants select which of a phonetically matched pair of possible answers is appropriate for answering a question (e.g., Which is a color? blue/blew). There are 60 questions (Sperling, 2004; Sperling et al., 2006) (see Appendices E and F for samples). Raw scores from these measures were used for the purposes of group comparison and correlation with neurophysiological measures.

4.3.3.2.3 Nonverbal visual reasoning/memory (Larson, Buethe, & Vitali, 1990; publisher Slosson Educational Publications, Inc.). This task is a subtest of the Comprehensive Test of Visual Functioning assessment which was modified to improve presentation of the images. Participants were presented with a series of shapes within an 8½" x 11" laminated flipbook format. Participants were asked to remember the sequence of shapes. The flipbook page was then turned, and the participant was asked to recall the sequence of shapes presented among four options. The task builds from two shapes to nine shape sequences. Raw scores from these

measures were used for the purposes of group comparison and correlation with neuro-physiological measures.

4.3.3.3 ERP experimental stimuli. The stimuli for the EEG experiments (see Figures 11 and 12 below) were generated and presented using Psykinematix software (KyberVision, Montreal, Canada, psykinematix.com). Each stimulus is intentionally biased toward the magnocellular pathway (motion stimulus) or the parvocellular pathway (color stimulus). The stimuli vary only in the characteristics that will evoke a pathway-specific response, and are otherwise identical, featuring an approximately 2° diameter circle centered on a monitor screen with a surrounding gray background (average mean luminance of 84.5 cd/m^2). A circle appears as a smooth Gaussian edge intended to minimize the generation of artifacts caused by distinct edges. The duration of target flash stimulus (color & motion flash) is 100 ms, and the baseline image stimulus interval (ISI) varies randomly from 600 to 1000 ms (possible intervals: 600, 700, 800, 900 or 1000 milliseconds; mean 800 milliseconds). Stimuli were presented in two separate blocks, one block for the motion stimulus and one block for the color stimulus, each consisting of 320 stimulus presentations. Each block consists of the baseline image, interrupted by 288 target 100 ms flashes, plus 32 randomly presented attentional targets (emoticons, representing 10% of total trials).

Comfortably seated 46" from the monitor with a lap desk and button box, participants were instructed to look at the center of the screen to view the experimental images. Participants were asked to press a button to advance the experiment only when an emoticon displayed (such as a running man or smiley face). Participants were advised that should they need to shut their eyes for a break or adjust positions in the seat, they should do so when an emoticon was on

screen. When ready to continue, the participant pressed a button to advance. This procedure was followed for both conditions.

4.3.3.3.1 Motion Null Task—Equiluminant color stimulus parameter calibration. The color stimulus consists of color bars (gratings) presented inside an approximately 2° diameter circle, center screen. The baseline image has blue/green bars, and the target color flash has red/green bars. To attribute a brain response to the parvocellular pathway, the color pairs of the color stimulus must be isoluminant, so that a response from the luminance-sensitive magnocellular pathway is not evoked (Lu & Dosher, 2014). Perception of luminance varies among individuals (Lu & Dosher, 2014), making it necessary to determine each participant's equiluminant settings (Cavanagh, 1991). To establish isoluminace between the bars of a color pair used in the color stimulus, this study utilized the motion null technique, sometimes referred to as the minimum motion technique, which has been used in animal studies as well as studies involving humans (Anstis & Cavanagh, 1983; Cavanagh, MacLeod, & Anstis, 1987; Logothetis & Charles, 1990).

The motion null task involved a two-step process. Step one established the individual's perception of equiluminance between blue and green. Seated 46" from the monitor, each participant viewed an approximately 2° circle with a smooth Gaussian edge presented center screen. With elbows resting on the arms of a chair and hands extended perpendicularly, participants were instructed to focus on the presenting image and to open and close their left or right hand in the direction of the "moving" bars. When the bars appeared to be flashing (motion null), the participant was directed to use both hands to gesture flashing. The investigator used a keyboard to input responses, hitting the left or right arrow key to increase or decrease the brightness of the color green relative to the color blue. When the participant indicated a flashing

gesture, the direction opposite the previously selected arrow was recorded, reversing directions. The procedure resulted in the convergence of an isoluminant value after a few reversals.

In step two, the luminance setting obtained for the color green from step one was manually inputted into the experimental script for step two. The procedure was then repeated for the other color pair, so that both stimulus color pairs (blue/green and red/green) ended up with equal perceived luminance values against a consistent gray background (mean luminance of 85 cd/m^2).

This technique can be affected by eye movements, which interfere with the stability of the percept. Additionally, the task is likely more difficult for some due to lack of experience with the task, or particular disorders. To address these issues, for each color pair, two measures were obtained and averaged to provide equiluminant settings. If the measures obtained differed by more than a few points, the instructions were explained again and the task repeated. Participants unable to complete the motion null task would have been excluded from the study. The motion null task took approximately 10 minutes to complete. A detailed explanation of the task parameters is given in Appendix A.

4.3.3.3.2 EEG color stimulus for the color condition of the experiment. The stimuli for the color condition (Figure 11) was designed to bias the parvocellular pathway. They are high-spatial-frequency gratings of 6 cycles per degree. In the baseline image, the bars are blue and green with luminance (RGB values) set for each participant based on the results of the motion null task described above. Baseline image duration was randomly varied from 600 ms to 1000 ms in 100ms steps. At the end of each baseline, there was a switch from blue/green color bars (baseline image) to red/green color bars (target flash). This switch lasted 100 milliseconds and appeared as a flash with no traverse movement, luminance, or pattern change. Because the

interval between flashes was relatively long, this is considered a low temporal frequency stimulus. The stimulus features (color-opponent, low-temporal frequency high-spatial frequency, and neutral contrast) have been found to engage the parvocellular pathway (Derrington & Lennie, 1984; De Valois, Albrecht, & Thorell, 1982; De Valois, Morgan, & Snodderly, 1974; Lennie et al., 1990; Livingstone & Hubel, 1988).

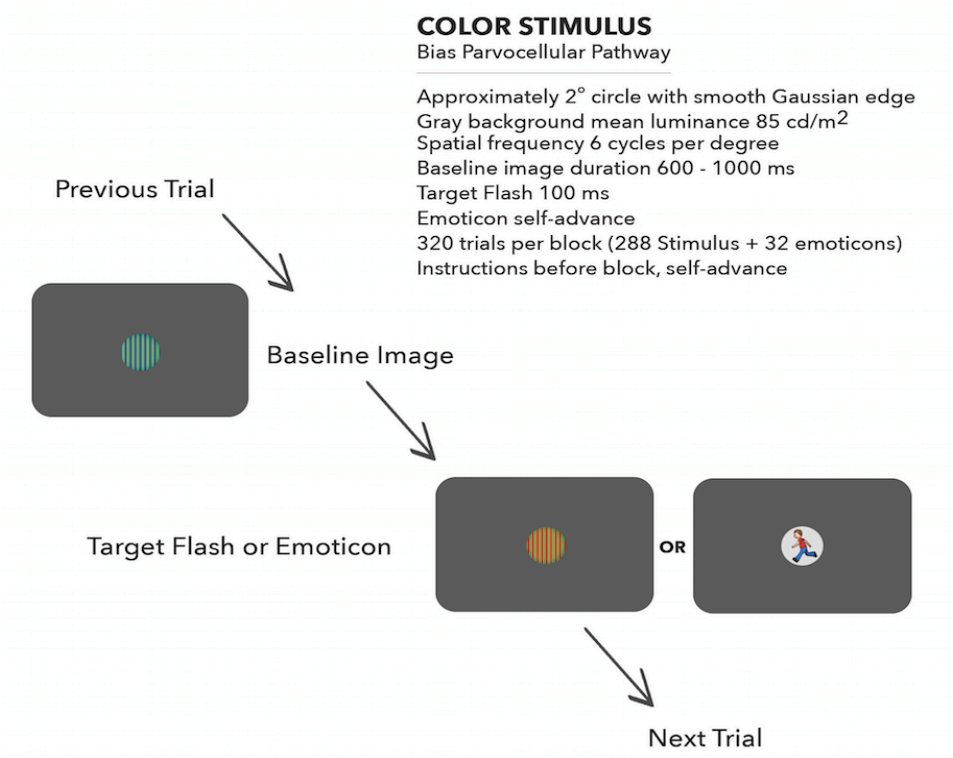

Figure 11. Steps for delivering the EEG color stimulus

4.3.3.3.3 EEG motion stimulus for the motion condition of the experiment. The stimulus for the motion condition (Figure 12) was designed to bias the magnocellular pathway. It is a monochrome grating with a spatial frequency of 1 cycle per degree and a smooth Gaussian edge (baseline). Baseline image duration was randomly varied from 600 ms to 1000 ms in 100ms steps. Periodically, the image traversed from left to right at approximately 10° per second (target

flash). This velocity was generated by a 72° phase-shift per frame, corresponding to a 1-cycle phase-shift (or 1° of visual angle) in 0.83 ms (5 frames), which is considered a high-temporal frequency. The stimulus contrast is at 8%, which is a low-contrast setting. These features of the stimuli (high-temporal frequency, low contrast) are known to engage the magnocellular pathway (Hubel & Livingstone, 1990; Jackson et al. 2013; Kaplan & Shapley, 1986; Murav'eva, Deshkovich, & Shelepin, 2009; Shapley, Kaplan, & Soodak, 1981; Tootell, Hamilton, & Switkes, 1988).

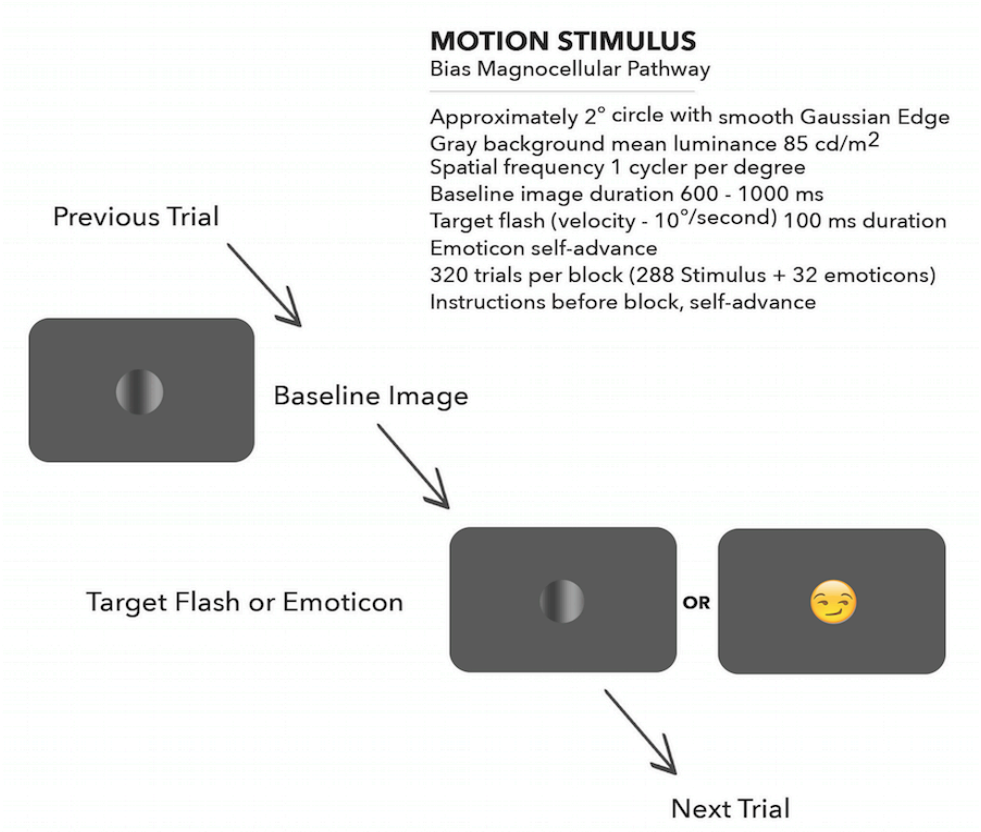

Figure 12. Steps for delivering the EEG motion stimulus

4.4 Experimental Equipment and Procedures

4.4.1 Equipment and Data Recording

A Power MAC mini, Intel Core 2 Duo (8 GB memory), including an NVIDIA GeForce 320M Graphic card, running OS X 10.10 (Yosemite), compatible with Psykinematix software (KyberVision, Sendai, Japan, psykinematix.com) was used to generate and run the motion null task, the EEG experimental conditions, and the direction discrimination task. Visual stimuli were presented on a 24" NEC MultiSync PA241W LCD display with a native resolution of 1920 x 1200 and a refresh rate of 60 Hz. The continuous EEG data were recorded using EGI's Netstation (v4.3) data acquisition software run on an Apple MAC Pro, sampling at 500 Hz (Electrical Geodesics Inc., Eugene, OR). Anti-aliasing filters (0.1-100 Hz band pass) were automatically applied during digitization of the analog recording. EEG data were recorded in a sound-attenuated, electrically shielded room with humidity and ambient temperature controlled.

4.4.1.1 Peripheral equipment. To calibrate the monitor's luminance, or brightness levels, a Minolta LS 100 photometer and a Spyder Elite 4 colorimeter were used. A Cedrus Response Box RB-730 was used to confirm participant engagement by recording button presses to the appearance of an emoticon during stimulus presentation. A Cedrus Stim Tracker was used to conduct timing tests and provide offset values for data preprocessing. The ambient room lighting was maintained at consistent levels throughout the stimulus calibration, behavioral task, and experimental blocks (18.1 volts—suitable for photopic vision).

4.4.1.2 Electrode nets. EEG data were collected from (130 series) high-density, 128-channel hydrocel Ag/AgC1 electrodes, made of carbon fiber silver chloride and embedded in soft sponges woven into a geodesic array connected to a high-impedance (200 series) amplifier manufactured by EGI (Electrical Geodesics; Tucker, 1993). This system permits the rapid

and accurate use of numerous electrodes in high-density arrays with minimal time while maximizing participant comfort and safety. Each net was soaked in a potassium-chloride solution (2 teaspoons potassium chloride, 1 liter of water purified by reverse osmosis, and 3 milliliters of Johnson & Johnson baby shampoo to remove oils from the scalp) for 5 minutes to minimize impedances and ensure optimal conductivity. The high-density hydrocel nets and associated high-impedance amplifiers were designed to accept impedance values ranging as high as 100kΩ, which permits the sensor nets to be used without scalp abrasion, recording paste, or gel (e.g., Ferree, Luu, Russell, & Tucker, 2001; Pizzagali, 2007). Impedances for all electrodes were measured before data collection and between blocks and were kept below 40kΩ.

4.4.2 Participant Lab Visit and Data Collection Overview

Thirty minutes prior to each EEG recording session, the experimental display monitor was turned on to allow the luminance levels to stabilize at 200 cd/m^2. Amplifier calibration, including zero and gain measurements, were conducted prior to each run. Additionally, timing tests to track stimulus offset, which permits identification of the interval between stimulus recoding and stimulus presentation, were conducted for each recording session.

Upon entering the lab, participants were given a tour of the facilities and provided with a full verbal description of the procedures and risks, including an opportunity to ask questions. Further opportunities and encouragement to ask questions were provided throughout the session. Participants signed the consent forms and were reminded that they could withdraw from participation at any time. Screenings were then conducted, followed by completion of the online handedness survey adapted by BrainMapping.org from the Edinburgh Inventory. A paper-and-pencil questionnaire was also completed by participants. After the initial screenings, the

sequence of the assessments and EEG recordings was counterbalanced across participants and a break provided between activities.

Regardless of the order of assessment or EEG recording, both were preceded by the motion null calibration and the direction discrimination task. Participants were comfortably seated and instructed to view the monitor. The motion null calibration was explained before and while practicing the task. Participants were given ample practice opportunities and, when ready, completed the task twice. Two values were obtained for each of the two steps of the calibration process and then averaged. The luminance values for both green and red were then used to customize the color stimulus (entered into the Psykinematix graphical user interface by the principal investigator). After completing the task, participants were asked to select a brain-shaped stress ball from a black nylon bag initially containing 20 blue and 20 purple stress balls. The random selection (no replacement based on 20 participants per group) of a ball provided the sequence of experimental conditions (blue/motion and purple/color) during the EEG portion of the session. This brief activity provided a break from the monitor. Participants then completed the direction discrimination task.

For the EEG portion of the experiment, participants were seated in a comfortable wooden chair 46" away from the monitor screen in the EEG chamber. The circumference of each participant's head was measured and the vertex of the participant's head marked to ensure accurate placement of the net. The appropriately-sized electrode net was then soaked in potassium chloride solution, as previously described, for 5 minutes, and the net was fitted on the participant's head. Before running the EEG session, impedances (loss of signal between scalp and sensor) were measured by feeding a minute (400 microvolt) electrical field through each electrode, and then having it measured by the acquisition system so that the amount of signal loss

could be calculated. Once impedances were measured and any problematic electrodes repositioned, those electrodes exceeding 40kΩ impedances were identified and noted in the run logbook. Before beginning the recording session, participants were shown how their brain data displays on the screen across electrodes and provided with instructions aimed at reducing movement artifacts. The instructions included a demonstration of the unwanted effects of various body movements (eye blinks and saccades, head turning, foot tapping, etc.) on the EEG recording, plus an explanation of when during the programmed presentation such movements would have the least impact (when emoticons are presented). Lastly, participants were asked to try to refrain from moving as much as possible and to wait for the appearance of an emoticon to blink or adjust.

Brief online instructions, a repeat of the previous verbal description of the task, were presented on screen at the start of the experimental block. Participants read the instructions and indicated via button press when they were ready to begin the experiment. All participants were continuously monitored by video feed to the data collection station outside the EEG chamber.

When the first condition (motion or color, counterbalanced) was completed, the experimenter entered the room to check on the participant and confirmed his or her willingness to continue. Electrode impedances were measured and re-established before proceeding with the second and final condition. At the conclusion of the EEG portion of the experiment, the net was removed from the participant's head.

The assessment portion of the session was completed in a quiet room with minimal distractions. Components of the CTOPP, WRMT-R, TOWRE-2, WAIS-IV were administered as well as the orthographic and homophone choice tasks and nonverbal reasoning/memory task.

Upon completion of the assessment and EEG portions of the experimental session, remuneration was provided and participants were encouraged to share their impressions of the experience and ask any further questions.

5. DATA PROCESSING AND ANALYSIS

5.1 Pre-/Post-Processing

Recorded raw EEG data were pre-processed following procedures outlined in Picton et al. (2000), Keil et al. (2014), and Luck (2014). NetStation software (v4.5.7, Electrical Geodesics, Inc.) was used to conduct data pre-processing.

First, continuous raw EEG data were digitally filtered offline using a 0.3 high-pass filter and a 30 Hz low-pass filter (FIR Passband Gain: 99.0 % [-0.1 dB], Stopband Gain: 1.0 % [-40.0 dB], Rolloff: 2.00 Hz). The data were segmented into epochs of 500 ms that included 100ms prior to stimulus onset and 400ms following stimulus presentation. The segmentation protocol also incorporated an offset that reflects a necessary millisecond correction due to an expected delay between the timestamp (time reported by experimental control module) and the actual time the stimulus was presented onscreen to the participant (Electrical Geodesics, 2015). The offset value was acquired by running timing tests prior to each run using a Cedrus Stim Tracker.

The segmented data were then subjected to automatic artifact detection and bad channel replacement protocols to remove eyeblinks and physiological artifacts (e.g., electrocardiogram, electromyogram, electrooculogram). Electrode channels that exceeded 200 microvolts (μV) were replaced using spherical spline interpolation from data acquired at surrounding sensors. Trials were discarded from analysis if they contained eye blinks (EOG >140 μV), or if more than 40% of the channels were bad. Following the automatic artifact rejection protocol, trial segments were manually reviewed and marked as bad if necessary.

Baseline correction was then carried out with respect to a 100ms portion of each epoch preceding stimulus presentation. This portion of the total epoch reflects random activity not associated with stimulus processing, which can introduce significant variance to the data, making group differences more difficult to observe. Baseline correction minimized such confounds by averaging the amplitude at all points across the pre-stimulus segment and then subtracting that value from the samples in the post-stimulus segment (Luck, 2014).

Data were then re-referenced from the vertex electrode (applied during recording) to the average of all electrodes. As a final step, all trials for each participant were averaged to generate the ERP waveforms within individuals and within conditions, so that event-related brain activity most relevant to the stimulus presentation could be observed and further analyzed (Luck, 2014). The pre-/post-processing protocol was completed for all recorded data.

5.2 Data Analysis Protocol

Post-processed averaged ERP data files for each participant and condition were exported from NetStation for statistical analysis. Post-processed data files were read into an R database (R Core Team, 2016) and measures of amplitude and latency obtained. R scripts developed in-house specifically for this experiment were used to obtain peak latency and adaptive mean amplitude measures for each component. Peak latency measures were calculated by identifying the maximum positive and negative voltage deflection within a pre-selected time window. These values were then used to calculate the adaptive mean amplitude, which selected five samples or a 10 ms window on either side of the identified peak latency and averaged the sampled amplitude values. Measures for each target component (P1, N1, P2) were calculated based on expected scalp topography represented by specified electrodes and time windows from prior studies (Coch et al., 2005; Luck, 2014; Woodman, 2010) and adapted for a high-density recording net.

