LITERACY MATURITY AND THE MAGNOCELLULAR THEORY OF DYSLEXIA: IMPLICATIONS FOR CLINICAL DIAGNOSIS

A Dissertation

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by

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ABSTRACT

Dyslexia diagnosis suffers from reduced diagnostic power without a diagnostic sign, or a distinguishing characteristic to pinpoint the diagnosis. Many argue that phonological skill provides a diagnostic sign (Ramus et al., 2003), yet not every child with dyslexia shows phonological weakness (Frederickson & Frith, 1998; White et al., 2006). Others argue that deviant magnocellular (m-cell) function may serve as a diagnostic sign (Stein, 2019). However, very few studies on m-cell deviance in dyslexia account for literacy acquisition in their models (Goswami, 2015), and it has been suggested that literacy acquisition may drive m-cell development (Olulade et al., 2013). This study tested the usefulness of m-cells as a diagnostic sign of dyslexia by comparing m-cell function in 8-to 14-year-old children with dyslexia to age-matched peers *and* younger, reading level-matched peers, accounting for nonverbal ability in a multiple regression model. M-cell function was assessed with a coherent motion task targeting area V5/MT. Results indicate that not all children with dyslexia have deviant m-cell performance in area V5/MT; however, the opposing theory of literacy-driven m-cell development also lacked support. Timed nonword reading appeared a more likely diagnostic sign than m-cell function, and results of an exploratory multiple regression analysis confirmed deviance in timed nonword ability for children with dyslexia, accounting for timed sight word reading and phonological ability. Though typically used to support a phonological-only approach to dyslexia, timed nonword reading has a heavy orthographic component that requires visual input. Given the biological basis of timed nonword reading, the role of m-cells in dyslexia could not be ruled out at this time. Implications for dyslexia diagnosis are discussed, and a cohesive rather than competitive approach to future research on dyslexia's biological origin is presented.

Keywords: magnocellular cells, magnocellular theory, dyslexia diagnosis, timed nonword reading

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APPROVAL OF THE DISSERTATION

This dissertation, "Literacy Maturity and the Magnocellular Theory of Dyslexia: Implications for Clinical Diagnosis," has been approved by the Graduate Faculty of the Curry School of Education in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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DEDICATION

This dissertation project is dedicated to the Batson family, who first challenged me to explain why reading was so difficult for their child.

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I have been blessed with many students and colleagues who have shaped my thinking about dyslexia and literacy over the last decade. To my dear friend, Jordan Buckrop, thank you for pushing me to carefully listen, take time to consider, and thoughtfully respond to the person, not the dyslexia.

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INTRODUCTION

Statement of the Problem

For over a century, children have been diagnosed with *dyslexia*, a puzzling or unexpected difficulty learning to read and spell. Efforts to identify and assist students with dyslexia have risen in recent years, leading to dyslexia legislation in 42 states as of 2018 (Youman & Mather, 2018). Despite the attention and over 100 years of diagnosis, researchers continue to debate the cause of dyslexia (Elliott & Grigorenko, 2014; Goswami, 2015). Many researchers believe that difficulties with language processing cause dyslexia (Adams, 1990; Gabrieli, 2009; Harm & Seidenberg, 1999; Ramus et al., 2003; Stanovich, 1994; Vellutino, Fletcher, Snowling, & Scanlon, 2004; Wagner & Torgesen, 1987); others believe that impairments with visual processing cause dyslexia (Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Livingstone, Rosen, Drislane, & Galaburda, 1991; Stein, 2001, 2018a; Stein & Walsh, 1997; Vidyasagar & Pammer, 2009). The absence of a proven cause of dyslexia reduces *diagnostic sensitivity*, the ability to identify those with dyslexia, and *diagnostic specificity*, the ability to accurately rule out dyslexia in others. Finding a *diagnostic sign* of dyslexia, or the presence of a distinguishing characteristic or set of characteristics that pinpoint the diagnosis, even if the cause remained unknown, would bring precision to dyslexia diagnosis. With low

diagnostic power and no diagnostic sign, prevalence rates of dyslexia range widely from 1.5 to 20% (Elliott & Grigorenko, 2014), raising suspicions of misdiagnosis.