For this study, the recording sites selected for statistical analysis focused on scalp locations in the occipital area. All components (P1, N1, P2) were represented by an electrode montage that included electrodes 69, 70, 74, 75, 82, 83, 89 (see Figure 13 below). This montage was used to identify peak latencies and calculate adaptive mean amplitudes for each component's pre-selected time window (P1: 95-150 ms; N1: 100-200 ms; P2: 200-300 ms). Individual files were grand-averaged together to produce ERP waveforms by group (experimental group/DYA vs. comparison group/TDA) for each condition and component (P1, N1, P2), using R.

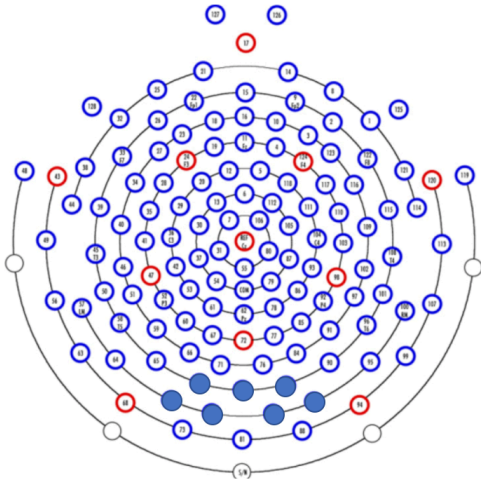

Figure 13. Sensor layout of the 128-channel hydrocel geodesic sensor net
Shows electrode distribution for the P1, N1, and P2 components

Based on the a priori research questions, the following statistical analyses were applied to the individual and grand-averaged files. Assumptions of homogeneity and normality were investigated prior to conducting between-group analyses (Levene's statistic and Shapiro-Wilk test). A mixed repeated-measures analysis of variance provided p-values for differences between group means for each of the components within the two conditions.

The relationships between the obtained latency and adaptive mean amplitude ERP measures and the behavioral measures were first explored using Pearson's product-moment correlations. The data were evaluated for linearity and normality as well as to identify outliers using scatterplots and the Shapiro-Wilk test.

In the next chapter, I report the results obtained using these parameters and methods.

6. RESULTS

6.1. Participants

Participants with a minimum of 110 usable trials out of a total of 288 trials (38% of trials per condition) were included in statistical analyses. This excluded four individuals from the comparison group (TDA: typically developing adults/individuals without dyslexia). All participants successfully completed the Motion Null and Direction Discrimination tasks. The final groups consisted of 12 individuals without dyslexia (the TDA group: 7 female, 5 male) and seven individuals with dyslexia (the DYA experimental group: 5 female, 2 male). The mean age was 20.42 years (SD = 2.35) for the final comparison group, and 23.43 years (SD =3.78) for the experimental group. While variance was homogeneous, as assessed by Levene's test of equality of variance (p = .220), the mean age difference between groups was statistically significant (t (17) = -2.16, p = .046).

The mean number of usable trials for the motion/magnocellular condition was 208.26 (SD = 50.24); by group, the average number of usable trials was 222.57 for the DYA group (SD = 55.35) and 199.92 for the TDA group (SD = 47.47). The mean number of usable trials for the color/parvocellular condition was 199.84 (SD = 47.08); by group, the mean number of usable trials was 212.29 for the DYA group (SD = 50.33) and 192.58 for the TDA group (SD = 45.72). For adults with and without dyslexia included in the analyses, there was homogeneity of variance, as assessed by Levene's test of equality of variance (motion stimulus: p = .823; color stimulus: p = .900). Data from both groups were normally distributed as measured by the Shapiro-Wilk's test (motion/magnocellular condition: DYA p = .070; TDA p = .419; color/parvocellular condition: DYA p = .060; TDA p = .590). For each condition, independent

sample t-tests established that the mean difference between groups in numbers of usable trials was non-significant (t (17) = -.945, p = .358 for the motion stimulus; t (17) = -.874, p = .394 for the color stimulus).

6.2 Results

6.2.1 Visual Acuity, Color, and Contrast Screening Results

Measures from the Snellen visual acuity screening, the Ishihara Color Blindness Test, and the Pelli-Robson Contrast Sensitivity Chart were obtained for all participants. All participants had visual efficiency within average range (20/10 vision to 20/25 vision), normal color vision (recognition of all 14 images), and contrast sensitivity (score of 1.60 or higher). There was no difference in contrast sensitivity function as measured by the Pelli-Robson Contrast Sensitivity Chart between the DYA group (M = 1.890, SD = .109) and the TDA group (M = 1.880, SD = .108; F (1, 17) = .016, p = .932).

6.2.2 Qualitative Measures Results

6.2.2.1 Edinburgh Handedness Inventory. Of the seven participants with dyslexia (DYA) and the 12 participants without dyslexia (TDA), all were right-handed. Handedness was not an exclusion criterion.

6.2.2.2 Participant questionnaire. Participant responses were reviewed and mirrored frequently observed differences between groups (see Appendix E for sample of questionnaire/ findings). Two sample questionnaire responses, consistent with anecdotal and research findings, are shown in Figure 14.

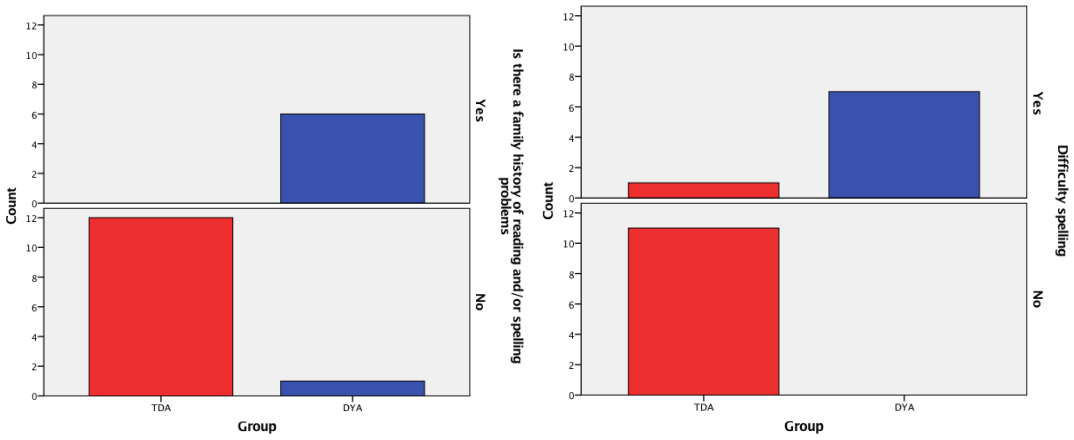

Figure 14. Questionnaire responses
Left: questionnaire responses to "Is there a family history of reading and/or spelling problems?" DYA (blue) yes = 6, no = 1; TDA (red) no = 12. Right: questionnaire responses to "Do you have difficulty with spelling?" DYA (blue) yes = 7; TDA (red) yes = 1, no = 11.

6.2.3 Quantitative results

6.2.3.1 Assessment measures. Four standardized assessments were selected for this study; however, only two assessments (Woodcock Reading Mastery and the WAIS-IV) provided standardized scores for the participants' age range (18 to 28 years). Though several of the participants were slightly older than the representative national sample upon which their standardized scoring was based, for the CTOPP and TOWRE-2 assessments, between-group differences were evaluated using analysis of covariance (ANCOVA) to account for age and gender—factors accounted for in standardized scores such as percentile rank. ANCOVAs were also used to evaluated between-group differences for other non-standardized assessments, including the Homophone Choice Task, Orthographic Choice Task, and Non-Verbal Reasoning/Memory Test.

Raw scores were obtained from the following measures: Homophone Choice Task, Orthographic Choice Task, CTOPP RAN Digit, CTOPP RAN Letter, CTOPP Elision, CTOPP Blending, CTOPP Phoneme Isolation, TOWRE-2 Sight Word Efficiency (SWE), TOWRE-2

Phoneme Decoding Efficiency (PDE), Non-Verbal Reasoning/Memory Test. Potential confounding variables such as age and gender were accounted for when comparing the normed assessments used in this study (Woodcock Reading Mastery Test Word Identification [ID] and Word Attack and WAIS-IV Digit Span). Among the assessments for which ANCOVAs were run, correlations between assessment and age (Pearson's correlation) were all non-significant, as were the correlations between assessment and age within groups (DYA/TDA) (Partial Correlations) (all *p*'s > .05; see Table 8). Group differences were explored as well, controlling for age and gender (see Table 8). Statistically significant differences between groups were found for the Orthographic Choice Task, TOWRE-2 SWE, and PDE assessments.

ANCOVA was used to determine whether groups differed in their orthographic abilities after co-varying for chronological age and gender. There was a statistically significant difference between the DYA group (M = 56.86, SD = 3.38) and the TDA group (M = 60.83, SD = 1.26) on the Orthographic Choice Task (F (1, 15) = 13.93, p = .002). Gender (p = .488) and chronological age in months (p = .250) did not significantly contribute to the between-subjects effect (see Figure 15 below).

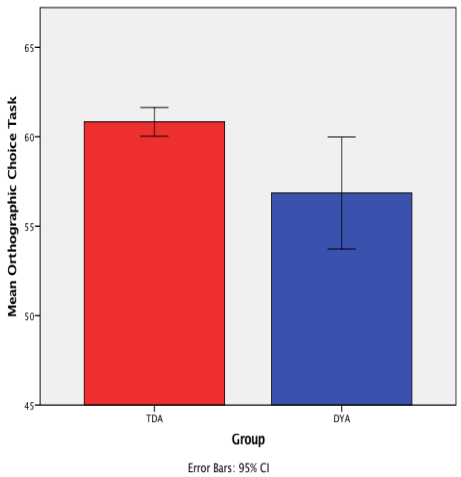

Figure 15. Group differences for the Orthographic Choice Task (Comparison group [TDA] = red; Experimental group [DYA] = blue). Mean raw scores: TDA 60.63, SD = 1.27; DYA 56.86, SD = 3.39

ANCOVA was applied to evaluate group differences in sight word efficiency after controlling for chronological age and gender. There was a statistically significant difference between the DYA group (M = 77.89, SD = 4.60) and the TDA group (M = 98.58, SD = 3.38) on the TOWRE SWE raw scores (F (1, 15) = 11.59, p = .004) (see Figure 16). Gender (p = .549 and chronological age in months (p = .269) did not significantly contribute to the between-subjects effect.

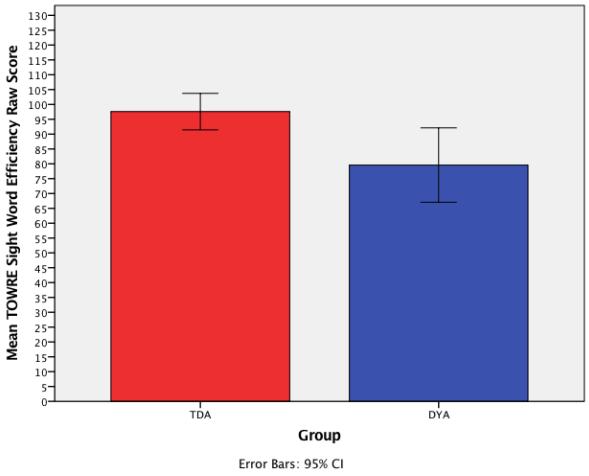

Figure 16. Group differences for the TOWRE-2 SWE assessment (Comparison group [TDA] = red; Experimental group [DYA] = blue). Mean raw scores: TDA 97.58, SD = 9.67; DYA 79.57, SD = 13.55

When conducting the between-group analysis for the phonemic decoding efficiency assessment, the assumption of homogeneity of variance was violated, as assessed by Levene's test (p = .017, see Table 8). Therefore, a Mann-Whitney U test was run. Raw scores for the DYA group (M = 41.00, SD = 11.90, Mean Rank = 4.5) were statistically significantly lower than the TDA group (M = 59.33, SD = 5.49, Mean Rank = 13.21) on the TOWRE-2 PDE (U = 3.5, p = .001).

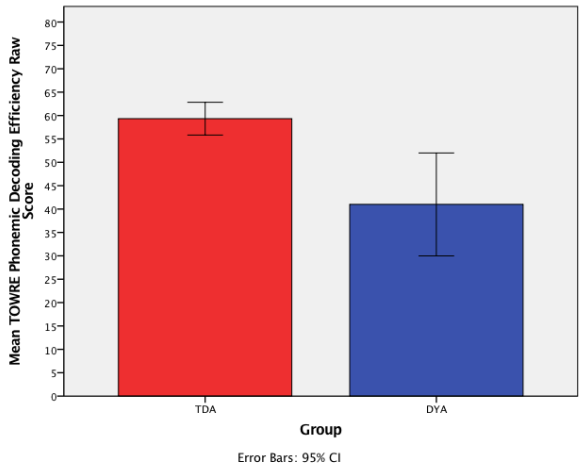

Figure 17. Group differences for the TOWRE-2 PDE assessment (Comparison group [TDA] = red; Experimental group [DYA] = blue). Mean raw scores: TDA 59.33, SD = 5.5; DYA 41.00, SD = 11.90.

ANCOVAs were applied to investigate between-group differences for all other raw score measures (Homophone Choice Task, CTOPP RAN Digit, CTOPP RAN Letter, CTOPP Elision, CTOPP Blending, CTOPP Phoneme Isolation, and Non-Verbal Visual Reasoning Memory Task); none of these differences were statistically significant (all p values > .05, see Table 8).

For the normed assessments used in this study, including the Woodcock Reading Mastery Test Word Identification (WRMT-R) Word ID, WRMT-R Word Attack, and WAIS-IV Digit Span), a one-way ANOVA was run for each, revealing only one statistically significant finding for the WRMT-R Word ID assessment. Group differences investigated using a one-way ANOVA found the mean percentile rank scores for the WRMT-R Word ID assessment between the DYA group (M = 51.14, SD = 28.45) and the TDA group (M = 77.25, SD = 11.94) to be statistically significant (F (1, 17) = 7.96, p = .012) (see Figure 18).

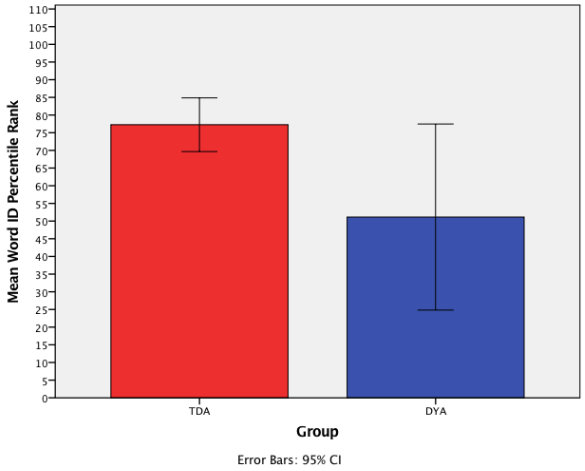

Figure 18. Group differences for the Woodcock Reading Mastery Test Word Identification assessment (Comparison group [TDA] = red; Experimental group [DYA] = blue). Mean raw scores: TDA 77.25, SD = 11.95; DYA 51.14, SD = 28.46

ANOVAs to investigate between-group differences for the WRMT Word Attack or the WAIS-IV Digit Span percentile ranks revealed no statistically significant differences (all *p* values > .05; see Table 8).

Table 8

Analyses Evaluating Between-Group Differences

Assessment	Pearson Correlation Age	Partial Correlation Group/Age	Levene's Test	ANCOVA Between Subject Effects		
Raw Score			df1=1; df2=17	Age	Gender	Group
Homophone Choice Task	.011, p = .963	.218, p = .386	F = 3.265, p = .088	p = .599	p = .290	p = .130
Orthographic Choice Task	-.060, p = .807	.350, p = .155	F = 3.262, p =.089	p = .250	p = .488	***p* = .002***
CTOPP RAN-Digit	.173, p = .479	.065, p = .799	F = 3.179, p = .092	p = .740	p = .743	p = .467
CTOPP RAN-Letter	-.071, p = .744	-.190, p = .499	F = .100, p = .756	p = .520	p = .838	p = .315
CTOPP Elision	.048, p = .847	.176, p = .485	F = 1.649, p = .216	p = .552	p = .852	p = .289
CTOPP Blending	-.074, p = .764	-.063, p = .804	F = 3.272, p = .088	p = .876	p = .798	p = .926
CTOPP Phoneme Isolation	.032, p = .895	.052, p = .839	F = .235, p = .634	p = .847	p = .986	p = .847
TOWRE-2 SWE	-.047, p = .849	.334, p = .176	F = .016, p = .902	p = .269	p = .549	***p* = .004***
TOWRE – PDE	-.260, p = .283	.114, p = .655	**F = 7.038, *p* = .017***			**Mann Whitney U test* U = 3.5, p = .001**
Non-Verbal Reasoning/Memory	.052, p = .834	.033, p = .897	F = .000, p = .991	p = .950	p = .847	p = .870
Percentile Rank				Mean	SD	One-Way ANOVA
WRMT-R Word ID	N/A	N/A	F = 3.704, p = .071	TDA = 77 DYA = 51	TDA = 11.95 DYA = 28.46	**F (1, 17) = 7.968, *p* = .012***
WRMT-R Word Attack	N/A	N/A	F = .090, p = .768	TDA = 79 DYA = 58	TDA = 19.61 DYA = 27.04	F (1, 17) = 3.829, p = .067
WAIS-IV Digit Span	N/A	N/A	F = .785, p = .388	TDA = 60.25 DYA = 38.57	TDA=31.25 DYA=23.95	F (1, 17) = 2.490, p = .133

Analyses evaluating between-group differences for the Comprehensive Test of Phonological Processing (CTOPP; Wagner, Torgensen & Rashotte, 1999) assessments: *RAN digit, RAN Letter, Elision, Word Blending, Phoneme Isolation;* Test of Word Reading Efficiency—Second Edition (TOWRE-2; Torgesen et al., 1999) assessments: *Sight Word Efficiency* (SWE) and *Phonemic Decoding Efficiency* (PDE), *Non-Verbal Reasoning/Memory Test*; Woodcock Reading Mastery - Revised (WRMT-R; Woodcock, 1998) assessments: *word identification* and *word attack* subtests; and Wechsler Adult Intelligence Scale—Fourth Edition (WAIS—IV/Pearson, 2009): *Digit Span*. Shaded rows indicate assessments for which between-group differences were statistically significant (p > .050). Due to violation of the assumption of homogeneity of variance, the Mann-Whitney U test was used.

6.2.3.2 Behavioral task results.

6.2.3.2.1 Direction discrimination task. Data were collected for the direction discrimination task from all participants. Results were evaluated for accuracy and within-subject differences by stimulus parameter for reaction time (RT). Across all participants, there was an accuracy rate of 79% and above for each direction (left/right). Therefore, the main purpose of

including the task was confirmed: each participant was able to judge the direction of motion accurately.

Table 9

Between-Group Differences on the Direction Discrimination Task

	4 cpd 20% cnt 10 cps	**4 cpd 20% cnt 2.5 cps**	**6 cpd 20% cnt 10 cps**	**6 cpd 20% cnt 2.5 cps**	**1 cpd 8% cnt 10 cps**
Participants showing significant reaction time (RT) differences by directions (Left/Right) (p > .05)	4 participants TDA = 3 DYA = 1	1 participant DYA = 1	3 participants TDA = 2 DYA = 1	4 participants TDA = 1 DYA = 3	0
TDA Mean RT (seconds) Left	M = .659 SD = .090	M = .692 SD = .005	M = .709 SD = .127	M = .738 SD = .104	M = .694 SD = .107
TDA Mean RT (seconds) Right	M = .658 SD = .088	M = .696 SD = .079	M = .699 SD = .093	M = .736 SD = .105	
DYA Mean RT (seconds) Left	M = .713 SD =.149	M = .696 SD = .063	M = .727 SD = .083	M = .835 SD = .365	M = .795 SD =.349
DYA Mean RT (seconds) Right	M = .705 SD = .091	M = .728 SD = .079	M = .704 SD = .08	M = .771 SD = .072	
Independent t-tests two-tailed p-values					t(17) = -.943 *p* = .359
Mean RT Left	t(17) = -.998 *p* = .332	t(17) = -.103 *p* = .919	t(17) = -.343 *p* = .736	t(17) = .-.876 *p* = .393	
Mean RT Right	t(17) = -1.108 *p* = .283	t(17) = -.862 *p* = .401	t(17) = -.116 *p* = .909	t(17) = -.790 *p* = .441	

cpd = cycles per degree, cnt = contrast, cps = cycles per second. M = Mean, SD = Standard Deviation. First row reports numbers of participants showing significant RT differences in direction discrimination. The far-right column with light gray shading displays condition 1 cpd, 8% cnt, 10 cps for which all responses are collapsed together to investigate mean RT differences between groups (TDA vs. DYA). For all other conditions, Left and Right mean RT considered separately. Shaded rows indicate *Left mean RT differences* for both groups and conditions, unshaded rows provide *Right mean RT differences* for both groups by condition. There was homogeneity of variance as assessed by Levene's test (all p-values > .05). Two-tail independent t-tests *p*-values reported assuming equal variance.

The mean RT for each parameter set was calculated for each participant for each direction (left/right). Paired-sample t-tests were conducted to evaluate differences between mean left/right RTs within subjects for each condition. For the first condition (1 cpd, 8% contrast, 10 cps), left/right mean RTs were not statistically significantly different within individual participants ($p > .05$). However, for the other conditions, individual differences in mean left/right RTs were found to be statistically significantly different. Therefore, group differences in mean RTs were investigated based on direction per condition (see Table 9).

For all conditions, the independent t-tests did not reveal any between group mean RT differences (two-tailed, $p > .05$).

6.2.3.3 ERP results. Based on the a priori component montage and time windows identified earlier (see Chapter 5), waveform plots were generated for each condition and component (see Figures 19, 20, 21). The time windows for the P100 and N100 overlap (95-150 ms and 100-200 ms, respectively), but the waveforms indicated that the response to each condition is best represented by a different component. For the motion/magnocellular condition within the 95-200 ms time window, both groups displayed a positive deflection, whereas for the color/parvocellular condition within the same time window, a more negative deflection was observed (see Figure 19).

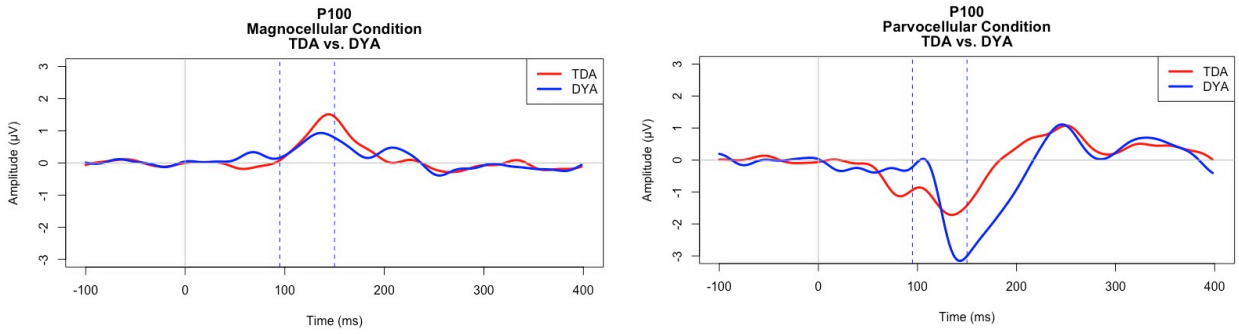

Figure 19. Time window for the P1 component
Time window for the P1 component (95-150 ms), showing a clear positive deflection for the Magnocellular/Motion condition, which is not evident in the Parvocellular/Color condition. Comparison group = TDA/red; Experimental group = DYA/blue

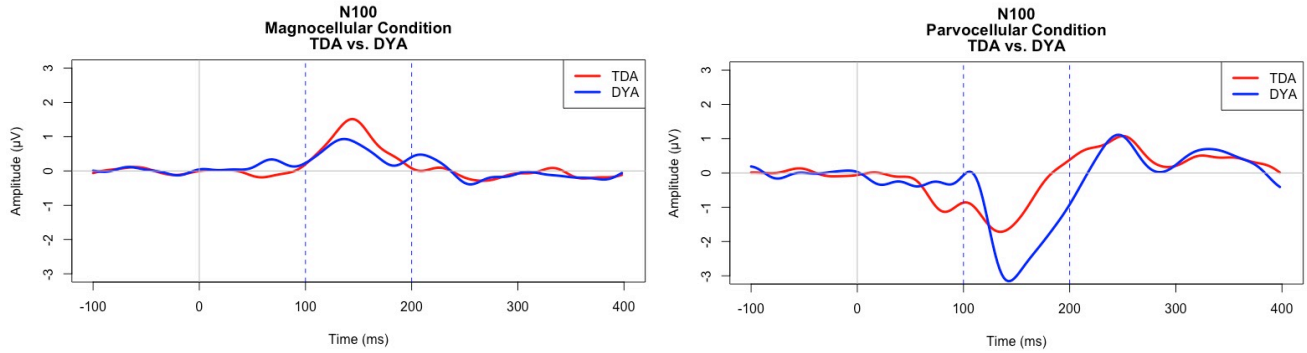

Figure 20. Time window for the N1 component
Time window for the N1 component (100-200 ms), showing a clear negative deflection for the Parvocellular/Color condition, which is not evident in the Magnocellular/Motion condition. Comparison group = TDA/red; Experimental group = DYA/blue

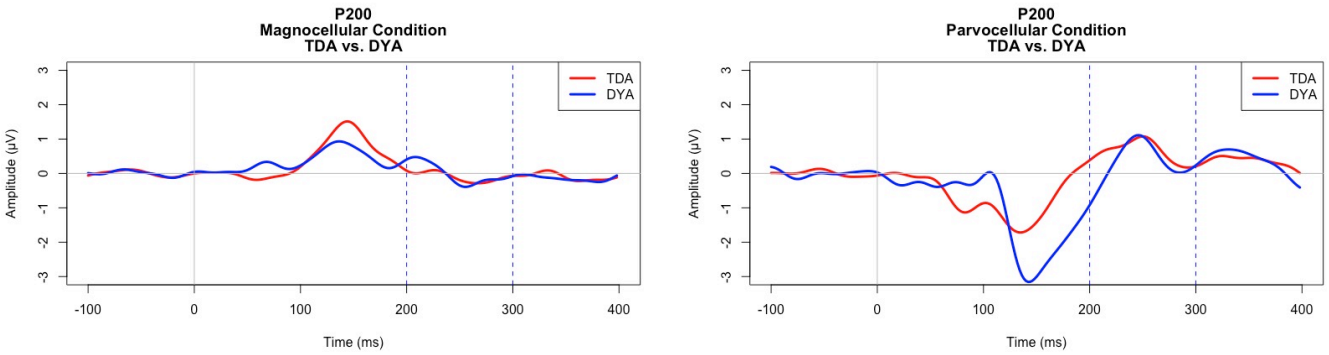

Figure 21. Time window for the P2 component
Time window for the P2 component (200-300 ms), showing a very slight positivity for the Magnocellular/Motion condition and a larger positive deflection for the Parvocellular/Color condition. Comparison group = TDA/red; Experimental group = DYA/blue

The time window for the P2 (Figure 21) reveals a positive deflection in response to the parvocellular/color condition, while there seems to be an attenuated response to the magnocellular/motion condition. Therefore, group comparisons were based on the adaptive mean amplitude and peak latency measures for the P1 in response to the Motion/Magnocellular condition, the N1 in response the Color/Parvocellular condition, and the P2 in response to both conditions.

6.2.3.3.1 Group analysis motion/magnocellular condition

Table 10

P1 and P2 Amplitude and Latency Group Measures for the Motion/Magnocellular Condition

Motion/Magnocellular Condition		
Group	**P1 Adaptive Mean** Amplitude in μV (SD)	**P2 Adaptive Mean** Amplitude in μV (SD)
DYA	1.051 (.545)	.610 (.672)
TDA	1.606 (.371)	.349 (.391)
Group	**P1 Peak Latency** Milliseconds (SD)	**P2 Peak Latency** Milliseconds (SD)
DYA	129.710 (19.508)	215.000 (14.729)
TDA	140.170 (12.104)	251.430 (37.981)

P1 and P2 amplitude and latency group measures (adaptive mean in microvolts/ mean latency in milliseconds, SD = standard deviation) for the motion/magnocellular condition

6.2.3.3.1.1 P1 amplitude/latency mean measures for the magnocellular condition. A one-way ANOVA was applied to evaluate between-group differences in the adaptive mean *P1 amplitude* in response to the magnocellular/motion condition. The experimental group (n = 7) and comparison group (n = 12) did not include any outliers, as assessed by visual inspection of boxplots. The data were normally distributed for each group, as assessed by the Shapiro-Wilk test (TDA p = .951; DYA *p* = .932), and there was homogeneity of variance, as assessed by the Levene's test of homogeneity of variance (*p* = .267). The adaptive mean *P1 amplitude* was greater in magnitude for the TDA group (Mean = 1.606, SD = .371) than for the DYA group

(Mean = 1.051, SD = .545); differences between groups were statistically significant (F (1, 17) = 6.995; p = .017, η^2 = .292).

One-way ANOVA was also used to evaluate P1 peak latency differences between groups in response to the magnocellular/motion condition. The experimental group (n = 7) and the comparison group (n = 12) included one outlier from each group. Outliers were not excluded from analysis, however, as they likely represented genuinely unusual data points and results were unaffected by their inclusion. However, while data from the DYA group were normally distributed, the data from the TDA group were not, as assessed by the Shapiro-Wilks test (DYA: p = .116; TDA: p = .001); but there was homogeneity of variance, as assessed by the Levene's test of homogeneity of variance (p = .259). The mean *P1 latency* was not statistically significantly different between groups (F (1, 17) = 2.108; p = .165, η^2 = .110).

6.2.3.3.1.2 P2 amplitude/latency magnocellular condition. One-way ANOVAs were similarly applied to the adaptive mean *P2 amplitude* and *P2 latency* for the experimental and comparison groups in response to the magnocellular/motion condition (See Figure 22). These analyses revealed no outliers. For the amplitude analysis, data were normally distributed for each group (Shapiro-Wilk test, TDA: p = .537; DYA: p = .145) and there was homogeneity of variance, as assessed by Levene's test (p = .149); however, no significant differences in adaptive mean P2 amplitude were found (F (1, 17) = 1.165; p = .295, η^2 = .064). For the P2 latency analysis, two critical assumptions were violated. Although data from the DYA group were normally distributed, data from the TDA group were not (Shapiro-Wilks test, DYA: p = .090; TDA: p = .035). Additionally, there was no homogeneity of variance, as assessed by Levene's test of homogeneity of variance (p = .001). Therefore, a Mann-Whitney U test was run to determine between-group differences using a non-parametric test. The P2 mean peak latency was

not statistically significant between groups (TDA Mean = 251, SD = 14.729, Mean Rank = 11.83; DYA Mean = 215, SD = 37.981, Mean Rank = 6.86; U = 20.000, p = .068).

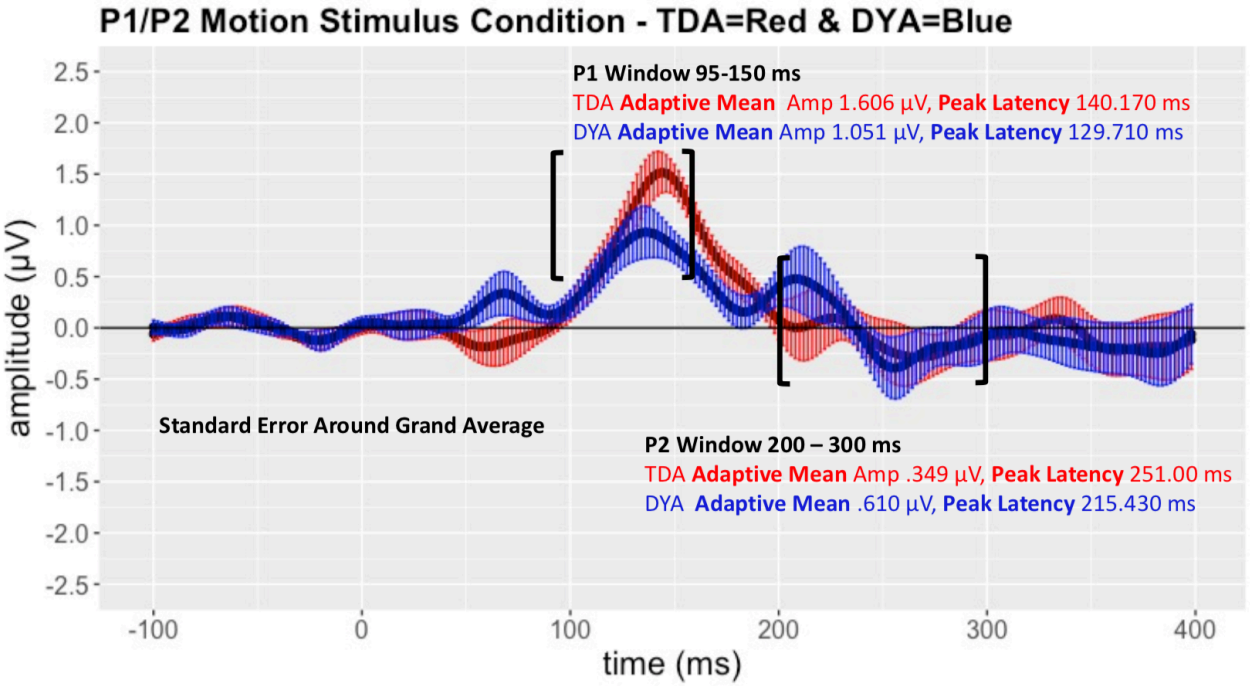

Figure 22. The P1/P2 waveform for the magnocellular/motion condition. The waveform depicts the mean and standard error for each sampled time point for both groups. The comparison group (TDA) appears in red and the experimental group (DYA) appears in blue. Electrode montages for the P1 and P2 components are identical.

6.2.3.3.2 Group analysis color/parvocellular condition.

Table 11

N1 and P2 Amplitude and Latency Group Mean Measures and Standard Deviations for the Parvocellular/Color Condition

Color/Parvocellular Condition		
Group	**N1 Adaptive Mean** Amplitude in μV (SD)	**P2 Adaptive Mean** Amplitude in μV (SD)
DYA	-3.327 (1.704)	1.204 (.772)
TDA	-2.691 (1.566)	1.748 (.668)
Group	**N1 Peak Latency** Milliseconds (SD)	**P2 Peak Latency** Milliseconds (SD)
DYA	136.000 (13.216)	254.875 (22.770)
TDA	134.667 (21.664)	240.667 (23.442)

6.2.3.3.2.1 N1 amplitude/latency mean measures for the parvocellular condition. As for the parvocellular condition analyses, one-way ANOVAs were applied to evaluate between-group differences in adaptive mean amplitude and peak latency, this time for the N1 component. For the N1 amplitude analysis, one outlier was identified in the DYA group but still included in the analysis. Data were normally distributed (Shapiro-Wilk test: TDA $p = .744$; DYA $p = .499$) and there was homogeneity of variance (Levene's test, $p = .780$). The mean N1 amplitude was not found to differ significantly between groups ($F(1, 17) = .685$; $p = .419$, $\eta^2 = .039$). Evaluation of the N1 latency data revealed four outliers from the TDA group, who were nevertheless included in the analyses. Data were normally distributed (Shapiro-Wilk test: TDA $p = .367$; DYA $p = .278$) and there was homogeneity of variance (Levene's test: $p = .476$). The mean N1 latency was

not found to be statistically significantly different between groups (F (1, 17) = .022; p = .885, η^2 = .001).

6.2.3.3.2.2 P2 amplitude/latency parvocellular condition. Also for the parvocellular condition, one-way ANOVAs were applied to evaluate P2 amplitude and latency differences between the groups. For the amplitude analysis, no outliers were apparent, the data were normally distributed (Shapiro-Wilk test: TDA p = .594; DYA p = .283), and there was homogeneity of variance (Levene's test: p = .725). The mean P2 amplitude was not significantly different between groups (F (1, 17) = 2.62; p = .124, η^2 = .134). For the latency analysis, one outlier from the DYA group was identified but maintained in the analysis. The data were normally distributed (Shapiro-Wilk test: TDA p = .894; DYA p = .454) and there was homogeneity of variance (Levene's test: p = .892). The mean P2 latency was not statistically significantly different between groups (F (1, 17) = 1.65; p = .216, η^2 = .089).

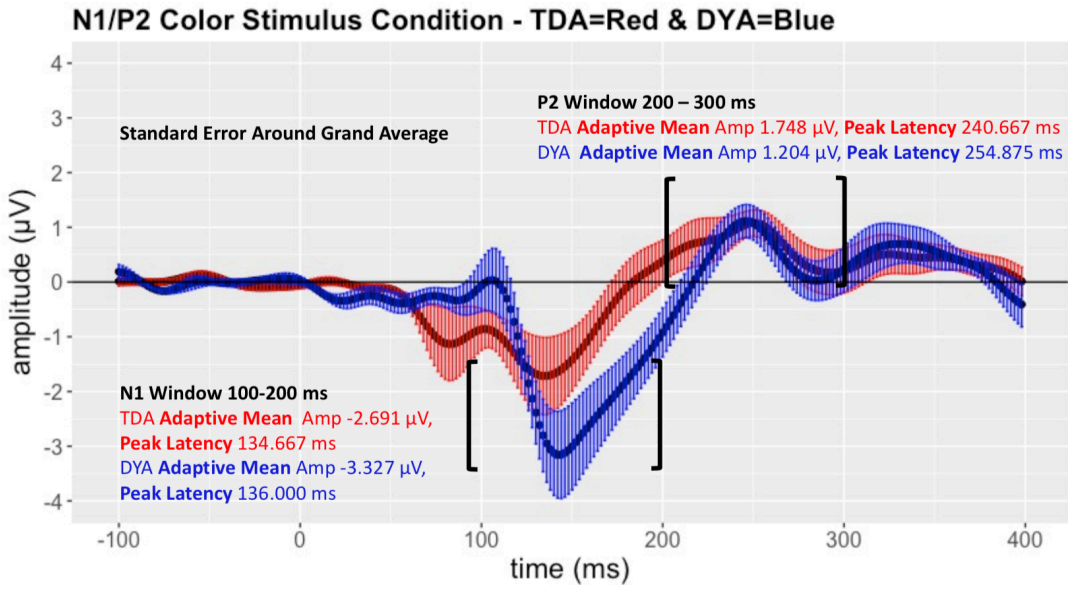

Figure 23. The N1/P2 waveform for the parvocellular/color condition. The waveform depicts the mean and standard error for each sampled time point for both groups. The comparison group (TDA) appears in red and the experimental group (DYA) appears in blue. Note that the electrode montage for the N1 and P2 components are identical.

6.2.3.3.3 Neurophysiological correlations with behavioral measures. The relationship between measures of amplitude and latency with behavioral measures was explored. Only the amplitude and latency measures for the P1 and N1 components were investigated. These two components provided the most differentiating response measures for the conditions designed to specifically bias each of the two major visual pathways. Dependent on meeting assumptions of linearity and normality, either the Pearson's product-moment correlation or the Spearman's rank-order correlation test, a non-parametric test, was conducted to evaluate the strength and direction of the association between amplitude and latency measures for both components/conditions and behavioral assessment raw scores. The behavioral measures include: Homophone Choice Task, Orthographic Choice Task, Non-Verbal Reasoning/Memory test, Comprehensive Test of Phonological Processing (CTOPP) subtests: RAN Digit, RAN Letter, Elision, Blending, Phoneme Isolation, Test of Word Reading Efficiency—Second Edition (TOWRE-2) subtests; Sight Word Efficiency (SWE), Phoneme Decoding Efficiency (PDE), Woodcock Reading Mastery Test-Revised (WRMT-R) subtests; Word Identification (ID) and Word Attack, and Wechsler Adult Intelligence Scale—Fourth Edition (WAIS-IV) Digit Span.

As is required to run either the Pearson product-moment correlation or Spearman's correlation, the data are continuous and involve paired observations. Scatterplots were generated for all the combinations of variables of interest to establish if a linear relationship existed between any two variables. See Appendix I for the scatterplots generated. The linear relationship was evaluated by visual inspection and reviewing the R-squared value to determine how well the data fit the regression line. As a very conservative cutoff, a value below .05 eliminated

combinations of variables from further investigation. Table 12 provides the R-squared values and cut off values. Variables identified for further investigation are highlighted in blue.

Table 12

R-squared Values Derived From Scatterplots to Determine Linearity

Component/Condition/ Measure	P1 MAG AMP	P1 MAG LATENCY	N1 PAR AMP	N1 PAR LATENCY
Assessment Raw Scores	**R Squared Value** The closer to 1.0, the better the fit of the regression line. Values below .05 deemed not to have a linear relationship.			
Homophone Choice	.216	.305	.018	.074
Orthographic Choice	.247	.474	.010	.001
CTOPP RAN Digit	.047	.054	.079	.010
CTOPP RAN Letter	.014	.179	.000	.003
Woodcock Word ID	.092	.437	.123	.000
Woodcock Word Attack	.269	.439	.044	.000
CTOPP Elision	.083	.000	.010	.000
CTOPP Blending	.014	.066	.002	.001
CTOPP Phoneme Isolation	.008	.050	.110	.126
TOWRE SWE	.058	.217	.000	.006
TOWRE PDE	.140	.332	.023	.000
Non-Verbal Reasoning/ Memory	.223	.001	.021	.015
WAIS-IV Digit Span	.015	0.239	.009	.206

Left column lists specific assessment. Top row provides the component (P1, N1), measure (AMPlitude/latency) and condition (MAGnocellular/PARvocellular). R-squared values highlighted in blue were further analyzed to determine if a statistically significant positive or negative relationship between an assessment measure and neurophysiological measure exists.