When children are misdiagnosed with dyslexia, misappropriated resources leave many other children untreated or placed in inappropriate treatments. Untimely and unsuitable remedial plans fail to help children with dyslexia, many of whom experience crippling effects at school. Most children with dyslexia read and spell well below same-aged peers (Catts & Adolf, 2011; Vellutino et al., 2004) and have negative dispositions toward reading (Polychroni, Koukoura, & Anagnostou, 2007; Shaywitz, 2003). Not only does dyslexia make reading and spelling more difficult, but math, science, and history can be challenging as reading is required across all subject areas. Teachers are often confused about dyslexia and how to meet the needs of the dyslexic child (Wadlington & Wadlington, 2005; Washburn, Binks-Cantrell, & Joshi, 2014; Washburn, Joshi, & Binks-Cantrell, 2011; Washburn, Mulcahy, Musante, & Joshi, 2017), leading to poorer outcomes. Researchers have even found increased rates of undiagnosed dyslexia among incarcerated populations (Moody et al., 2000; Shaywitz, 2003). Left undiagnosed, misdiagnosed, or untreated, dyslexia leads to significant problems.

Improvements to the diagnostic process could provide the first step to assist children with dyslexia. Accurate diagnosis that is both diagnostically sensitive and specific could lead to earlier remedial efforts and better outcomes. However, the current debate over dyslexia's origin perpetuates problems with misdiagnosis.

Origins of Dyslexia

A schism among dyslexia researchers in the mid-20th century resulted in competing theories – language-based and visually-based – to explain the origin of

dyslexia. The most recent approach to dyslexia is language-based. In this theory, dyslexia originates from problems with *phonological processing*, the awareness of the sounds of our language, the ability to store representations of those sounds, and the ability to retrieve those sounds quickly and accurately (Wagner & Torgesen, 1987). Researched extensively, the language-based description is widely accepted, in part because of technology advances in the late-20th century (Elliott & Grigorenko, 2014). Brain imaging of dyslexics confirmed activation and structural differences in a portion of the parietal lobe adjacent to the temporal lobe (i.e., supramarginal gyrus) where letters and sounds are linked for decoding and in a section of the medial temporal-occipital gyrus (i.e., where the middle temporal gyrus and occipital gyrus meet) important to word recognition (Goswami, 2006; Richlan, Kronbichler, & Wimmer, 2013; Shaywitz, 2003; Strauss, 2010). Imaging studies support the widely-accepted *phonological core deficit hypothesis*, which suggests that most reading problems related to automatic word recognition (i.e., dyslexia) can be traced back to difficulties with the phonological components of language (Pugh et al., 2013; Stanovich, 1998).

Despite a preponderance of evidence to support the phonological view, some researchers continue to advocate for the visual approach to dyslexia. In this theory, letter and word difficulties in dyslexia are thought to originate in the *magnocellular pathway* of area V5 (i.e., visual area 5) in the visual cortex (Cornelissen et al., 1995; Livingstone, et al., 1991; Stein, 2001; Stein, 2018a; Stein & Walsh, 1997; Vidyasagar & Pammer, 2009). This area processes visual motion, location, and spatial analysis. During a reading task, the magnocellular pathway assists the reader when moving sequentially across letters and words. The magnocellular theory suggests that weakened magnocellular cells result in a

faulty attentional spotlight (i.e., mechanism used in visual attention), which causes imprecise timing movements across visual symbols (Vidyasagar & Pammer, 2009). The imprecise movements are hypothesized to cause difficulties with letter sequencing, which results in poor reading outcomes (Stein, 2018a).

Tackling the issue of competing theories has resulted in decades of research to defend and discredit both viewpoints, all hoping to improve the specificity and sensitivity of dyslexia diagnosis by identifying a diagnostic sign or a biological cause. Though most researchers ascribe to the phonologically-based theory (Elliot & Grigorenko, 2014; Vellutino et al., 2004), opponents continue to suggest that differences in the magnocellular pathway cause some readers to develop dyslexia (Stein, 2018a). Yet, a critical analysis of the magnocellular argument reveals a significant and often overlooked problem – magnocellular function in dyslexics is repeatedly evaluated in the context of studies that lack methodological rigor.

Methodological Quality and Literacy Maturity

The measurable and replicable difference in magnocellular function found between dyslexics and controls (see Benassi, Simonelli, Giovagnoli, & Bolzani, 2010) has led many to believe that dyslexics have *deviant* magnocellular function that alters the ability to read and spell like same-age peers. Deviant growth follows an alternate path independent of the typical developmental path at any age with the gap between typical and deviant widening over time (Voigt, Barbaresi, Weaver, & Katusic, 2006). Deviant growth can be contrasted to *delayed* growth, which follows the typical path of development albeit at a slowed pace yet improves toward the typical level (see Figure 1).