Across conditions and component measures, the neurophysiological measures were normally distributed, as assessed by Shapiro-Wilk's test ($p > .05$), except the P100 latency measure for the motion/magnocellular condition ($p < .001$). Additionally, not all of the behavioral measures were normally distributed. Table 12 provides an overview of those behavioral measures that were not normally distributed, based on Shapiro-Wilk's test. It also provides a visualization of which test (Pearson's or Spearman's correlation) was applied to the evaluation of the relationship between variables: for cells highlighted in blue, the variable(s) were not normally distributed; therefore, a Spearman's correlation was applied to evaluate the

strength and direction of the relationship. For cells not highlighted, the Pearson's correlation was applied. Additionally, Table 13 lists the correlation coefficient and p-value for significance at the 0.05 level (2-tailed) from the indicated test. Results listed in bold are significant and explained in further detail in the following sections.

Table 13

Relationships between behavioral measures and amplitude/latency reported

Behavioral Assessment	Shapiro Wilk	**P1 MAG AMP**	**P1 MAG Latency**	**N1 PAR AMP**	**N1 PAR Latency**
		p = .708	p = .000*	p = .409	p = .586
		Correlation Coefficient/ p-value (2-tailed)	Correlation Coefficient/ p-value (2-tailed)	Correlation Coefficient/ p-value (2-tailed)	Correlation Coefficient/ p-value (2-tailed)
Homophone Choice	p = .002*	.227 p = .351	.262 p = .279		.355 p = .135
Orthographic Choice	p = .022*	.338 p = .157	**.498 p = .030***		
CTOPP RAN Digit	p = .015*	.199 p = .415	-.185 p = .448	-.190 p =.435	
CTOPP RAN Letter	p = .029*		-.272 p = .260		
Woodcock Word ID	p = .029*	.215 p = .377	**.500 p = .029***	.402 p = .088	
Woodcock Word Attack	p = .011*	.438 p = .061	.299 p = .214		
CTOPP Elision	p = .044*	.155 p = .528			
CTOPP Blending	p = .052		.421 p = .073		
CTOPP Phoneme Isolation	p = .327		-.181 p = .458	.332 p = .165	
TOWRE SWE	p = .181	.241 p = .320	**.510 p = .026***		
TOWRE PDE	p = .019*	.261 p = .280	.427 p = .068		
Non-Verbal Reasoning/ Memory	p = .131	**-.473 p = .041***			
WAIS-IV Digit Span	p = .033*		.403 p = .087		.343 p = .150

The left column lists investigated behavioral measures. The shaded gray column and upper row provide the p-values for the Shapiro-Wilk's test of normality. Assessment and component measures with a p-value < .05 were determined not to be normally distributed, and the Spearman's correlation was used to investigate relationships between variables (blue shading). For normally distributed variables (no shading), Pearson's correlations were used to investigate relationships between variables. Empty cells indicate combinations of variables that were determined to be non-linear and therefore not investigated further (see Table 13 above). Significant correlation findings are indicated in bold.

6.2.3.3.3.1 Pearson's correlation analysis—Significant findings. Pearson's product-moment correlations were conducted to assess the relationships between neurophysiological measures and selected behavioral measures. Preliminary analysis showed the relationship between multiple variables to be linear with both variables normally distributed (Shapiro-Wilk's test $p > .05$) (see Table 13). Of the relationships tested, the negative correlation between the P1 amplitude for the magnocellular/motion condition and the Non-Verbal Reasoning and Memory test was statistically significant ($r(17) = -.473$, $p = .041$), with the P1 amplitude accounting for 22% of the variation in raw scores obtained for the Non-Verbal Reasoning and Memory test. Cohen (1988) provided basic guidelines for interpreting the strength of a correlation coefficient, and the findings in this range suggested a moderate association between these variables.

6.2.3.3.3.2 Spearman's correlation analysis—Significant findings. For those variables indicated in Table 13 as monotonic, as assessed by scatterplots, the Spearman's rank-order correlation was applied to investigate the relationship between the P1 latency for the magnocellular/motion condition and three assessments: the Orthographic Choice Task, the WRMT-R Word ID subtest, and the TOWRE-2 SWE subtest. There was a moderate positive correlation between the P1 latency for the magnocellular/motion condition and the Orthographic Choice Task ($r_s(17) = .498$, $p = .030$). A strong positive correlation was observed for the P1 latency for the magnocellular/motion condition and both the WRMT-R Word ID subtest ($r_s(17) = .500$, $p = .029$) and the TOWRE-2 SWE subtest ($r_s(17) = .510$, $p = .026$).

6.2.3.4 Summary of study findings. In brief, the main experimental findings are as follows:

1. Waveforms comparing both groups/conditions revealed that the P100 component best represents a brain response to the motion/magnocellular stimulus condition and the

N100 component best represents a brain response to the color/parvocellular stimulus condition. Each stimulus condition produced a response during the 200 to 300 ms time window. However, the response to the color/parvocellular condition produced a definitive P200, whereas the motion/magnocellular condition produced an ambiguous early positivity and mid-window negativity.

2. Group comparisons revealed component amplitude and latency differences (see Table 14) for both conditions (motion/magnocellular and color/parvocellular); however, those differences were only statistically significant for the P1 amplitude for the motion/magnocellular condition.
3. Correlations between ERP responses across conditions and behavioral measures were generally not significant, apart from four significant correlations:
   a negative correlation between the P1 amplitude for the magnocellular/motion condition and the Non-Verbal Reasoning and Memory test ($r(17) = -.473$, $p = .041$); a moderate positive correlation between the P1 latency for the magnocellular/motion condition and the Orthographic Choice Task ($r_s(17) = .498$, $p = .030$); and strong positive correlations with the P1 latency for the magnocellular/motion condition and both the WRMT-R Word ID subtest ($r_s(17) = .500$, $p = .029$) and the TOWRE-2 SWE subtest ($r_s(17) = .510$, $p = .026$).

Table 14

Summary of Mean Amplitude/Latency Measures for All Components/Conditions, by Group

		Group	Mean	SD	One-way ANOVA	η^2
Magnocellular Condition	P1 adaptive mean amplitude	DYA	1.051 μV	.545	F (1, 17) = 6.995 *p* = .017*	.292
		TDA	1.606 μV	.371		
	P1 peak latency	DYA	129.710 ms	19.508	F (1, 17) = 2.108 *p* = .165	.110
		TDA	140.170 ms	12.104		
	P2 adaptive mean amplitude	DYA	.610 μV	.672	F (1, 17) = 1.165 *p* = .295	.064
		TDA	.349 μV	.391		
	P2 peak latency	DYA	215.000 ms	14.729	Mann Whitney U Test U = 20, *p* = .068	
		TDA	251.430 ms	37.981		
Parvocellular Condition	N1 adaptive mean amplitude	DYA	-3.327 μV	1.704	F (1, 17) = .685 *p* = .419	.039
		TDA	-2.691 μV	1.566		
	N1 peak latency	DYA	136.000 ms	13.216	F (1, 17) = .022 *p* = .885	.001
		TDA	134.667 ms	21.664		
	P2 adaptive mean amplitude	DYA	1.204 μV	.772	F (1, 17) = 2.62 *p* = .124	.134
		TDA	1.748 μV	.668		
	P2 peak latency	DYA	254.875 ms	22.770	F (1, 17) = 1.65 *p* = .216	.089
		TDA	240.667 ms	23.442		

TDA = typically developing/comparison group; DYA = individuals with dyslexia/experimental group; μV = Microvolts; ms = milliseconds. Significant between-group differences indicated with an asterisk.

7. DISCUSSION

Few studies have used high-density electroencephalography to explore early visual processing differences between adults with and without dyslexia to stimuli developed to bias each of the major visual pathways. This study succeeded in generating distinct responses to each of the experimental conditions, as evidenced by the group averaged waveforms. The stimuli were based on previous studies (Armstrong et al., 2002; Coch et al., 2005) that explored the differing developmental trajectories of the magnocellular and parvocellular pathways and were modified for use with an LCD monitor and programmed using software that will facilitate replication.

The magnocellular/motion condition produced a P1 component, whereas the parvocellular/color condition was associated with the N1 component. Both conditions also produced voltage deflections during the 200 to 300 ms time window: a distinct positivity for the parvocellular/color condition (P2 component) and a more ambiguous response for the magnocellular/motion condition.

While the ERP waveforms suggested that grand averaged group responses to each condition differed, the differences in adaptive mean amplitude and peak latency across both conditions were only statistically significant between groups for the P1amplitude in response to the magnocellular/motion condition. The P1 amplitude was greater for the comparison group than the experimental group in response to the motion/magnocellular condition. No other statistically significant differences in neurophysiological measures were found between groups.

The first aim of this study was to investigate differences in early visual responses as measured by event-related potentials to stimuli developed to separately and preferentially bias the magnocellular or parvocellular visual pathways between groups of adults with and without

dyslexia. The significant between-group difference in P1 amplitudes supports the Magnocellular Theory of Dyslexia, though the component latency measure in response to input that biases the magnocellular pathway (high temporal, low contrast, and low spatial resolution) was not significant. As predicted, there were no differences between the group of adults with and without dyslexia in response to the color stimulus (biasing the parvocellular pathway) in the ERP amplitude or latency measures.

Although this pattern of responses broadly supports the Magnocellular Theory of Dyslexia, many have suggested that differences in motion sensitivity within the visual system provide evidence of a disruption to the transient magnocellular/dorsal pathway contributing to reading disability (Mascheretti et al., 2017; Stein & Walsh, 1997; Vidyasagar & Pammer, 2010). Others are committed to the interpretation of such findings as a consequence of reading experience (Goswami, 2015; Olulade et al., 2013). Many studies investigating visual motion differences in individuals with and without dyslexia have used random dot kinematogram (RDK) stimuli (e.g., Cornelissen et al., 1995; Downie, Jakobson, Frisk, & Ushycky, 2003; Gori, Seitz, Ronconi, Franceschini, & Facoetti, 2016; Hill & Raymond, 2002; Samar & Parasnis, 2007). Each has demonstrated that individuals with dyslexia benefit from a greater number of coherently moving dots to detect motion. More recently, an fMRI study using stimuli that biased the magnocellular and parvocellular pathways (e.g., magnocellular/ monochrome, low-spatial frequency, high-temporal frequency, high-luminance contrast and parvocellular/high-color contrast, high-spatial frequency, low-temporal frequency, low-luminance contrast) demonstrated that such stimuli can elicit differential BOLD responses from the two subdivisions of the LGN, defining the boundaries of the segregated pathways (Denison, Vu, Yacoub, Feinberg, & Silver, 2014). Further, Giraldo-Chica et al. (2015) used high-resolution proton-density weighted MRI

scans to establish that the overall volume of the left LGN was significantly smaller in adults with than without dyslexia (22-26 years of age), whereas no differences in volume were observed in the right LGN. This finding suggested that differences in early-stage visual processing may contribute to reading disability. In comparing their observed volume differences in the right and left LGN with behavioral assessments, only the left LGN measure was found to be positively correlated with spelling (p = .045) (Giraldo-Chica et al., 2015). This current study builds on such work by demonstrating that stimuli-biasing receptive preferences at the LGN can index early-stage visual processing via the P1 and N1 ERP components, and that differences between groups are exclusively observable in response to stimuli that bias the magnocellular pathway.

Magnocellular neurons are found throughout the brain and are specialized for temporal processing. Ninety percent of the projections to the dorsal stream involve magnocellular neurons, forming an attentional stream for the allocation of visual attention and visually directed motion (Stein, 2018). This pathway is thought to contribute to reading by facilitating the rapid recognition of letters within the ventral stream, directing cognitive resources to the sequence of objects/features, shifting attention, and directing eye movements (Vidyasagar & Pammer, 2010). The magnocellular theory has received criticism because it does not account for the phonological deficits so frequently observed in dyslexia and considered the core causal issue (Hornickel & Kraus, 2013; see Gabrieli, 2009, for review). However, as noted previously by Goswami (2015) and Olulade et al. (2013), differences in motion processing are not causal but can be attributed to reading experience. Boets et al. (2011) demonstrated that magnocellular/dorsal stream functioning can improve over time, measuring coherent motion detection in children before and after formal instruction was introduced. Children later diagnosed with dyslexia showed significantly poorer coherent motion sensitivity before reading instruction and threshold

differences provided an index of dorsal stream functioning that was predictive of later reading and spelling problems. Findings from an fMRI study (Olulade et al., 2013) revealed abnormal visual motion processing in individuals with dyslexia in response to stimuli that preferentially bias the magnocellular/dorsal pathway. This finding was interpreted as the consequence of an altered reading experience rather than a contributing factor to reading disability (Olulade et al., 2013).

Our understanding of a phonological deficit in individuals with dyslexia suffers from a similar quandary. While such deficits are strongly associated with reading disability, the underlying mechanisms remain to be clarified. Ehri (1989) suggested that to some degree, phonological sensitivity is a consequence of reading instruction. Further, Vidyasagar and Pammer (2010) proposed that normal input from the visual system to those brain regions supporting the development of phonological awareness may be essential to forming grapheme-phoneme correspondences. Boets et al. (2011) speculated that the type of bidirectional relationship between phonological awareness skills and reading acquisition might point to a similar relationship between various dorsal stream processes tapped during reading acquisition. Phonological representations develop in children at the syllable level and over time become further segmented with linguistic input (Metsala & Walley, 1998). This input must be explicit because although spoken words are delivered in separable syllables, written words are represented as graphemes that must be translated into phonemes that are not separated in speech (Liberman, Shankweiler, & Studdert-Kennedy, 1967). Castro-Caldas et al. (1998) found that for illiterate adults, phoneme-level representations did not develop automatically; rather, literacy instruction was shown to alter phonological development. Therefore, it seems within reason to assume that the magnocellular/dorsal visual stream might also develop with experience. It is

possible that, for individuals with dyslexia, standard reading instruction is not sufficient to develop the underlying mechanisms, or that disruptions in information processing interfere with required practice. If the inputs to low- or high-level processes are processed differently during reading acquisition, within one system (auditory, visual or linguistic) or when systems interact, then it is conceivable that different combinations of disruptions in a forming network would result, thus revealing the continuum of reading ability observed. The etiology of reading disability will only be understood if the neural contributors that underpin sensory and cognitive processes, and how they form networks during skill acquisition, can be identified.

One interesting observation, while not statistically significant, was that the P1 peak latency in response to the magnocellular/motion condition was shorter for individuals with than without dyslexia. This observation runs contrary to psychophysical studies of temporal processing in developmental dyslexia, which suggested a sluggish magnocellular/dorsal pathway (see Edwards et al., 2004). Most psychophysical studies involving measures of magnocellular/ dorsal functioning have reported higher thresholds for individuals with dyslexia as compared to typical readers. Studies measuring visual evoked potentials (Livingstone et al., 1991; Schulte-Körne, Bartling, Deimel, & Remschmidt, 2004) typically reported delayed and/or attenuated amplitude responses in individuals with dyslexia. A review of psychophysical studies (Grinter, Maybery, & Badcock, 2010), comparing thresholds primarily between children with and without dyslexia, suggested that mixed results were not easily interpretable and further neuro-physiological research and a better definition of the disorder are needed. An intriguing consideration is that in this study, participants were not required to respond to the presentation of the stimuli, and so the ERP responses reflected change detection solely by the visual system for each condition.

To speculate, perhaps it is not the speed of the signal but the number of contributing neurons to, for example, attentional mechanisms, which resulted in apparent "sluggishness" of the magnocellular/dorsal pathways. Left LGN volume differences were reported between adults with and without dyslexia, as reported by Giraldo-Chica et al. (2015) as well as post-mortem studies (Galaburda & Livingstone, 1993)—findings that support such speculation. It should be pointed out, however, that all participants in this study accurately perceived the direction of motion tested at two contrast levels and at different spatial and temporal frequencies (the direction discrimination task). Across all conditions, we found no group reaction time differences, though individual differences in RT were observed.

This study classified participants based on prior diagnosis of a specific learning disability with impairment in reading (dyslexia). We must assume a great deal of heterogeneity in terms of learning profiles within the group of individuals with dyslexia (experimental group). Additionally, reading ability is thought to exist along a continuum (Shaywitz & Shaywitz, 2005), and so it is possible that within the group of individuals without dyslexia, there could also be heterogeneity in skill sets contributing to reading. While four of the behavioral measures differentiated groups (Orthographic Choice Task, Test of Word Reading Efficiency—Second Edition [TOWRE-2], Sight Word Efficiency and Phonemic Decoding Efficiency, and Woodcock Reading Mastery Test—Revised [WRMT-R] Word Identification), the overlap in other scores suggested that, at least for this sample of adults including highly remediated individuals with dyslexia, assessments such as rapid automatized naming—which are highly predictive earlier in reading development—do not measure the same process or skill at every age.

The difficulties in defining developmental dyslexia in part stem from a failure to identify the underlying etiology. The consensus in the reading research community is that developmental

dyslexia is identified solely as a linguistically-based neurodevelopmental disorder. This is reinforced by the idea that because reading is a linguistic skill, reading disability must then be related to some aspects of linguistic processing (Vellutino, 1979b). However, reading is a complex behavior requiring that multiple brain systems work in a coordinated fashion. This suggests there could be multiple reasons for encountering difficulty learning to read and reading fluently, a view that is gaining more attention (e.g., Pennington et al., 2012). Carroll, Solity, and Shapiro (2016) suggested a multiple-deficits view, as opposed to a single deficit, as a more likely explanation of dyslexia and other developmental disorders. Such a view aligns with the heterogeneous nature of most neurodevelopmental disorders and with findings from genetic research.

To begin to explore the potential underlying mechanisms that might contribute to visual processing differences between individuals with and without dyslexia, the second research question for the present study relates to the relationships between ERP indices of early visual processing for both the parvocellular/color condition and magnocellular/motion condition and measures obtained from multiple behavioral assessments. No correlations were observed between the neurophysiological response measures to the parvocellular/color condition and any of the behavioral measures. However, a negative correlation between the magnocellular P1 amplitude and the Non-Verbal Reasoning and Memory task was observed, as well as a positive correlation among the P1 latency and several behavioral measures (Orthographic Choice Task, Test of Word Reading Efficiency—Second Edition, Sight Word Efficiency subtest, and the Woodcock Reading Mastery Test—Revised, Word Identification subtest). These behavioral tasks are not thought to measure phonetic decoding skills but rather word decoding (the ability to pronounce printed words accurately and fluently).

Developing and drawing on knowledge and awareness of graphemes and blending them in the serial decoding of words is time-consuming and error-prone, and consumes cognitive resources (Ouellette & van Daal, 2017). In the transition to effortless word recognition, orthographic learning and relevant mental representations are crucial (Ouellette & van Daal, 2017). Ehri (2014) made the connection between orthographic learning and skilled reading, proposing that experience with and exposure to printed text permits development of the ability to utilize memory representations for an increasing number of letter sequences and ultimately entire words. When learning to read, multiple skills become integrated, as depicted by Scarborough's (2001) Rope Model of Reading. One thread of the rope involves word recognition which becomes increasingly automatic over time. The model also holds that phonological awareness, decoding, and sight recognition are crucial for word recognition. Ehri (2014) suggested that the ability to read words accurately and automatically from memory is supported by orthographic mapping. If we take this to be a critical factor in skilled reading, it is possible to make a connection between the three behavioral measures this study found to be correlated with the P1 latency measure in response to the magnocellular condition.

While reading words accurately and automatically from memory may be supported by a cognitive skill such as orthographic mapping, fluent reading also requires the coordination of eye movements, accommodation, and fixation. The coordination of such processes allows for the rapid spatiotemporal processing of visual input (Laycock & Crewther, 2008). It has been suggested that learning to read recruits multiple dorsal stream processes such as selective visual attention and eye movements during the acquisition of orthographic representations (Boden & Giaschi, 2007; Chase & Stein, 2003). Grainger et al. (2016) proposed that skilled readers develop mechanisms for computing letter identity alongside location relative to fixation and position

within a word. With practice, letter-level representations can bypass phonology and provide direct access to associated meaning. A neural network approach to visual object recognition proposes that input from the fast dorsal stream may modulate early visual processing (Breitmeyer, 2014). Vidyasagar and Pammer (2010) proposed that attentional mechanisms controlled by the dorsal visual stream help in serial scanning of letters and any deficits in this process will cause a cascade of effects, including impairments in visual processing of graphemes, their translation into phonemes, and the development of phonemic awareness. Processing disruptions in the magnocellular pathway could contribute to reading difficulties by interfering with orthographic processing via attentional mechanisms (e.g., Lovegrove, Martin, Blackwood, & Badcock, 1980) or through a more general temporal processing disruption that alters both visual and auditory processes (e.g., Tallal, Miller, & Fitch, 1993). Multiple studies have demonstrated that motion-perception thresholds correlate with measures of orthographic and not phonological processing skills (see Talcott et al., 2000). Against this background, the observed relationships between the Orthographic Choice Task, the Test of Word Reading Efficiency—Second Edition, Sight Word Efficiency subtest, and the Woodcock Reading Mastery Test—Revised, Word Identification subtest support further investigation of how the magnocellular/dorsal pathway may support word decoding.