If magnocellular development is simply delayed in dyslexics, performance would follow the typical path at an earlier point of development (i.e., poor performance of dyslexics would be similar to younger children on a typical developmental trajectory). If magnocellular development is truly deviant in dyslexics, performance would be independent of peers at earlier points of development (i.e., poor performance of dyslexics would not be similar to younger children on a typical trajectory).

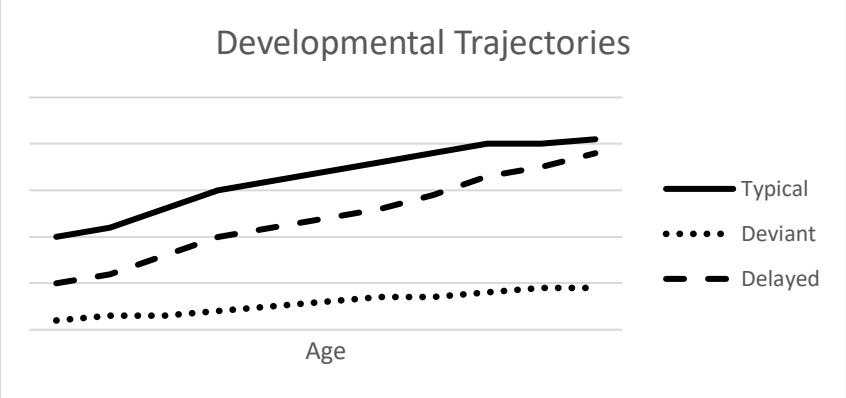

Figure 1. Developmental Trajectories of Deviance and Delay

Deviance in magnocellular function, coupled with symptoms of poor reading and/or spelling, could serve as a diagnostic sign to directly indicate dyslexia. Though the idea of deviance in magnocellular function to improve the diagnosis of dyslexia is enticing, the comparisons used to argue in favor of deviance lack the necessary rigor to become widely accepted.

Numerous correlational and group comparison studies support a difference in magnocellular function between dyslexics and same-age controls (see Benassi et al., 2010). However, using same-age controls in the comparison neglects the developmental trajectory of magnocellular function and the impact that learning to read could have on the visual cortex. Researchers have demonstrated that magnocellular development does not peak until around age 13 (Boden & Giaschi, 2007; Crewther, Crewther, Klistorner, &

Kiely, 1999; Goswami, 2003), suggesting a course of development through the school-age years. Children progress through many developmental milestones that impact the synaptic pruning (i.e., efficiency process), maturity, and precision of the visual cortex during the school-age years. Developmental milestones such as learning to read could confound the comparison of magnocellular function in dyslexics and same-age peers because dyslexics do not reach similar levels of reading development as same-age peers.

Though the vast majority of group comparisons do not consider how learning to read could impact magnocellular ability, Olulade, Napoliello, and Eden (2013) hypothesized that the act of learning to read may facilitate magnocellular development because reading requires attention to visual symbols while the eyes are in motion. These researchers theorized that reading may enable and enhance magnocellular development, implying that a dyslexic may have reduced magnocellular function because weaker reading skills could not bootstrap the maturity of the magnocellular pathway. Other researchers have shown that reading impacts the maturity and activation of the visual cortex (Carreiras et al., 2009; Szwed, Ventura, Querido, Cohen, & Dehaene, 2012), strengthening Olulade and colleagues' theory (2013).

When testing their theory in a group-comparison design, Olulade et al. (2013) found that English-speaking dyslexics and controls could not be reliably separated by magnocellular function when the groups were matched on reading levels instead of age. That is, the hypothesized deviance in magnocellular function disappeared when the researchers accounted for the impact of learning to read. Instead of deviant development, magnocellular function appeared delayed, or like younger controls at similar reading levels. If magnocellular function was truly deviant, dyslexics' magnocellular

performance should have been on a path independent from typically-developing, younger peers. Olulade and colleagues called for additional studies to explore the role of learning to read and magnocellular development. As most group comparisons do not account for learning to read, we do not fully understand the magnocellular function of individuals with dyslexia at this time.

Purpose and Significance

Clarifying the role of magnocellular function is germane to improving dyslexia diagnosis. Deviant magnocellular function that is independent of literacy development could provide a diagnostic sign of dyslexia that could be assessed prior to literacy instruction, leading to an earlier and more accurate diagnosis. A majority of researchers assert that dyslexics have deviant magnocellular function that serves as an important sign of dyslexia. However, their designs neglect *literacy maturity* as a possible confounding variable. Literacy maturity is the ability to automatically group, or consolidate, letters and sounds into meaningful units so reading becomes effortless and automatic.