7.1 Study Limitations

The current study has several limitations and delimitations. In addition to a small sample size, the groups were unbalanced (DYA = 7, TDA = 12). The experimental group included individuals who were college-bound, college students, and graduates now working as young professionals. Their reading experience and exposure may differ from the comparison group over their lifetimes. However, based on participant reports of daily reading (see questionnaire

responses in Appendix E), it appears that the experimental group read as much as the comparison group, resulting in a sufficient amount of exposure to automatize the reading network. This can reasonably be expected to result in differences that relate more specifically to residual disruptions within the brain rather than social issues.

Individuals with co-occurring conditions, excluding major psychiatric illness, were included in the study; for instance, one individual in the comparison group reported having ADHD and six of the seven individuals in the experimental group reported having ADHD. Although the decision to include these individuals constrains generalizability, it does reflect more accurately the heterogeneity and comorbidity that are typical of people diagnosed with a reading disorder.

In light of these factors, it is only with caution that one would generalize these findings to the greater population of individuals with reading disability/dyslexia.

7.2 Future Directions

While this study adds to the body of literature that explores how the magnocellular/dorsal stream differs between adults with and without dyslexia, this line of research can be extended in a number of directions. First, collecting data from a much larger sample may help to reveal patterns within the data that will allow for sub-grouping by factors such as co-occurring conditions (ADHD) or early or late ERP responses within a component time window. Isolating such factors and evaluating the neurophysiological differences and potential corresponding behavioral measures would provide insights that could lead to better definitions of reading disorders and help to clarify why findings in this area of research have produced such mixed results.

The adults who participated in this study represent the product of years of reading instruction, reading experience, and exposure to print more generally. A longitudinal study of pre-reading children at high risk and low risk would provide the opportunity to observe changes in both phonological and visual processing development as children become skilled readers. Corroborating neurophysiological development, as measured by ERPs elicited in both visual and auditory domains, with reading development measures between children at high and low risk would help to elucidate the interactions between nature and nurture while acquiring a complex behavior like reading.

7.3 Conclusions

This study demonstrates the feasibility of using high-density EEG to measure differences in early-stage visual processing in two major pathways: magnocellular and parvocellular. Together, the results of this study reinforce the view that a phonological deficit may not in all situations be the exclusive disruption to the reading network that is associated with dyslexia. With further research, the P1 component in response to a magnocellular-evoking stimulus could serve as a biomarker that either independently or in concert with specific behavioral screenings could help to identify individuals at risk for reading difficulty *before* they experience reading failure, which should be our collective goal.

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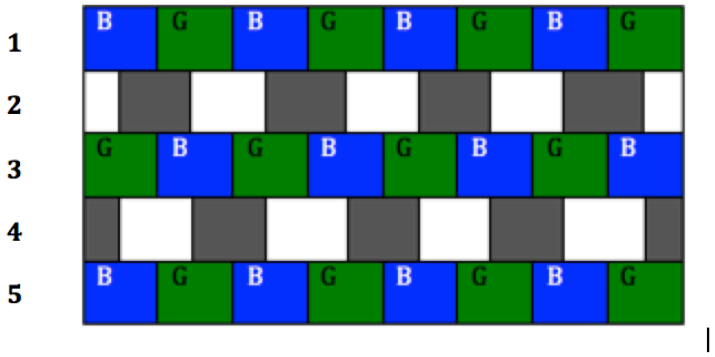
Appendix A

Further Explanation of the Motion Null Technique Used for
Individual Equiluminant Color Parameter Calibration

Isoluminance will depend on multiple parameters such as size, location, and pattern (Lu & Dosher, 2014; Cavanagh, Anstis, MacLeod, 1987). Hence, the image presented for motion null task replicates the parameters of the EEG color condition (6 cpd vertically presented 2° smooth Gaussian edge). The sandwich display used is an amplifier motion display includes test frames and amplifier frames, which alternate (see figure X below.) The odd frames show blue bars that will consistently present at 33% contrast and the green bars will initially present at 20%. The achromatic gratings will present at high contrast. The odd and even frames will be presented sequentially for 100 ms each, 5 times a second, resulting in a flicker rate of 5 Hz.



An illustration of the sandwich display for isoluminant color calibration.
Odd frames (1, 3, 5) = blue-green (red-green) sine wave gratings with 180-degree phase shift between. Even frames (2, 4) = "amplifier frames" are luminance sine-wave gratings also with a 180-degree phase shift. Phase shift between successive frames is 90 degrees. Each frame, presented as a circular figure center screen and consisting of either chromatic or achromatic amplifier gratings, is presented sequentially for 100ms, 5 times a second (Lu & Dosher, 2014).

The even frames are amplifier frames intended to exaggerate the luminance differences. This effect is achieved in part by displacing the luminance gratings by one-quarter cycle (90°; see above) from the chromatic grating. Differences in the luminance values of the colors presented

create the illusion of motion (Cavanagh, 1991). The direction of the perceived motion depends on the relative brightness of the colors represented in the chromatic gratings. In a blue/green comparison, when the blue bars appear brighter than the green bars, the motion appears to traverse right; when the green bars appear brighter than the blue, the motion appears to traverse left (Anstis & Cavanagh, 1983). However, neither the chromatic gratings nor the amplifier frames independently contribute to the perception of motion; rather, it is the combined effect of the sequentially presented odd and even frames that create the percept of motion (Anstis & Cavanagh, 1983; Lu & Dosher, 2014; Lu & Sperling, 2001). When both colors are of equal luminance, the percept is generally a balance of leftward and rightward motion or flashing. The sandwich display used to present the motion null task is generated using Psykinematix software (KyberVision, Sendai, Japan, psykinematix.com). For further information on calibration and measurement procedures, see Lu and Sperling (2001).

Appendix B

IRB Approval Letter

TEACHERS COLLEGE
COLUMBIA UNIVERSITY

Teachers College IRB *Modification Approval Notification*

To: Lisa Levinson
From: Curt Naser, TC IRB Administrator
Subject: IRB Modification Approval: 14-264 Protocol
Date: 04/03/2018

Please be informed that as of the date of this letter, the Institutional Review Board for the Protection of Human Subjects at Teachers College, Columbia University has approved a *modification* to your study, entitled "*Neural correlates of early-stage visual processing differences in developmental dyslexia*" on 04/03/2018. This modification acknowledges the change of the protocol title, which was originally submitted in 2016 but which was never updated on the protocol record.

The approval remains effective until **05/25/2018**.

The IRB Committee must be contacted if there are any changes to the protocol during this period. **Please note:** If you are planning to continue your study, a Continuing Review report must be submitted to either close the protocol or request permission to continue for another year. Please submit your report by **04/27/2018** so that the IRB has time to review and approve your report if you wish to continue your study. The IRB number assigned to your protocol is **14-264**. Feel free to contact the IRB Office (212-678-4105 or IRB@tc.edu) if you have any questions.

Best wishes for your research work.

Appendix C

What Participants Can Expect

TEACHERS COLLEGE COLUMBIA UNIVERSITY

Thank you for your interest in this research project.

Learning to read involves direct instruction and a lot of practice so that different brain processes and regions can work in coordination efficiently. For example, the language system has to learn to work with the visual system. This study explores early-stage visual processing in individuals with and without dyslexia. Dyslexia is described as an inability to achieve reading competency despite adequate motivation, cognition, sensory levels and appropriate educational input. Using electroencephalography (EEG), a non-invasive technique for recording electrical activity generated by the brain, stimuli designed to target the visual system will be used to explore early visual processing in individuals with dyslexia and typical readers.

Prior To Your Visit - Hair & Health

It is best if you wash your hair the night before. On the day of your visit, your hair needs to be loose to allow us to place EEG sensors on the scalp. If your hair is braided, woven, or in dreadlocks, this may prevent easy access to the scalp. Please discuss this with your lab contact prior to your visit. Please do not use hair spray, oils, cream rinse or gel after your hair has been washed. Please do not schedule any dental work within 24 hours of the lab visit. Please contact us to reschedule if you are running a fever.

What To Expect

Time Commitment: All of the activities in this study will about 2 1/2 hours to complete, including some break time.

Orientation: We will begin by introducing you to the research team and the equipment in the lab. The data we are collecting requires a controlled environment that is shielded from electrical interference and lined with sound-proof padding. The environment may feel or appear unusual to you, but most people adjust to the unusual environment during the orientation. The research team will describe the planned activities and answer any and all questions. With your full consent we will proceed.

WWW.TC.EDU/NEUROCOG 917-882-1934 LML2155@TC.COLUMBIA.EDU

The Experiment: You will be given several basic vision screenings. Then, after a brief handedness inventory, equipment calibration, and computerized task we will complete seven brief assessments that index reading skills. When finished we will take a break.

Questionnaire: You will then be asked to complete a brief survey.

The EEG experiment will follow. You will be reminded of the procedure and we will answer any additional questions. A net of EEG sensors soaked in a saline solution will be placed on your head. Once the EEG net is comfortably fitted, you will complete a computerized task. The task will be presented in two 10 minute blocks with breaks in between.

Approximate Timeline

Activity	Length
Orientation & Introductions	10 minutes
Consent, Vision Screening, Questionnaire, Equipment Calibration Exercise, Direction Discrimination Task	40 minutes
Assessments	50 minutes
Break and Snack	10 minutes
Experiment setup and ERP data collection	40 minutes
TOTAL APPROXIMATE TIME	2 1/2 hours

Note: The order of activities may change.

WWW.TC.EDU/NEUROCOG 917-882-1934 LML2155@TC.COLUMBIA.EDU

Directions to the Lab

Teachers College is located on the Upper West Side of Manhattan on 120th Street between Broadway and Amsterdam Avenue. (525 W.120th Street, NY, NY 10023). The College is easily accessible by subway. There is limited parking in the area if you travel by car — see the following instructions and contact us if you have any questions.

SUBWAY: The subway station serving Teachers College is the 116th Street stop of the local "1" train (red line). Be sure that you are on (or transfer to) the local line at the 96th Street Station — the express lines (No. 2 or No. 3 trains) do not serve Columbia University. For more information: Visit the MTA website www.mta.info.

Once you get out of the the subway at 116th and Broadway, walk north to 120th and Broadway, cross to the far (north) side of the sidewalk and turn right. As a landmark, the building on that corner is called Horace Mann. Midway down the street you will see a set of brownstone stairs, that is the main entrance to TC, the Zankel Building (pictured on the bottom right).

Once inside, you will need to show ID and tell the security guard that you are going to 1155 Thorndike Hall to participate in a research study. A member of the research team will pick you up from the front security desk. If you do not see someone there to meet you, have security call the lab at extension 8169 or call from your cell phone (212)-678-8169.

DRIVING: The Henry Hudson Parkway (West Side Highway) in New York City runs parallel to the Hudson River and offers convenient access to Teachers College. The highway can be reached from most of the main routes entering New York City. The nearest major highway link to it is Interstate Highway 95 (I-95).

Whether driving north or south on the Henry Hudson Parkway/West Side Highway, exit at 95th Street. At the first traffic light, turn north (left) onto Riverside Drive; at 120th Street turn east (right) and go two blocks east to the College. The main entrance is located midway between Broadway and Amsterdam Avenue, on the north side of West 120th Street.

Parking: While West 120th Street offers metered parking on both sides of the street, parking on New York City streets in the Columbia University area is limited. Nearby off-street parking facilities include:

- GMC Parking, West 122nd Street (between Amsterdam and Broadway) (212) 961-1075
- Riverside Church Garage, 120th Street
  (between Riverside Drive and Claremont Avenue), (212) 870-6736

Privacy: We take your privacy very seriously. You will be assigned a code number and the data we collect will only be linked by that number. We store paper information in locked file cabinets and electronic data on secured servers and computers that can only be accessed by the research team. Contact information will be deleted upon completion of the experiment.

Thank you for participating: Participants will receive $50 cash for participating. We recognize the valuable contribution of time participants are making to further our understanding of the potential underlying neural mechanisms that may contribute to dyslexia. The gift card is a small token of appreciation.

Contact the Research Team

The principal investigators for this study are Dr. Karen Froud & Ms. Lisa Levinson. All members of the research team are trained EEG researchers with extensive experience. If you have any questions related to the study or your eligibility, you can contact the research team using the email address and phone number on the bottom of the page.

The Teachers College Institutional Review Board (IRB) has approved this study.
Protocol Number: 14-264
Approved Until: 5/25/18

TEACHERS COLLEGE COLUMBIA UNIVERSITY

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Appendix D

Informed Consent

Subject #_____

TEACHERS COLLEGE

COLUMBIA UNIVERSITY

Department of Biobehavioral Sciences

INFORMED CONSENT

Neural Correlates of Early-Stage Visual Processing Differences in Developmental Dyslexia

DESCRIPTION OF THE RESEARCH: You are invited to participate in a research study that will explore the neural correlates of early-stage visual processing differences in developmental dyslexia. You have been selected EITHER because you received a diagnosis of dyslexia from a professional (e.g., school psychologist, neuropsychologist) at one point during your educational experience, OR because you are a fluent reader with no history of neurological problems and can be a comparison participant in the study.

WHERE THE RESEARCH TAKES PLACE: All research and data analysis takes place in the Neurocognition of Language Lab, Thorndike 1155, Teachers College, Columbia University.

WHAT YOU WILL BE ASKED TO DO: First, you will be introduced to the researchers, given a tour of the lab and provided an opportunity to ask any questions you may have about the experiment and the lab. All members of the research team are experienced with EEG and happy to answer any and all questions at any time.

We will then begin the experimental activities, including following:

1. We will determine your visual acuity (naming letters on an eye chart), color vision (identifying numbers embedded in color patterns), and contrast sensitivity (reading letters on an special eye chart). The total time should be no more than 10 minutes.

Should the visual acuity or color vision screening test indicate that you have a visual deficiency, either with your visual acuity (despite corrective glasses or contact lenses) and/or with your color perception, we will let you know and you will be offered the choice to continue or not continue with the experiment. These screening tools are not diagnostic, but they provide the researchers with an indication that you may not be sensitive to our visual stimuli.

2. You will then complete a brief questionnaire and separately respond to questions regarding hand-use preferences. This should take approximately 10 minutes.

3. One of the experimental conditions involves obtaining individual perceptual values of brightness. We measure these parameters using the Motion Null Technique. Participants are seated at the computer and asked to judge the direction of vertical bars presented on the monitor screen by indicating with your left and right hand the direction of motion to the principal investigator. There will be an opportunity to practice and then each step of the technique will be completed twice, or four times in total. This is a very brief but important part of the experiment. A brief Direction Discrimination Task is also presented on the monitor screen and participants

are asked to judge the direction of the vertical bars by pressing arrow keys on a standard keyboard. This takes about 15 minutes.

To follow are the assessments and EEG recording. The order of the sequence changes but a break is included between activities.

Seven brief assessments will then be administered. You will be asked to read words, manipulate words in different ways, repeat back numbers forward and backward and completed some visual processing tasks. This part of the experiment will last a little less than an hour.

4. Electroencephalography (EEG) recording
EEG is a very safe and non-invasive way for us to measure brain activity while people are carrying out different tasks. In this study, we want to learn more about how your brain processes visual stimuli. So, we will use our EEG recording equipment to do that.

Our equipment for measuring brain activity uses a set of 128 sensors, embedded in small sponges and connected to each other by fine elastic to make a kind of hairnet. The sensor nets are certified to the highest safety standards, and are used worldwide in research and clinical environments, including with children and even newborns.

We will first show you the EEG recording equipment and explain what it does. When you are ready, we will carry out the following steps:

- measuring your head and selecting the right-sized sensor net.
- soaking the sensor net in a warm-water solution that contains potassium chloride and a little baby shampoo. This solution makes the net more comfortable to wear and also improves its ability to pick up signals generated by the brain.
- marking the center of your head (for electrode positioning). We use a safe, soft, washable marker to do this.
- positioning the net on your head and checking the position of each electrode.
- connecting the net to the amplifier and computers, all of which are protected by electrical isolation transformers (special devices that separate the equipment from the mains power source) to a very high rating.
- measuring how well each electrode is working. This involves us passing an extremely tiny electrical current through each electrode – well below what you can feel – about the same strength as the electrical field generated by a refrigerator magnet. The computers read these tiny signals back and give us a reading that shows which electrodes have good connections. We will work on the electrode positioning until each electrode has a good reading.

When all these steps are complete, the experiment can begin. The instructions will be verbally explained while applying the net. However, at the beginning of the experiment, they will appear on the monitor as well. There are two 10-minute segments of the experiment. During a brief break in between, we will confirm that the electrodes are still making good connections. This part of the experiment takes about 40 minutes. When the second segment is done, we will remove the electrode net and the experiment will be all done.

This research will be conducted by Lisa Levinson, MS and Professor Karen Froud, PhD in the Biobehavioral Sciences Department at Teachers College, Columbia University.

RISKS AND BENEFITS: There is always a risk when you agree to participate in research. For this study, one risk is that you might get bored and/or tired. We try to reduce this risk by making sure that you get a break during the experiment and by explaining what we are doing as we go along. During the experiment, you are asked to be still and passively watch the center of the monitor. To make this a more interesting experience, random "emoticon" figures will appear on the screen, and you will need to push a button whenever this happens.

The EEG portion of the study carries a small risk of electric shock, which we keep as low as possible by using very stringent safety procedures. We use high-rated circuit breakers and isolation transformers (special devices that separate the equipment from the mains power source) to make sure that anyone using our equipment is very safe. Because of our safety precautions, the risk of getting an electric shock is smaller than the risk involved in using a toaster, hairdryer or other small domestic appliance.

There is also a small risk of discomfort or skin infection from having the electrodes applied to the scalp. We keep this risk very low by taking great care in disinfecting our equipment and by using sensors that are embedded in small sponges, so that they are softer and more comfortable to wear.

When we put the electrode net on your head, it is wet, which can be uncomfortable at first. We try to keep the discomfort to a minimum by using warm water to soak the net, by providing towels to catch any drips, and by applying the net as quickly as possible.

You can stop taking part in the study AT ANY TIME.

The benefits to taking part in this study are ***indirect benefits*** – that is, the study will not directly benefit you. The study will help us find out more about how individuals with developmental dyslexia process and respond to specific visual stimuli. We hope that one day, this will help us to develop a better understanding of developmental dyslexia and its underlying causes.

VIDEO/AUDIO RECORDING CONSENT: We use VIDEO during the EEG part of the experiment for participant monitoring so we can make note of excessive movement, eye blinks, and general level of attention during the experiment. We do not actually record, so no video recording is saved and stored.

We will record audio of participant responses during the reading assessments. This is for detailed analysis later on and is essential to the study.

I agree _____ **OR** I do NOT agree_____ (Please check one)
to the audio-recording during assessments that is carried out for the purposes of this research study. Participant Initials _____

PAYMENTS: In addition to receiving a stress ball shaped in the form of a brain (value $1.00), participants will receive a $50.00 cash for participating. We recognize the valuable contribution of time participants are making to further our understanding of the potential underlying neural mechanisms that may contribute to dyslexia. The stress ball and gift card are a small token of appreciation.

DATA STORAGE TO PROTECT CONFIDENTIALITY: Your privacy is VERY important to us, and we are extremely careful to protect your identity.

Computer files will be stored on password-protected computers that can be accessed only by members of the research team. Electronic data will be stored in encrypted form. Paper copies of assessments and consent forms will be stored separately from identifying information in locked file cabinets that can only be accessed by authorized lab members. Data files are identified by numbers that are assigned separately to each participant. The only place where your name and identifying number will be stored together is on this consent form. You will be given a copy of this form to keep if you choose. The original copy will be stored in a locked filing cabinet in the laboratory that can be accessed only by members of the research team.

When we report results from our studies (e.g. at meetings to discuss research, or in professional journals), we usually report results from many individuals together, as averages. We NEVER use names when reporting data. We will not have access to your health records or any other identifying information.

TIME INVOLVEMENT: Your participation will take approximately 2 hours and 30 minutes, plus travel time.

HOW RESULTS WILL BE USED: The results of the study will be used in professional reports for publication in journals, and for presentation at professional and academic conferences.

CONSENT: Please sign below if you agree to take part in this study. By signing below, you agree that you have understood the nature of the study and what you will be asked to do, and you agree to take part. Please feel free to ask any questions if you are not sure.

I agree to _____[print name] take part in the study entitled **Neural Correlates of Early-Stage Visual Processing Differences in Developmental Dyslexia.**

I have had an opportunity to ask questions about the study, and I understand what is involved.

Signed: (Participant) _____

Date (mm/dd/yyyy): _____//_____//_____

Subject #_____

VERIFICATION: You are being asked to participate in this study EITHER because at some point during your educational career you received a diagnosis of developmental dyslexia from a professional _____ **OR** because you do NOT have any such diagnosis or family history of dyslexia (parent/sibling) or other neurological problem and will participate in our study for comparison purposes _____. (Please check one).

If you did receive a formal diagnosis of dyslexia, please indicate below that the diagnosis was obtained after a complete evaluation and was arrived at by a qualified professional (e.g., school psychologist, neuropsychologist). Please provide the age or grade of diagnosis _____. Note: You will not be asked to provide any specifics regarding this evaluation.

My signature below confirms that I received a diagnosis of developmental dyslexia by a qualified professional, such as a school psychologist or a neuropsychologist:

_____
(Participant Signature)

Please also sign the Participant's Rights form that follows.

Subject #_____

Teachers College, Columbia University
PARTICIPANT'S RIGHTS

Co-Investigators: Prof. Karen Froud and Ms. Lisa Levinson

Research Title: **Neural Correlates of Early-Stage Visual Processing Differences in Developmental Dyslexia**

- I have read and discussed the Research Description with the researcher. I have had the opportunity to ask questions about the purposes and procedures regarding this study.
- My participation in this research is voluntary. I may refuse to participate or may withdraw from participation at any time without jeopardy to future medical care, employment, student status, or other entitlements.
- The researcher may withdraw me from the research at his/her professional discretion.
- If, during the course of the study, significant new information that has been developed becomes available that could affect my willingness to continue to participate, the investigator will provide this information to me.
- Any information derived from the research project that personally identifies me will not be voluntarily released or disclosed without my separate consent, except as specifically required by law.
- If at any time I have any questions regarding the research or my participation, I can contact either co-investigators, Prof. Karen Froud or Ms. Lisa Levinson, who will answer my questions. Dr. Froud may be reached at (212) 678-8169, and Ms. Levinson may be reached at (917) 882-1934.
- If at any time I have comments or concerns regarding the conduct of the research or questions about my rights as a research subject, I should contact the Teachers College, Columbia University Institutional Review Board (IRB). The phone number for the IRB is (212) 678-4105. Or, I can write to the IRB at Teachers College, Columbia University, 525 W. 120th Street, Box 151, New York, NY, 10027.
- I can expect to receive a copy of the Research Description and this Participant's Rights document.

My signature means that I agree to participate in this study.

Participant's Signature:
_____

Print Name: _____
Date:____/____/____

Neurocognition of Langauge Lab
Teachers College, Columbia University
www.tc.edu/neurocog
212-678-8169
NCLLAB@TC.Columbia.edu

Appendix E

Participant Questionnaire and Responses

Confidential

Participant Questionnaire - Adult (Dyslexia Study)

TEACHERS COLLEGE COLUMBIA UNIVERSITY

Neural Correlates of Early-Stage Visual Processing Differences in Developmental Dyslexia

Please answer the following questions. Thank you for taking the time to provide us with this valuable background information.

Today's Date (M-D-Y): _____

Gender:
- ◯ Male
- ◯ Female

Date of Birth (D-M-Y): _____

Is English your primary (first) language?
- ◯ Yes
- ◯ No

To the best of your knowledge, were you early or late to begin any of the following:

Note: All children develop at their own rate. Early or late onset of milestones is not necessarily an indication of atypical development.

Crawling?
- ◯ Early (6 months)
- ◯ Typical Development (9 months)
- ◯ Late (12 months)
- ◯ Unsure

Walking?
- ◯ Early (9 months)
- ◯ Typical Development (12 months)
- ◯ Late (18 months)
- ◯ Unsure

Talking? (single words)
- ◯ Early (9 months)
- ◯ Typical Development (12 months)
- ◯ Late (2+ years)
- ◯ Unsure

Tying shoes?
- ◯ Early (4 years)
- ◯ Typical Development (5 years)
- ◯ Late (6+ years)
- ◯ Unsure

Handedness:
- ◯ Right Handed
- ◯ Left Handed
- ◯ Ambidextrous

Do you sometimes have difficulty telling left from right?
- ◯ Yes
- ◯ No

Do you have an excellent memory for experiences, locations, and faces but difficulty recalling facts or information not experienced?
- ◯ Yes
- ◯ No

Do you lose track of time?
- ◯ Yes
- ◯ No

Have you been diagnosed with any of the following (check all that apply):	☐ Dyslexia ☐ Dysgraphia (poor to illegible handwriting) ☐ Dyscalculia (significant difficulty with math) ☐ Attention Deficit Hyperactivity Disorder (ADHD) ☐ Other (please specify below), or none of the above
Please specify any additional or other diagnoses here:	_____
When recounting a story, do you find that you frequently mix up the sequence of events?	◯ Yes ◯ No
Is there a family history of reading and/or spelling problems?	◯ Yes ◯ No
You consider your reading speed to be:	◯ Slow ◯ Average ◯ Fast
Do you have difficulty with grammar?	◯ Yes ◯ No
When writing, do you have difficulty expanding your ideas?	◯ Yes ◯ No
When reading directions or assembling a piece of furniture, do you prefer written instructions or diagrams?	◯ Written Instructions ◯ Diagrams ◯ Both
When you encounter an unfamiliar word, do you find it difficult to "sound it out"?	◯ Yes ◯ No
Do you have difficulty with spelling?	◯ Yes ◯ No
Do you ever use an incorrect word in place of a similar sounding word, sometimes resulting in a humorous phrase?	◯ Yes ◯ No
Do you avoid reading aloud in public?	◯ Yes ◯ No
When you read a passage, do you focus more on the details or the big picture?	◯ Details ◯ Big Picture ◯ Both to the Same Extent
When you are tired, under a time constraint, or emotionally stressed do you notice that you become more easily confused and makes more mistakes than usual?	◯ Yes ◯ No ◯ Unsure
How much reading do you do daily?	◯ Less than 30 minutes ◯ From 30 minutes to an hour ◯ From 1 to 2 hours ◯ From 2 hours to 4 hours ◯ More than 4 hours
Do you enjoy math?	◯ Yes ◯ No

Which, if any, of these math skills do you have trouble with (check all that apply)?

- ☐ Addition
- ☐ Subtraction
- ☐ Multiplication
- ☐ Division
- ☐ Word Problems
- ☐ None

Do you sometimes reverse numbers (for example, write 24 for 42)?

- ◯ Yes
- ◯ No

Do you generally enjoy reading?

- ◯ Yes
- ◯ No

As a child, did you receive any tutoring outside of school?

- ◯ Yes
- ◯ No

If so, how many times a week and for how long? _____

Do you notice that when reading you find it difficult to track a sentence across the page with your eyes?

- ◯ Yes
- ◯ No
- ◯ Unsure

Do you notice that sometimes your eyes rush back and forth horizontally for short periods of time while reading?

- ◯ Yes
- ◯ No
- ◯ Unsure

Do you frequently need to reread words, sentences, or passages multiple times to comprehend them?

- ◯ Yes
- ◯ No

Do you find it difficult to focus on the words on the page, that they blur and/or drift to other lines?

- ◯ Yes
- ◯ No

Please feel free to make any specific comments about your experience of learning to read and/or with the experience of reading today.

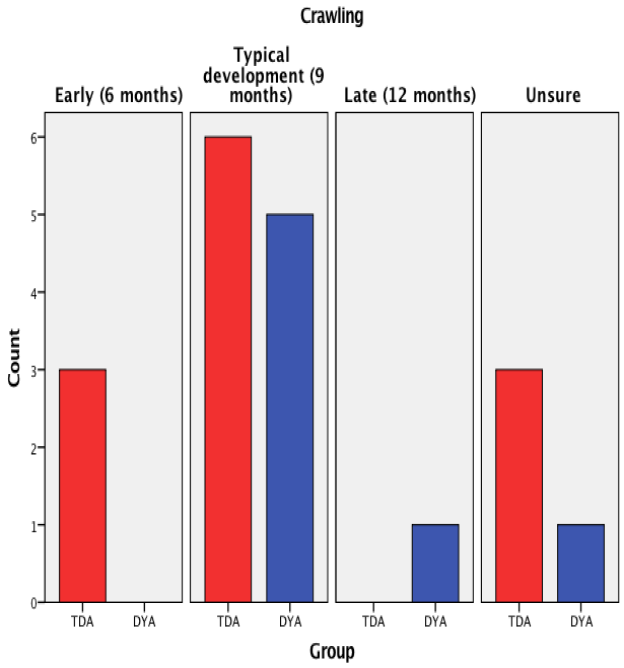
Some of the questions from this survey were adapted from the checklist featured on the Bristol Dyslexia Centre website (http://www.dyslexiacentre.co.uk/signs-of-dyslexia/) as well as the International Dyslexia Association (http://www.interdys.org/AreYouDyslexic_AdultTest.htm). These questions probe issues with spelling, memory, organization, sequencing and the reading experience.

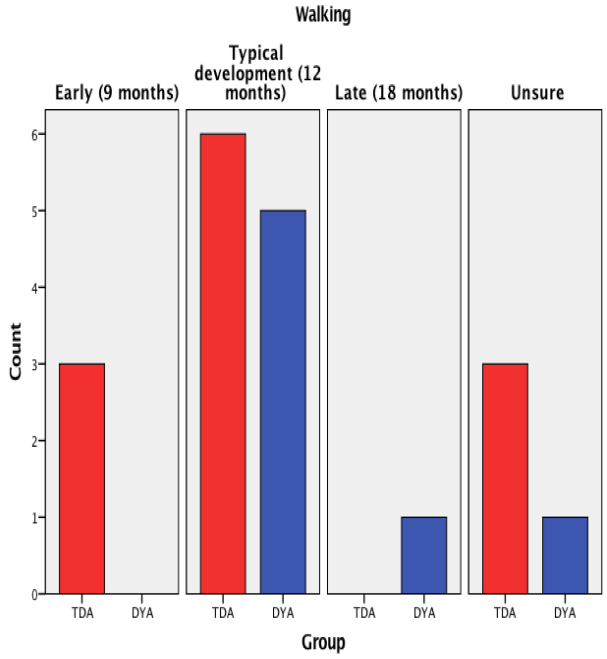
Questionnaire Responses

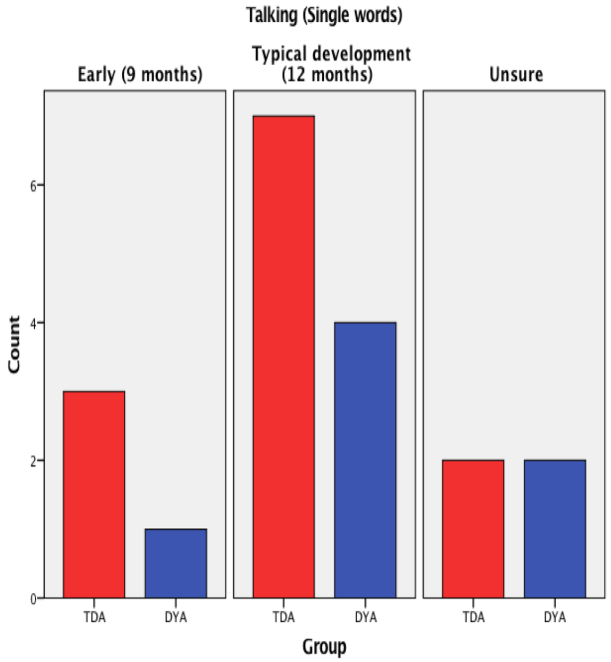
Experimental Group/Individuals with Dyslexia/DYA (n=7)

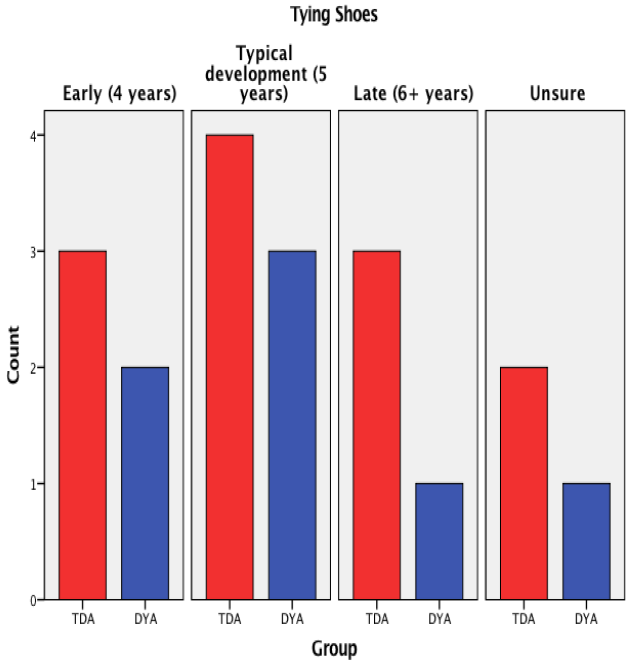
Comparison Group/Individuals without Dyslexia/TDA (n=11)

To the best of your knowledge, were you early or late to begin any of the following:









Handedness

	TDA	DYA
Right	12	7
Left	0	0

Do you sometimes have difficulty telling left from right?

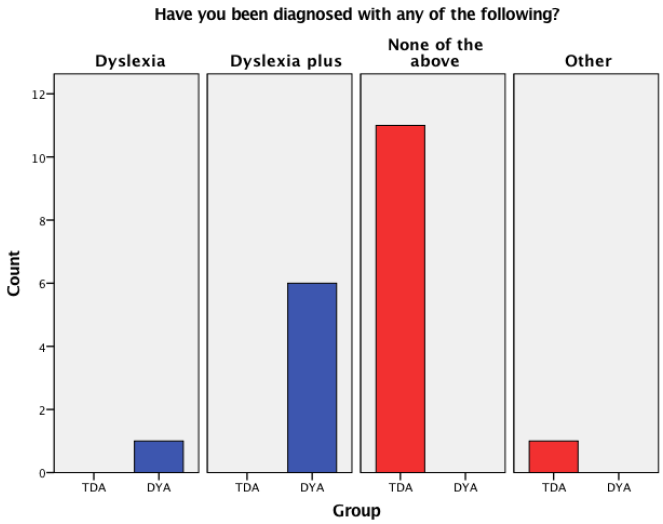
	TDA	DYA
Yes	3	5
No	9	2

Do you have an excellent memory for experiences, locations, and faces but difficulty recalling facts or information not experienced?

	TDA	DYA
Yes	2	7
No	10	0

Do you lose track of time?

	TDA	DYA
Yes	7	6
No	5	1



When recounting a story, do you find that you frequently mix up the sequences?

	TDA	DYA
Yes	0	4
No	12	3

Is there a family history of reading and/or spelling problems?

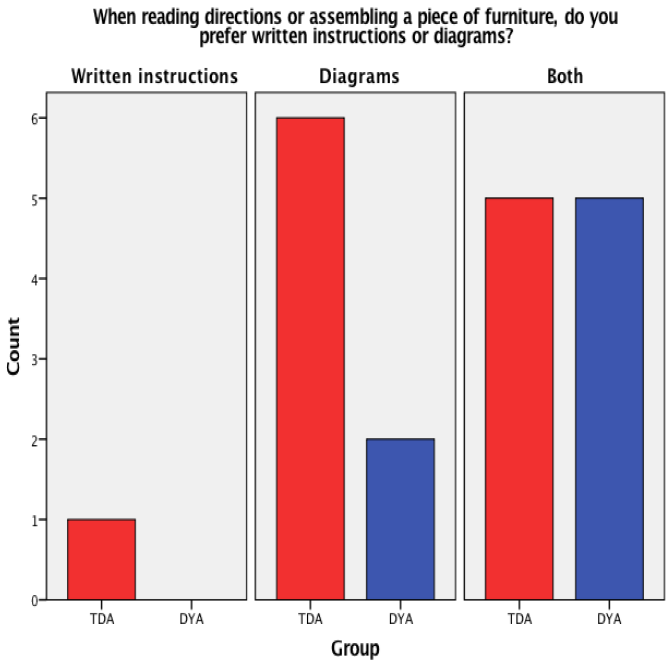
	TDA	DYA
Yes	0	6
No	12	1

Reading Speed

	TDA	DYA
Fast	4	
Average	8	2
Slow		5

Do you have difficulty with grammar?

	TDA	DYA
Yes	1	6
No	11	1



When writing, do you have difficulty expanding your ideas?

	TDA	DYA
Yes	1	6
No	11	1

When you encounter an unfamiliar word, do you find it hard to "sound it out"?

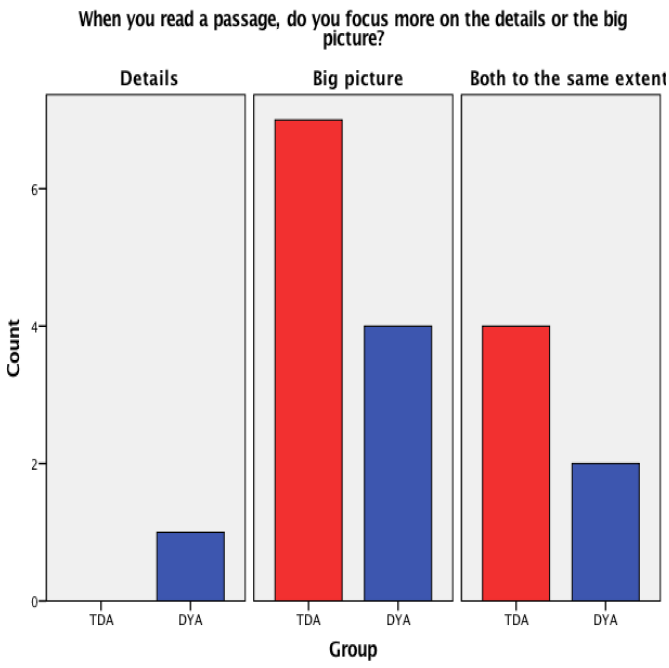
	TDA	DYA
Yes	3	6
No	9	1

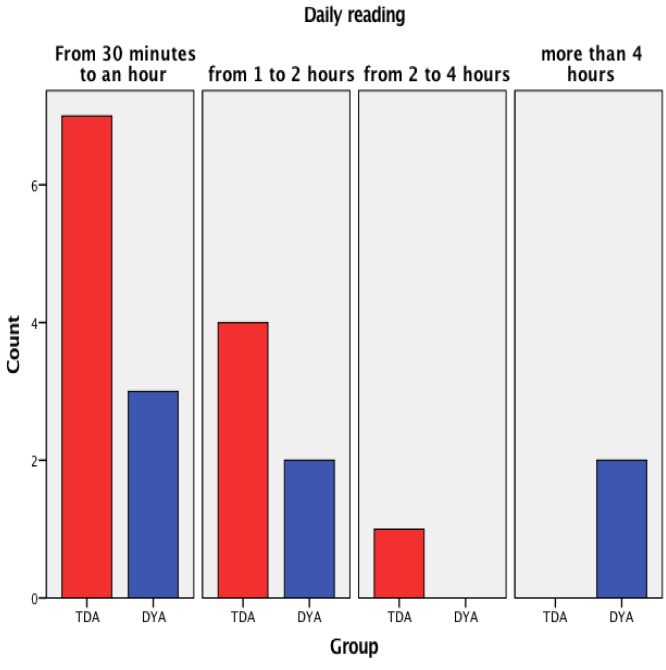
Do you have difficulty spelling?

	TDA	DYA
Yes	1	7
No	11	0

Do you ever use an incorrect word in place of a similar sounding word, sometimes resulting in a humorous phrase?

	TDA	DYA
Yes	2	5
No	10	2





Do you avoid reading aloud in public?

	TDA	DYA
Yes	3	7
No	9	0

When you are tired, under a time constraint, or emotionally stressed do you notice that you become more easily confused and make more mistakes than usual?

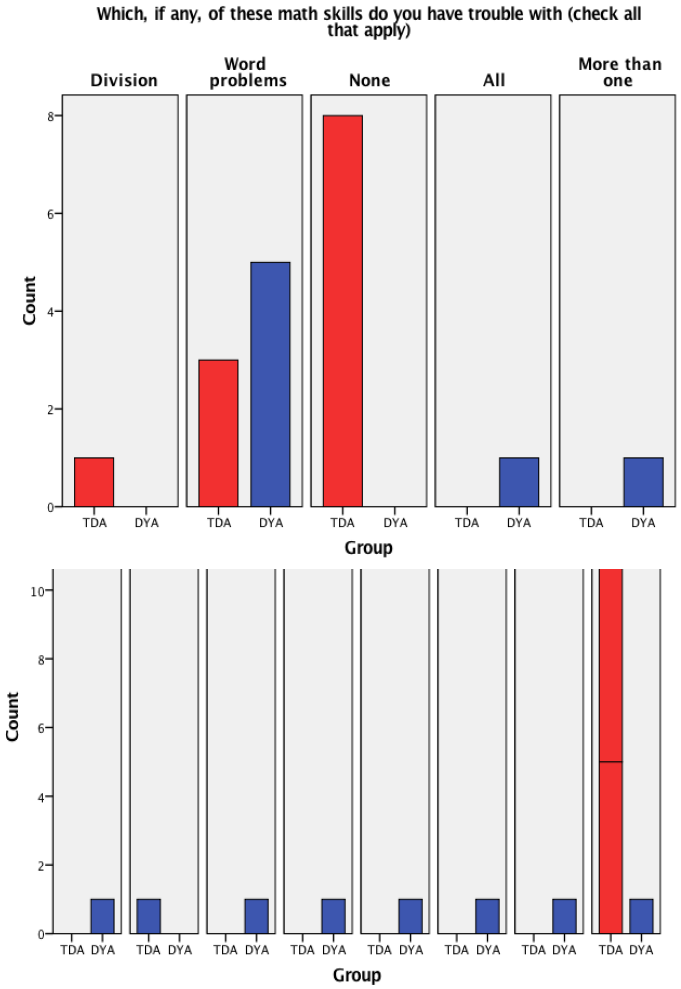
	TDA	DYA
Yes	8	6
No	3	0
Unsure	1	1

Do you enjoy math?