A minority group of researchers have explored magnocellular function while accounting for literacy maturity (Cornelissen et al., 1998b; Gori et al., 2016; Joo et al., 2017; Olulade et al., 2016; Talcott et al., 2013; Taylor et al., 2018). Yet their data are not conclusive and come with limitations, including novel measures for magnocellular function and sampling procedures that do not target dyslexics. Goswami (2015), a prominent researcher who studies the neuroscience of dyslexia, has called for additional studies that consider literacy maturity as an important confounding variable in models of magnocellular function. Vidyasagar and Pammer (2010) also called for a clearer understanding of the reciprocal role of reading and magnocellular function.

In this study, I used an observational, group comparative design to explore the usefulness of magnocellular function as a diagnostic sign of dyslexia. Comparisons of magnocellular function between children with dyslexia and peers at similar ages and similar levels of literacy maturity attempted to clarify the deviance versus delay debate of magnocellular function in dyslexics. As this line of research is relatively unexplored, I draw from several bodies of literature to assemble a conceptual model of magnocellular function and literacy maturity in dyslexia.

Key Terms

Deviant development: a unique, atypical path of development; does not progress along the typical phases of development; gap between typical and deviant widens over time

Delayed development: a slowed path of development; parallel to typical development but at a different rate; progresses along the typical phases of development at a slower pace yet improves toward the typical level

Diagnostic sign: the presence of a distinguishing characteristic to pinpoint a diagnosis

Diagnostic sensitivity: the ability to accurately identify an individual with a disorder

Diagnostic specificity: the ability to accurately identify those without the disorder

Dyslexia: a neurobiological difficulty with accurate and quick decoding and/or encoding in individuals without other major cognitive difficulties; dyslexia can cause slowed fluency and secondary comprehension problems (Lyon, Shaywitz, & Shaywitz, 2003)

Grapheme: visual symbols (i.e., letters) that represents sound (i.e., phonemes)

Literacy maturity: the ability to automatically group, or consolidate, letters and sounds into meaningful units so reading becomes effortless and automatic

Magnocellular pathway: an area in the visual cortex found across region V5/MT that processes visual motion; it is required to move quickly across letters and words

Phoneme: the smallest unit of sound in language

Phonological Deficit Hypothesis: suggests that most reading problems related to automatic word recognition can be traced back to phonology (Stanovich, 1998)

Phonological processing: the awareness of the sounds of language, the ability to store representations of those sounds, and the ability to retrieve those sounds quickly and accurately.

CHAPTER II

REVIEW OF THE LITERATURE

The following chapter begins with an overview of the major competing dyslexia definitions and the diagnostic problems associated with the current definition. Then I provide a foundational view of reading and explain what goes awry in dyslexia. Next, I offer an extensive review of the empirical literature on the magnocellular theory of dyslexia related to this dissertation. After, I outline the methodological limitations of this current body of research. Following this, I detail a conceptual framework to combine the major foundational theories of learning to read with dyslexia. Finally, I discuss the gaps in the relevant literature and the research questions to be addressed in this dissertation.

Dyslexia Definitions

Visual Definitions

As a construct first described over 100 years ago, the definition of dyslexia has evolved over time. In the early-20th century, ophthalmologists diagnosed unexpected word-reading problems as *word blindness*, a congenital disorder that caused difficulty storing visual representations of words (Hinshelwood, 1902; 1917). Symptoms included an inability to read words, the absence of visual blindness, and normal intellect. As many with word blindness also reported letter reversals when reading, the definition soon expanded to include *strephosymbolia*, or mixed up letters (Orton, 1925). Letter reversals

were thought to be caused by cerebral dominance difficulty, where the brain stores competing copies of visual images when reading (Orton, 1925).

Around the time that word blindness was introduced, the German physician Rudolf Berlin used the term *dyslexia* to describe stroke patients who had lost some reading ability yet retained oral language ability (Berlin, 1887). After hearing of Berlin's new term dyslexia, Pringle Morgan argued for the term to also apply to children with unexpected reading difficulties seen in word blindness (Morgan, 1896). Morgan combined ideas from Hinshelwood, Orton, and Berlin, suggesting that dyslexia was a childhood congenital disorder that caused difficulties with visual representations of words despite average reasoning and intelligence.

Phonological Definition

Dyslexia remained a visual word-reading problem until the seminal work on *phonology* – the sound system of language – and learning to read (Chomsky, 1970; Lieberman, 1970; Lieberman, Meskill, Chatillon, & Schupack, 1985; Lieberman & Shankweiler, 1985; Mattingly, 1972; Wagner & Torgesen, 1987). Linguists and psychologists posited that reading depended on the ability to bring spoken language to the level of explicit awareness. That is, emerging readers must learn to manipulate and separate units of language into constituent sounds so they can later map those sounds onto printed letters. Most children develop this awareness with little instruction and their sound-to-letter matches become reciprocally reinforced during practice (Wagner & Torgesen, 1987).

Proponents of the phonological view argue that children with dyslexia fail to make secure sound-letter matches because of deficits in *phonological processing* – the

awareness of the sounds of our language, the ability to store representations of those sounds, and the ability to retrieve those sounds quickly and accurately (Wagner & Torgesen, 1987). Interestingly, the symptoms described in strephosymbolia did not disappear with the move toward a phonological definition. Individuals with dyslexia continued to report visual difficulties including letter crowding, letter reversals, and difficulty with moving letters. Advocates for the phonological definition attributed the visual problems to inefficient and incomplete sound-letter matches because of phonological weakness (see Vellutino et al., 2004).

Current Definition

The most widely-accepted definition of dyslexia was the result of a multidisciplinary effort in the early-2000s. Prominent researchers in medicine, neuroscience, psychology, and education, along with The International Dyslexia Association (IDA) and National Institute of Child Health and Human Development (NICHD) published a definition of dyslexia still used today:

> 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. (Lyon, Shaywitz, & Shaywitz, 2003, p. 2)

This definition highlights the word recognition difficulties first described over a century ago. However, the new definition expands specific symptoms of dyslexia to include difficulties with decoding, spelling, and fluent, or automatic and prosodic, word recognition. In addition, the added term "neurobiological" pinpoints the source of the reading problem as the brain and reflects a boom in imaging research to map the reading brain. The new definition also points to phonological deficits as the cause of the reading problem, implying that dyslexia is not due to visual dysfunction.

Problems with Diagnosis Under the Current Definition

The current IDA and NICHD definition of dyslexia is widely cited; however, debates over symptomology and phonology remain relevant to dyslexia diagnosis. Dyslexia can manifest with one or a number of symptoms listed in the definition (e.g., word reading, spelling, decoding, fluency, comprehension) and not all dyslexics will present with the same symptoms (Hoskyn & Swanson, 2000; Vellutino et al., 2004). Further, clear cut-off points for what constitutes true dyslexia in any of the symptoms have yet to be established (Elliott & Grigorenko, 2014). A clear picture of dyslexia's symptomology and what scores qualify for true dyslexia remain unknown at this time.

Debates over phonology also reduce the sensitivity and specificity of dyslexia diagnosis. Most researchers support the collective IDA and NICHD definition of dyslexia, but the inclusion of phonology is not without dispute. Some researchers cite empirical evidence that not every dyslexic has phonological difficulties (Frederickson & Frith, 1998; White et al., 2006). Other researchers acknowledge that phonological difficulties exist in some dyslexics but point to problems with the visual system that cannot be explained by phonological weakness (see Stein, 2018a). In fact, the IDA and

NICHD definition captures this problem. The word "typically" in the phrase "difficulties typically result from a deficit in the phonological component" (Lyon, Shaywitz, & Shaywitz, 2003, p. 2) suggests that not every dyslexic will have problems with phonology. This phrase also implies that the biological system implicated in dyslexia is not always phonological processing, yet the definition lacks alternate causal explanations.

Diagnosis depends on a clear understanding of the relevant construct to be measured. Unfortunately, our current understanding of dyslexia often lacks the diagnostic power needed to make an accurate diagnosis. In the ongoing search for a diagnostic sign of dyslexia, the biological basis of dyslexia continues to be researched.

Foundational Dyslexia Frameworks

Though diagnosis benefits from an operational definition of dyslexia, associated problems with the clarity of symptoms and biological etiology leave remaining questions. To explore the biological foundations of dyslexia and related presentation of symptoms, two perspectives of dyslexia's theorized etiology will be presented. First, a linguistic-based framework will be presented, followed by a related theory of phonological dysfunction in dyslexia. Then, a visually-based framework will be presented, including a related theory of visual dysfunction in dyslexia.

The Reading System: Four Language Processors

Adams (1990, 2013) offers a prominent model of linguistic reading processes in her Reading System model. Seen in Figure 2, the Reading System is comprised of four major processors, three of which are closely connected. The phonological processor is responsible for attending to and manipulating the sounds of language, including the pronunciations of words. The orthographic processor is responsible for the visual images

and visual patterns of letter sequences within words. The meaning processor is responsible for the meanings of words and is influenced by the context processor, which is responsible for constructing a coherent interpretation of the text.

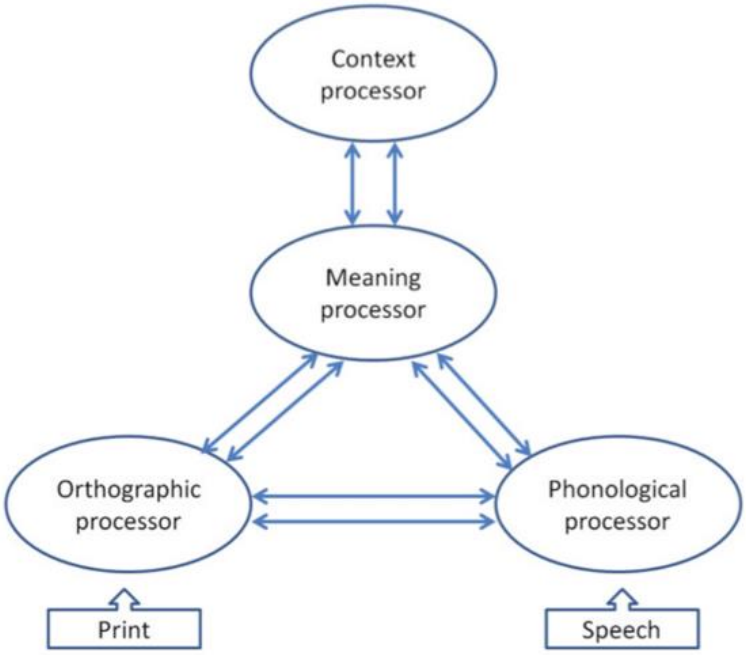

Figure 2. Adams's Model of the Reading System: Four Processors. Based on Adams (2013).

The Reading System receives information to all processors simultaneously during efficient reading and includes feedforward and feedback loops, making the model neither top-down nor bottom-up. Relevant to this dissertation are the connections among the orthographic and phonological processors. As words are encountered in print (i.e., via the orthographic processor) that are familiar in speech (i.e., via the phonological processor), the connections between the sounds and symbols become strengthened. The arrows seen in Figure 2 represent the two-way connections that must develop with practice and instruction. "Ultimately it is these bonds, these interrelations – as they pass excitation and inhibition among the elements that they link together – that are responsible for the

fluency of the reader" (Adams, 2013, p. 789). As the following discussions will focus on the phonological and orthographic aspects of reading, a closer examination of Adams's (1990, 2013) phonological and orthographic processors is warranted.

Phonological processor. The phonological processor fills two roles in the Reading System: linchpin from print to meaning and aid to verbal memory (Adams, 1990, 2013). First, the phonological processor acts as the linchpin between a child's spoken repertoire housed in the meaning processor and the printed word processed by the orthographic processor. Printed visual symbols themselves are meaningless until the child applies enough phonological information to the symbols that a recognized word comes to mind. Put another way, children see letters they recognize, begin sounding out the word, and realize the word is familiar from their spoken vocabularies. Without the phonological processor, there is no link between the unknown printed words on the page and the known meanings of spoken words. Even when young readers become more proficient and read words automatically, the phonological processor continues to work. That is, even if a word is recognized immediately at first sight (Ehri, 2017) and does not need to be sounded out, simply seeing the word will still activate phonological representations (Perfetti, Bell, & Delaney, 1988; VanOrden, 1991) in order to access meaning. Therefore, at no point in the reading process can the phonological processor be shut off or outgrown.

Not only does the phonological processor serve as a linchpin to meaning, it also aids in memory during reading. As the access point to language, the phonological processor preserves the spoken words, even if read silently, as readers progress along a line of text. In other words, the auditory system encodes temporal patterns of speech and rhythm as readers progress along a line of text. Without this encoding, readers would

have to rely on memory of the spatial patterns of letters along the entire line of print. Assigning a spoken label to the visual pattern enables access to meaning faster, allowing for longevity of the words read along the line of text.

In sum, the phonological processor supports early attempts to connect print to meaning, acts as the inaudible foundation for automatic translation of print directly to meaning, and aids in auditory verbal memory during reading.

Anatomy. Although Adams (1990, 2013) presents the phonological processor as one unit or module, phonological information is processed across the brain. Speech sounds are received by the auditory cortex in the temporal lobe and translated to phonetic units that carry meaning in Wernicke's area, found in the left superior temporal gyrus (Eagleman & Downer, 2016; Kolb & Wishaw, 2015). In beginning readers and readers with phonological dysfunction, both the left and right temporal lobes are active during phonological tasks (Pugh et al., 2013). With instruction and practice, readers refine the functional connectivity and primarily use the left temporal lobe during phonological tasks (Pugh et al., 2013). If speech is required during the phonological task (e.g., name a word that rhymes; tell the third sound, etc.), analyzed speech information moves from Wernicke's area to Broca's area (i.e., the left inferior frontal gyrus) via the arcuate fasciculus for response formulation (Eagleman & Downer, 2016; Glezer et al., 2016; Kolb & Wishaw, 2015). Finally, the response is sent from Broca's area to the premotor and motor cortex for the response to be sent to the mouth (Eagleman & Downer, 2016; Kolb & Wishaw, 2015). Thus, the idea of a single module simplifies how phonology is processed. Phonological processing requires many steps to make sense of language sounds.

Orthographic processor. The orthographic processor fills three main roles in the Reading System: receive and analyze information from print by interacting with the visual cortex (Adams, 1990, 2013), facilitate statistical and analogical learning (Steacy, Elleman, & Compton, 2017), and ultimately provide direct access to the pronunciation and meaning of printed words (Ehri, 2017).

Visual interaction with orthography. The orthographic processor translates print to speech, so the first job of this processor is to interact with the visual cortex via saccadic movements and letter processing (Adams, 2013). When looking at a line of text, the eyes do not move smoothly from left to right. Instead, they have quick movements, or saccades, that move the eyes to various fixation points. On average, the eyes can capture information three letters to left of the fixation point and 6 to 10 letters to the right of the fixation point (Adams, 2013). As information comes into the visual cortex and the lines, shapes, and angles are recognized as letters, the orthographic processor begins working at a rapid pace to keep up with the amount of visual information coming into the system. To mitigate the quantity problem, the orthographic processor consolidates units or patterns from the printed text and activates memory for spelling patterns, allowing letter strings to be perceived simultaneously as consolidated units.

Surprisingly, the visual system is remarkably inefficient at processing letter order, even in skilled readers (Adams, 2013). To increase the speed of letter order confirmation, the orthographic processor searches for familiar units among the possible letter combinations. For familiar spellings, the connected letter forms activate each other, "causing all to be recognized nearly at once and to hang together in the reader's mind as a familiar, cohesive spelling pattern" (Adams, 2013, p. 790). For unfamiliar spellings, there

is no familiarity with other letters of the unit, so it takes longer to process each possible combination to sort out the correct order (McClelland & Rumelhart, 1981). In other words, the associations between letters within a spelling pattern allow for faster processing of the word. For example, when reading *seat*, the visual cortex will process every letter, no matter the familiarity of the word. However, the orthographic processor can assist with recognition by signaling combinations of letters that could make a word and related words with similar patterns (e.g., SEAT: *eat, seal, at, seat*).

Adams (1979) tested the letter connection phenomenon with good and poor readers. When asked to report the letter order of regularly spelled nonwords (e.g., *zock, yain*), good readers had nearly perfect performance. Poor readers, on the other hand, had lower accuracy because they were unable to apply spelling patterns to retain letter order. When the same readers were tested on irregularly spelled nonwords (e.g., *phqi, bgyl*), good and poor readers had similar levels of difficulty. This study illustrates the power of the orthographic processor to assist in the visual analysis of words.

Statistical and analogical learning. The second job of the orthographic processor is to facilitate statistical and analogical learning (Steacy et al., 2017; Steacy, Compton, et al., 2019). It would be impossible for readers to learn every word they might encounter in texts across a lifetime. Beginning readers need a way to multiply their efforts in word learning and apply known patterns to unknown words. Statistical learning enables readers to use the interconnections of letters, or sub-word orthographic-phonological connections (Adams, 2013; Perfetti, 1992) of quasi-regular orthographies – the spelling system in languages without direct one-to-one matching of phonemes and graphemes – to read words with variable sound-to-symbol correspondences (Steacy et al., 2019). In other

words, the orthographic processor can analyze the variable spelling patterns found in English to suggest the most likely symbol-sound correspondences. Statistical learning develops at various levels of the orthography, including how letter clusters relate to phonemes (e.g., *ch-, dr-*), how letter clusters relate to rimes (e.g., *-ate, -ick*), and how letter clusters relate to syllables (e.g., *ra-don, rap-ture*). As units increase, readers acquire "overlearned knowledge about likely and unlikely sequences of letters" and their sounds (Steacy et al., 2017, p. 792), hastening statistical inductive word learning.

For example, the letter *i* has similar visual properties in the words *wild* and *sink.* The vowel follows one consonant, the vowel is the second phoneme of each word, and the vowel is followed by a two-consonant coda (e.g., *ld* and *nk*). The difference between *wild* and *sink* is the sound of the vowel, which could lead many inexperienced readers to read *sink* as /saɪnk/ or *wild* ad /wɪld/. The orthographic processor can apply the statistical properties of English to recognize the pattern *ild* most often occurs as a long-vowel pattern (e.g., *child, mild*) and *ink* most often occurs as a short-vowel pattern influenced by the nasal *-nk* (e.g., *drink, think, mink, link*).

As the orthographic processor analyzes words, even inductively, greater statistical refinement occurs (Steacy et al., 2017; Steacy, Compton, et al., 2019). A sophisticated orthographic processor is familiar with the statistical properties of English and stores letter patterns at a level of explicit awareness. That is, the *-ild* and *-ink* rimes become consolidated patterns in the orthographic lexicon (i.e., dictionary of the mind) so that recognition in print becomes faster (Ehri, 2005). Not only does pattern recognition speed up, it also allows for analogical learning, or transfer to novel words (Steacy et al., 2017). Readers use the orthographic processor to apply knowledge of spelling patterns learned

explicitly and through statistical learning to similar words not part of an instructional routine (i.e., readers use *wild* to read *mild*). Analogical reasoning adds words to automatic reading vocabularies and enhances the efficiency of the Reading System.

Direct access to words. The final role of the orthographic processor is to provide direct access to the pronunciation and meaning of printed words (Ehri, 2017). Statistical and analogical learning facilitate early attempts at direct access, but the orthographic processor will continue to consolidate orthographic patterns until the entire word becomes stored as a single unit (Ehri, 2017). That is, the mere sight of the word will automatically activate the sound of the entire word and its meanings (Ehri, 2005, 2017). Word reading automaticity is paramount so working memory capacity is not overwhelmed during decoding attempts, leaving adequate cognitive capacity for constructing meaning (Perfetti & Lesgold, 1979). Without an efficient orthographic processor, readers would be left translating every word's visual symbols into partial sound units until a recognizable word came to mind.

Anatomy. Similar to the phonological processor, Adams (1990, 2013) presents the orthographic processor as one biological module. Yet orthographic processing is the result of multiple areas of the brain working together. Starting with the words on the page, orthographic processing begins in the visual cortex of the occipital lobe. The lines, edges, and angles of letters become more sophisticated as they move up through the visual cortex. After leaving the visual cortex, information moves to the left angular gyrus, which analyzes visual signals and maps those signals onto language and meaning units (Horwitz, Rumsey, & Donohue, 1998; Kearns, Hancock, Hoeft, Pugh, & Frost, 2019; Kolb & Wishaw, 2015). The angular gyrus sends information on to Wernicke's area,

which processes sounds and meanings associated with visual forms; the angular gyrus also sends information to the supramarginal gyrus, which links letters and speech sounds (Kearns et al., 2019).

Information moves from Wernicke's area to the occipito-temporal region for word recognition; this area is known as the Visual Word Form Area (VWFA; Kolb & Wishaw, 2015). The VWFA is specialized for analyzing abstract patterns or objects that become recognition units after multiple encounters (Goswami, 2006; Kronbichler et al., 2003; Richlan, Kronbichler, & Wimmer, 2013; Shaywitz, 2003; Strauss, 2010). In other words, the VWFA allows for automaticity as orthographic units are stored at larger sizes and whole words are stored as unique visual entries. Though most activity during orthographic processing occurs in the left hemisphere, the right inferior parietal lobe and prefrontal networks assist with spatial analysis of the visual forms and attention to the forms across multiple lines of text (Kronbichler et al., 2003; Pugh et al., 2013; Shaywitz, 2003). As orthographic processing requires the linking of letter sounds and speech of the printed word, information is eventually sent forward to Broca's area for speech sequencing and then ahead to the precentral gyrus for articulation (Kearns et al., 2019).

In sum, the orthographic processor is essential to reading as it facilitates the only means of interaction between the reader and the text. It also enables readers to transfer word patterns and units to new words via statistical and analogical learning and provides direct access to whole word pronunciations and meaning by consolidating