	TDA	DYA
Yes	11	3
No	1	4

Do you sometime reverse numbers (for example, write 24 for 42)?

	TDA	DYA
Yes	0	6
No	12	1



As a child, did you receive any tutoring outside of school?

	TDA	DYA
Yes	1	6
No	11	1

Do you generally enjoy reading?

	TDA	DYA*
Yes	9	3
No	2	3

*Missing one response

Do you notice that when reading you find it difficult to track a sentence across the page with your eyes?

	TDA	DYA

Yes	0	5
No	11	1
Unsure	1	1

Do you notice that sometimes your eyes rush back and forth horizontally for short periods of time while reading?

	TDA	DYA
Yes	2	2
No	8	2
Unsure	2	3

Do you frequently need to reread words, sentences, or passages multiple times to comprehend them?

	TDA	DYA
Yes	3	7
No	9	0

Do you find it difficult to focus on the words on the page, that they blur and/or drift to other lines?

	TDA	DYA
Yes	1	3
No	11	4

Appendix F

Orthographic Choice Task

Worksheet for Orthographic Choice task –**Adult** Participant No: _____
Instructions: *Look at the list of words in each section. In each pair there is a real word and a non word (they may look and sound like real words but they are not). Circle the* real word *in each pair.*

Section A:

1.	tight / tite	17.	sheep / sheap
2.	minuscule / miniscule	18.	little / littel
3.	onsomble / ensemble	19.	liquify / liquefy
4.	cologne / colone	20.	store / stoar
5.	opening / opaning	21.	tyre / tire
6.	meant / ment	22.	between / betwean
7.	biscuit / biscut	23.	style / stile
8.	poultry / poltry	24.	sheltor / shelter
9.	tortus / tortoise	25.	trowsers / trousers
10.	debris / debree	26.	pius / pious
11.	indite / indict	27.	culprit / culpret
12.	villain / villen	28.	grammar / grammer
13.	weird / weerd	29.	stimey / stymie
14.	meringue / merang	30.	victer / victor
15.	clenz / cleanse	31.	sallary / salary
16.	benine / benign	32.	fascinate / fassinate

Section B:

1.	impune / impugn	17.	warrant / warrent
2.	ake / ache	18.	stait / state
3.	caraffe / carafe	19.	airloom / heirloom
4.	muscle / mussle	20.	didactic / dydactic
5.	court / cort	21.	guyser / geyser
6.	gaurd / guard	22.	decrepit / dicrepit
7.	column / collum	23.	smoke / smoak
8.	pagent / pageant	24.	raisin / raizen
9.	vacume / vacuum	25.	nostrils / nostrels
10.	knife / nife	26.	pursute / pursuit
11.	slite / sleight	27.	license / lisense
12.	forfeit / forefit	28.	seperation / separation
13.	epilog / epilogue	29.	tempal / temple
14.	caviat / caveat	30.	sophomore / sofmore
15.	subtle / suttle	31.	surprise / surprize
16.	believe / beleave	32.	preceed / precede

Appendix G

Homophone Choice Task

Instructions and response sheet for homonym choice task –**Adults**
Participant No: _____

Practice Items
I am going read you a question and want you to point to the word on your chart that answers the question.
For example,

1. Which is a color? **blue** / blew

Point to the word on your list that is a color. *If subject points incorrectly or does not understand instruction, point to the word blue and say* see blue is a color.
Now lets try some more:

2. Which is a food? **chili** / chilly
3. Which has 7 days? weak / **week**

Section A: Test Items (correct responses in bold; circle participant's response)

1. What did the cow do? **mooed** / mood
2. What do the kings do? rein/ **reign**
3. Who heads the school? **principal** / principle
4. Who rides a broomstick? **witch** / which
5. What do you do with milk? pore/ **pour**
6. Where is your belly button? **waist** / waste
7. Which is to brings up? raze / **raise**
8. Which came before? passed / **past**
9. Which comes from a bank? **loan** / lone
10. Which contains prisoners? **cell** / sell
11. What can you do with vegetables? great / **grate**
12. Which is a flower? rows / **rose**
13. Which is a food? beat / **beet**
14. Which is a food? **cereal** / serial
15. Which is a location? **there** / their
16. What do muscles do? flecks / **flex**
17. What do politician care about? **poll** / pole
18. What happened to the lawn? moan / **mown**
19. What is a clump of hay? **bale** / bail
20. Where does the nobleman live? **manor** / manner
21. Which belongs in a lab? **vial** / vile
22. Which goes on a hotdog? **mustard** / mustered
23. Which has recorded music? disk / **disc**
24. Which is a grain? **rye** / wry
25. Which is a location? **site** / cite
26. Which is a measure of speed? **mach** / mock

27. Which is a paradise? idol **/ idyl**
28. Which is a plant? **yew** / ewe
29. Which is an animal? **hart** / heart
30. Which is a number? ate / **eight**

Section B: Test Items

1. Which is an instrument? liar / **lyre**
2. Which is a number? too / **two**
3. Which is part of the body? **feet** / feat
4. Which is an animal sound? nay / **neigh**
5. Which is an animal? **horse /** hoarse
6. Which is clothing? genes / **jeans**
7. Which is found on a ship? massed / **mast**
8. Which is not allowed? **banned /** band
9. Which is part of a car? **brake /** break
10. Which may be flat? **board** / bored
11. Which means ownership? **have** / halve
12. Which one are you on? sighed / **side**
13. Who came to visit? **guest** / guessed
14. What happens to library books? overdo / **overdue**
15. What keeps up posters? tax / **tacks**
16. Which is found on a garment? rough / **ruff**
17. Which is in the supermarket? **aisle** / isle
18. Which is individual? discreet / **discrete**
19. Which is part of a hairstyle? plate / **plait**
20. Which is really not much? sleight / **slight**
21. Which is used for painting? **palette /** palate
22. Which is used for tying? **cord** / chord
23. Which is very tiny? might / **mite**
24. Which lives under the sea? soul / **sole**
25. Which opens? **pries** / prize
26. Which sound does a bell make? peel / **peal**
27. Which surrounds a castle? mote / **moat**
28. Who is a friend? **peer** / pier
29. Who is really tall? **titan /** tighten
30. Who leads the soldiers? martial / **marshal**

Section A + B total = / 60

Word List : Homonym Choice – Adults

A

mooed	**mood**
rein	**reign**
principal	**principle**
witch	**which**
pore	**pour**
waist	**waste**
raze	**raise**
passed	**past**
cell	**sell**

great	**grate**
rows	**rose**
beat	**beet**
cereal	**serial**
there	**their**

flecks	**flex**
poll	**pole**
moan	**mown**
bale	**bail**
manor	**manner**

vial	**vile**
mustard	**mustered**
disk	**disc**
rye	**wry**
site	**cite**

mach	**mock**
idol	**idyl**
yew	**ewe**
hart	**heart**
ate	**Eight**

liar	**lyre**
too	**two**
feet	**feat**
nay	**neigh**
horse	**hoarse**

genes	**jeans**
massed	**mast**
banned	**band**
brake	**break**
board	**bored**

have	**halve**
sighed	**side**
guest	**guessed**
overdo	**overdue**
tax	**tacks**

rough	**ruff**
aisle	**isle**
discreet	**discrete**
plate	**plait**
sleight	**slight**

palette	**palate**
cord	**chord**
might	**mite**
soul	**sole**
pries	**prize**

peel	**peal**
Mote	**moat**
peer	**pier**
titan	**tighten**
martial	**marshal**

Appendix H

Non-Verbal Visual Reasoning/Memory

Nonverbal Visual Reasoning/Memory
Directions/Scoring Sheet

Participant number _____

Directions:
Show the participant the Sample/Practice card and say, "I want you to look carefully at the order of the design on this card and to remember it."

Show the stimulus card for 10 seconds. Then flip to the next card and say, "which one of the group of designs are exactly the same as the one you were just shown? Please point to the one that is the same.

The participant responds by pointing.

I am going to show you some additional designs followed by four items as I have here, just point to the exact design previously presented.

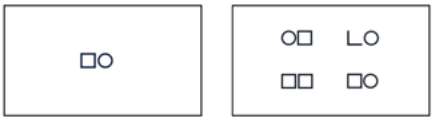
Indicate on the adjacent scoring template which design the participant pointed to, do not prompt.

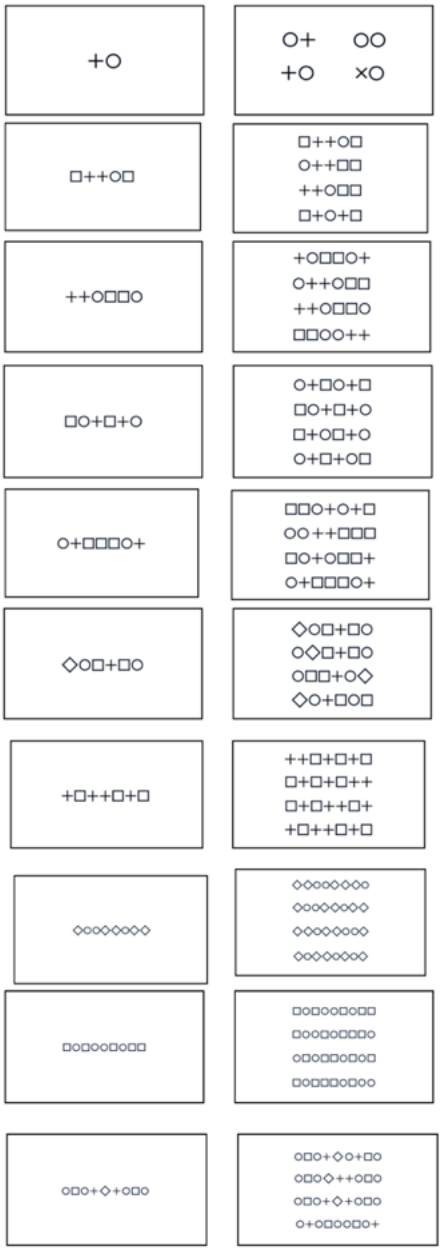
Scoring
1 point for each correct response.

Max. Score is 10.

Raw Score: _____

SAMPLE/PRACTICE – First Card

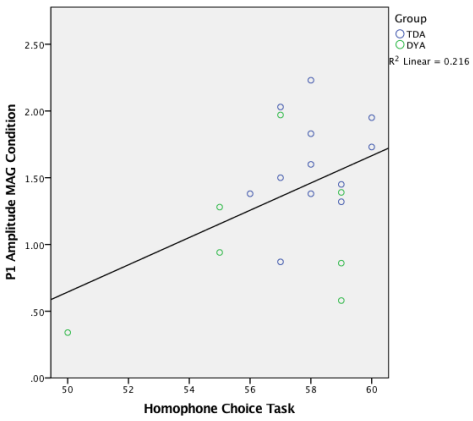


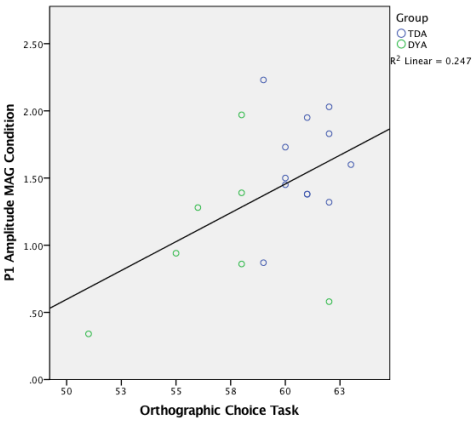


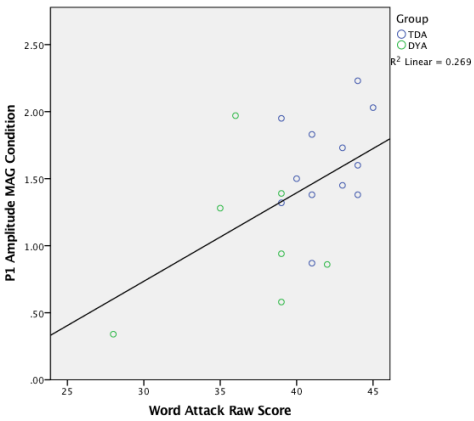
Appendix I

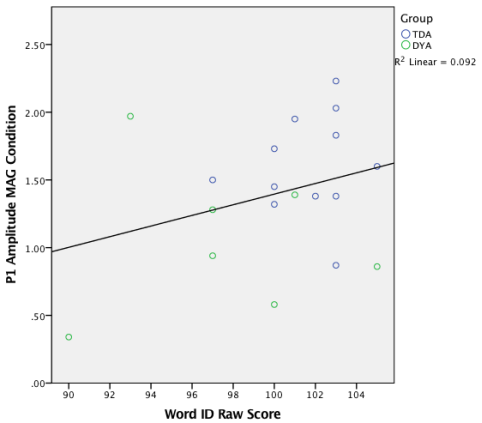
Scatterplots

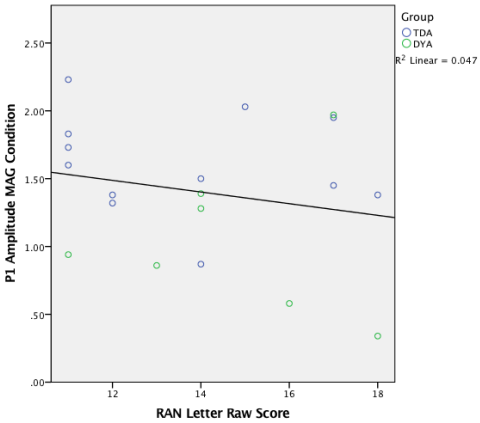
P1 Amplitude – magnocellular/motion condition with behavioral assessments:

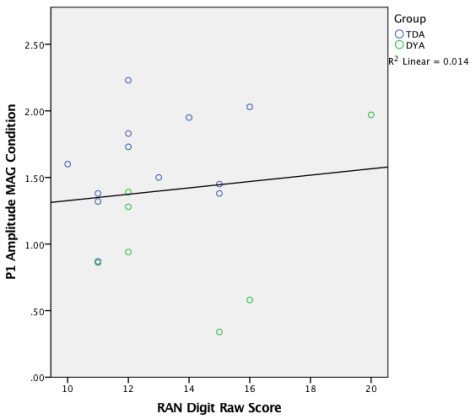












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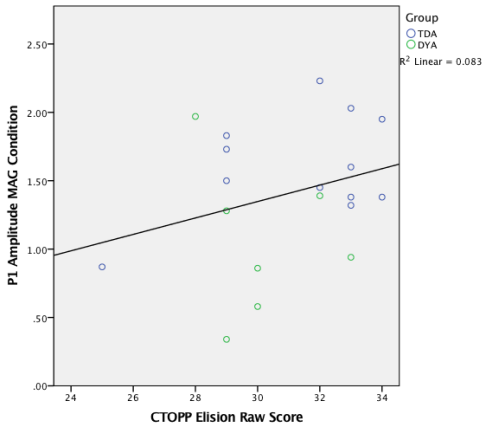

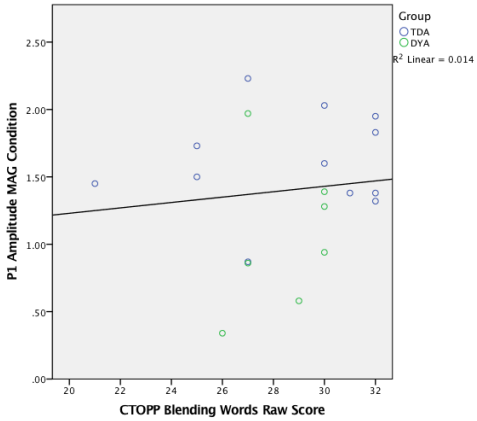

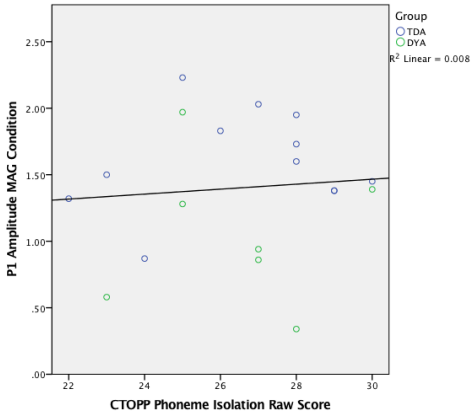

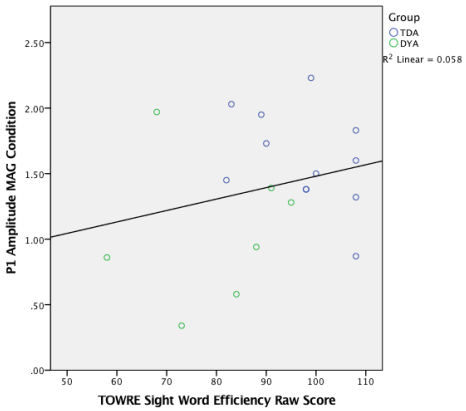

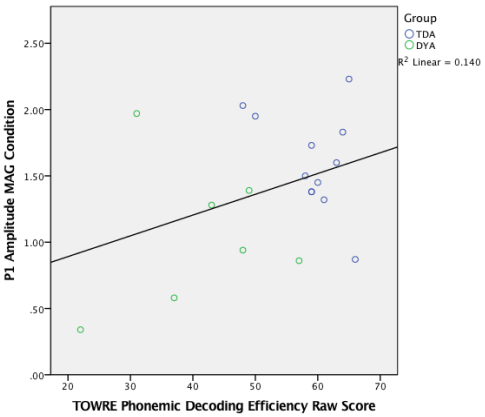

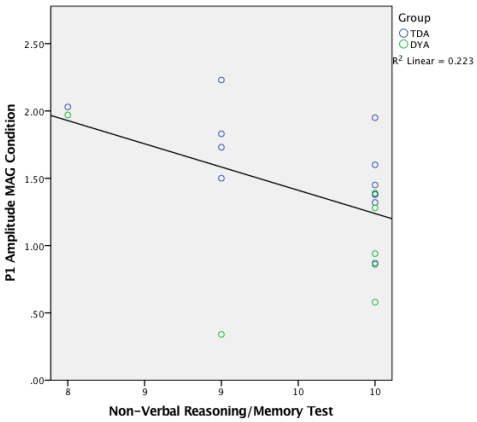

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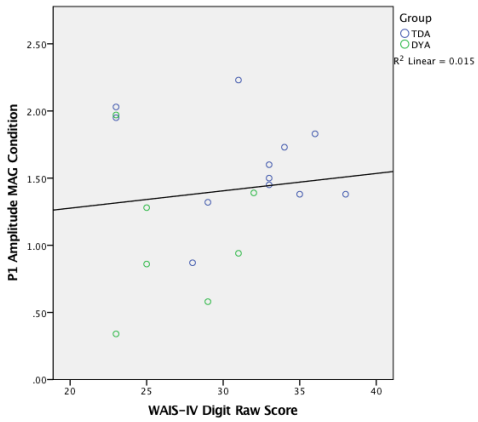
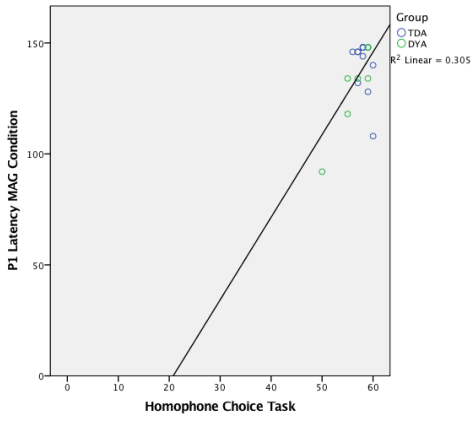
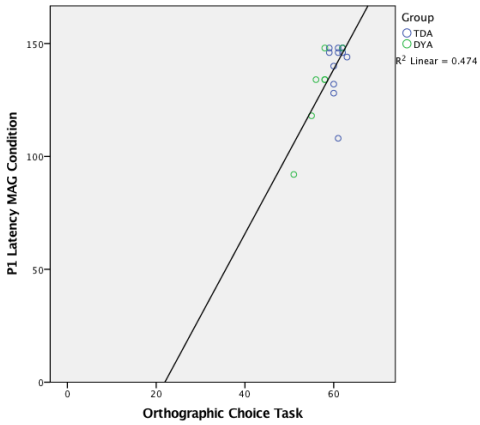


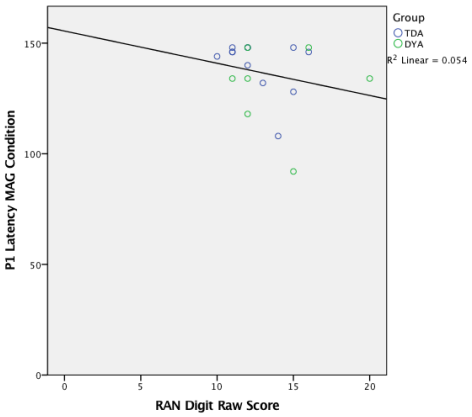
P1 Latency - magnocellular/motion condition with behavioral assessments:

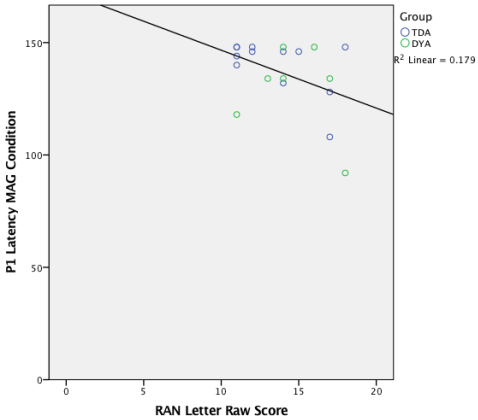


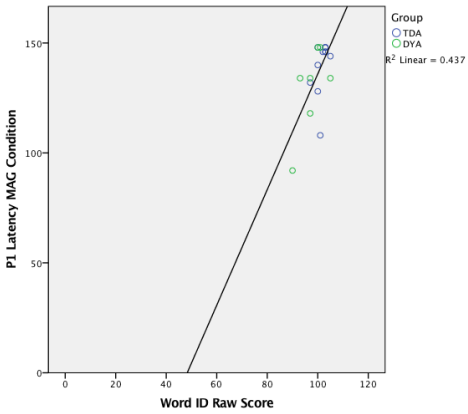


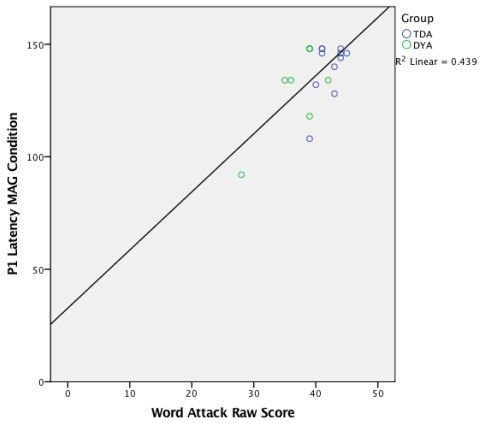
Continued P1 Latency - magnocellular/motion condition with behavioral assessments:

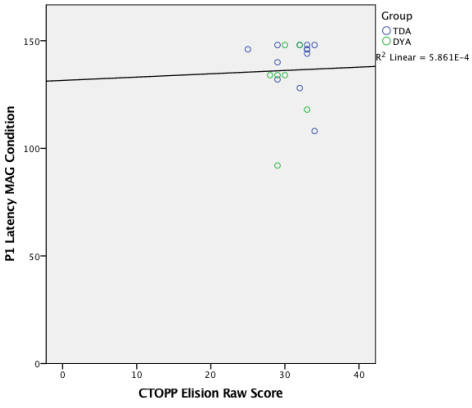
Continued P1 Latency - magnocellular/motion condition with behavioral assessments:

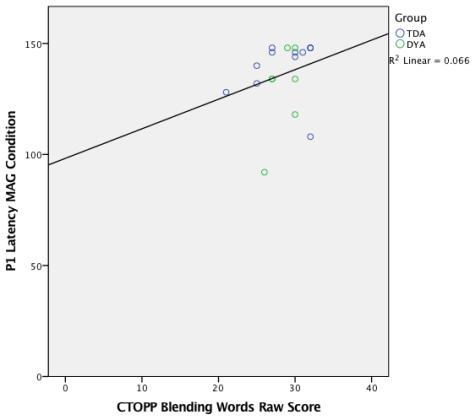




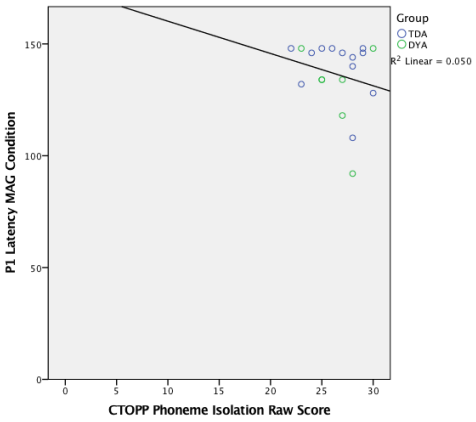


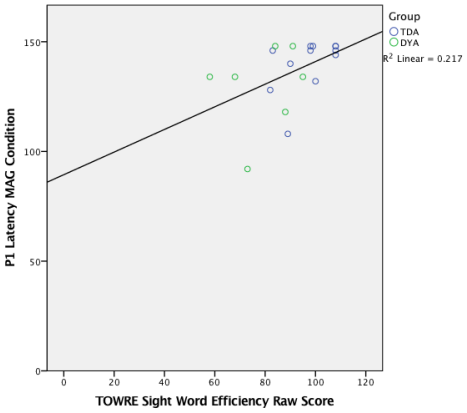


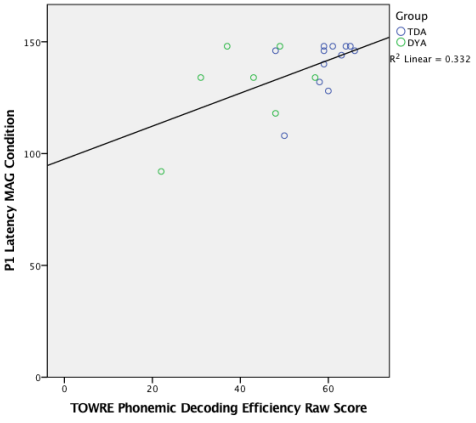


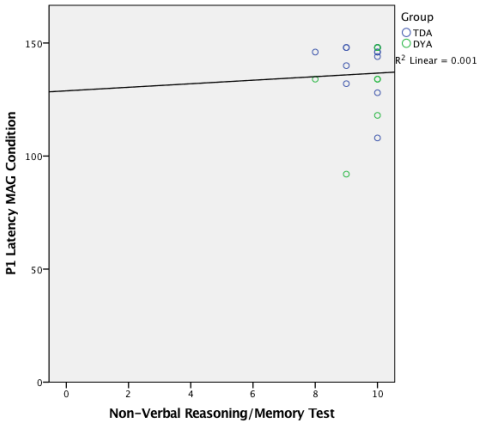


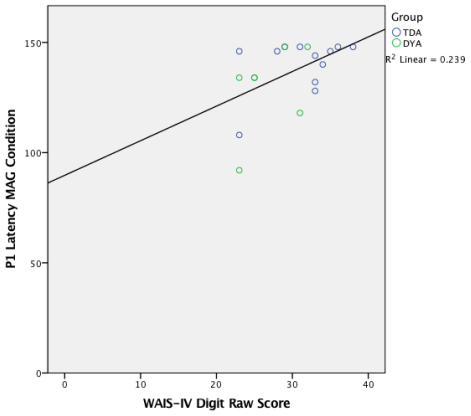
Continued P1 Latency - magnocellular/motion condition with behavioral assessments:



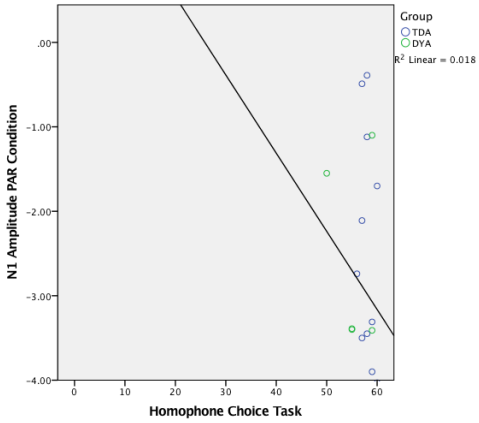


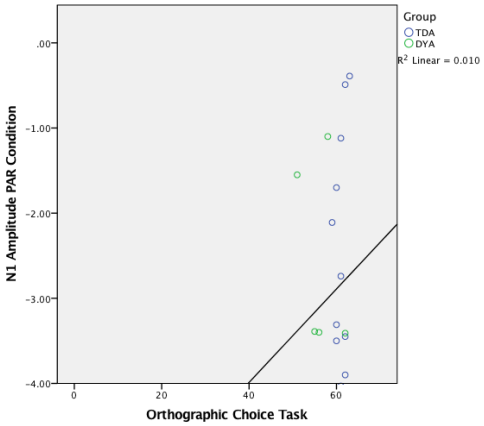


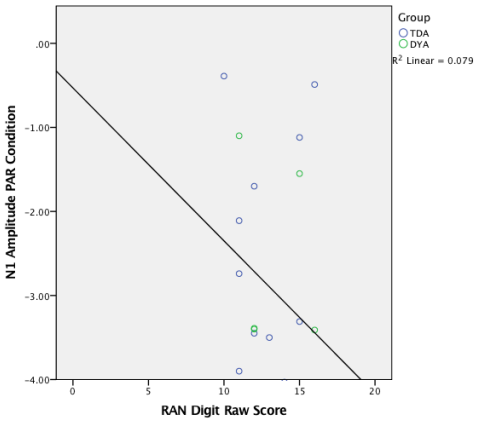


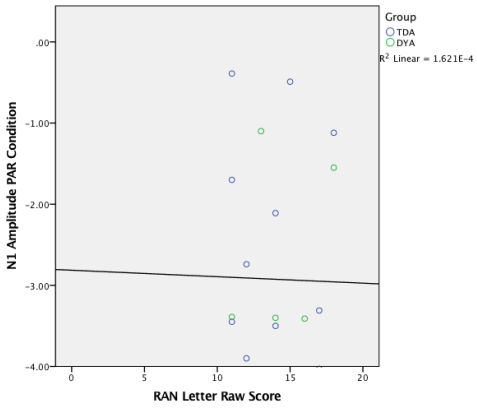


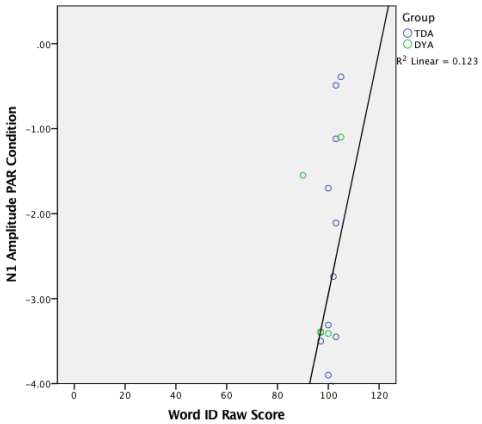
N1 Amplitude – parvocellular/color condition with behavioral assessments:

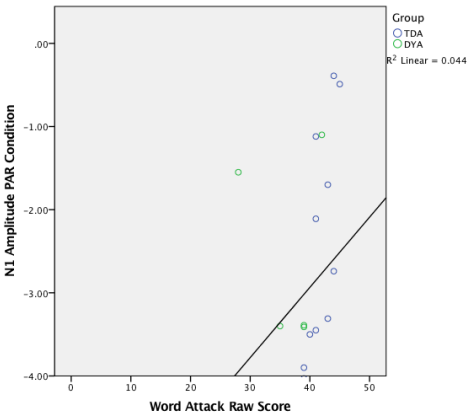




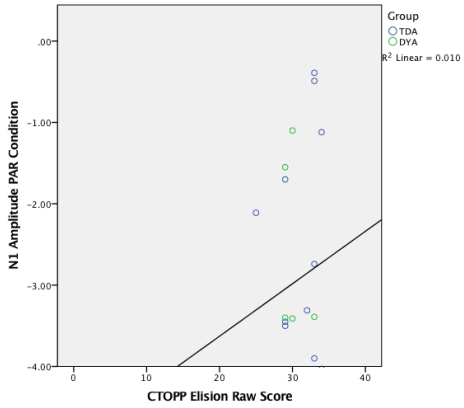


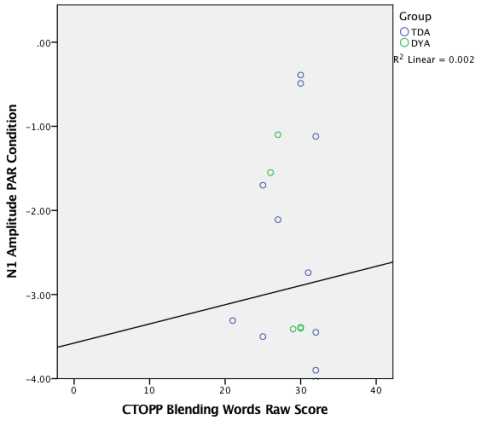


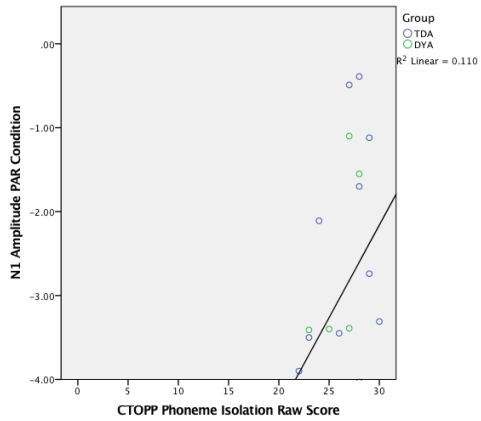


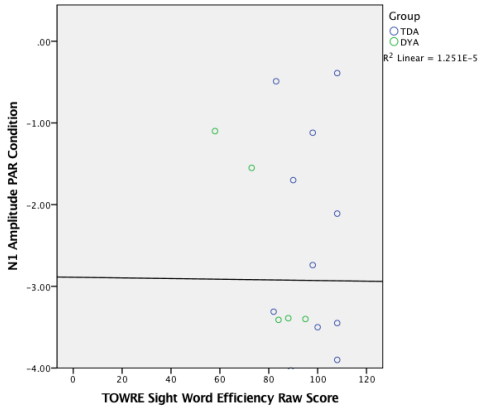


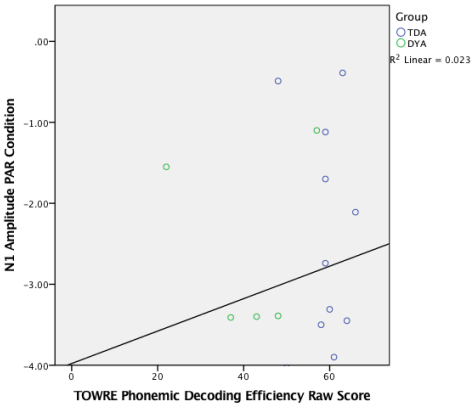
Continued N1 Amplitude – parvocellular/color condition with behavioral assessments:

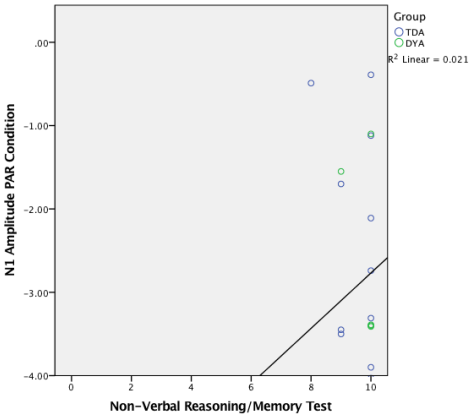




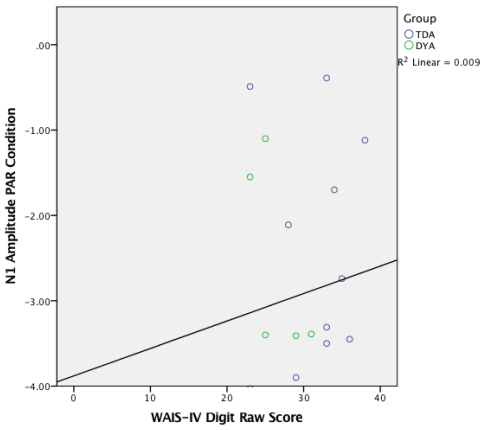




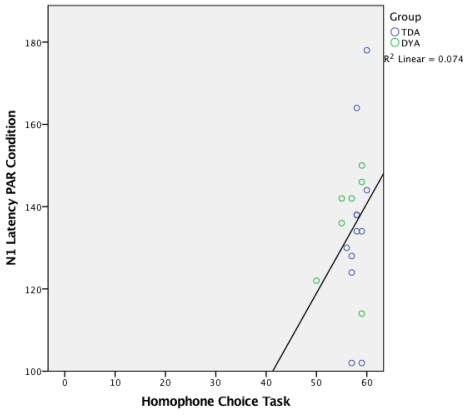


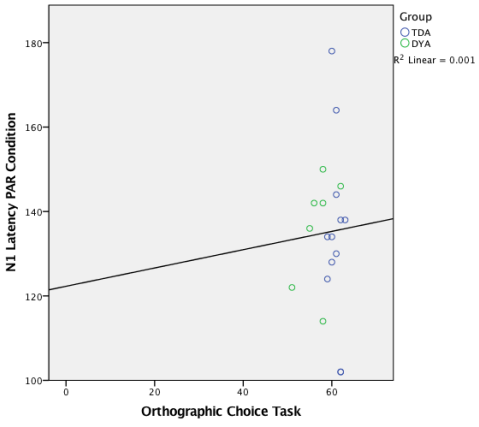


Continued N1 Amplitude – parvocellular/color condition with behavioral assessments:

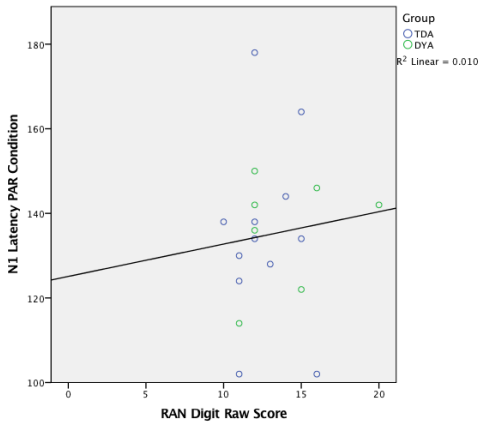


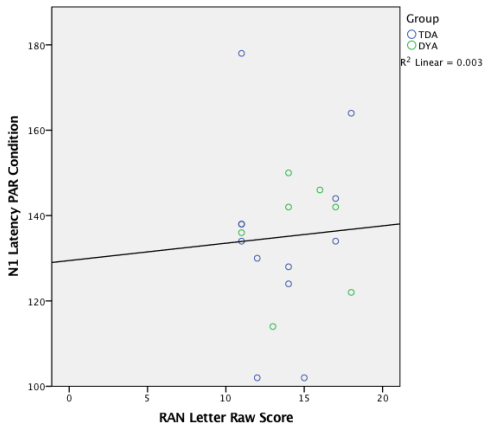
N1 Latency – parvocellular/color condition with behavioral assessments:

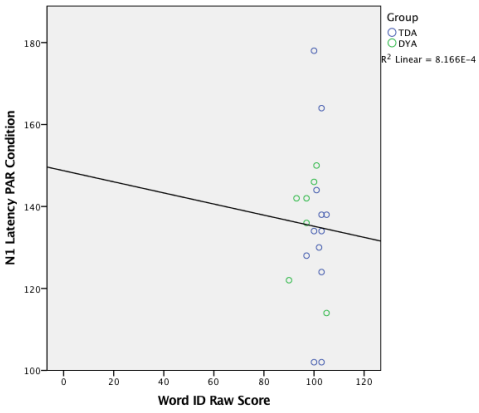


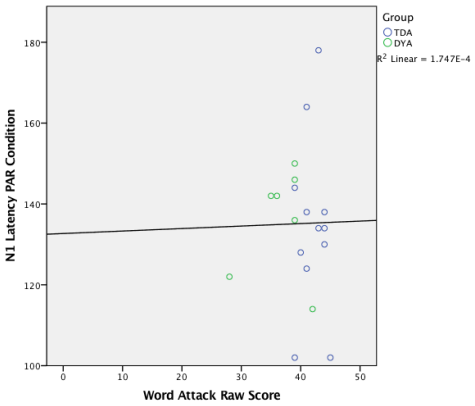


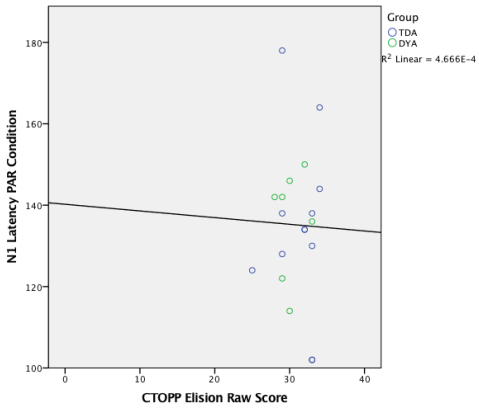
Continued N1 Latency – parvocellular/color condition with behavioral assessments:

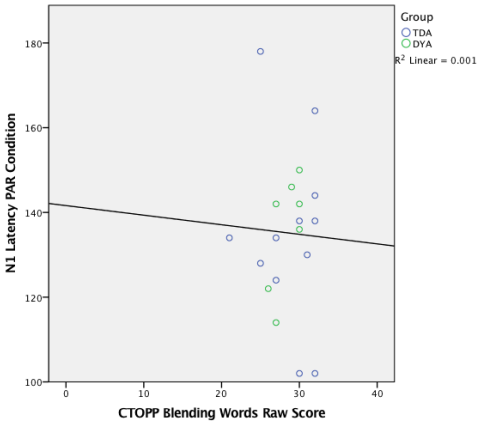












Continued N1 Latency – parvocellular/color condition with behavioral assessments: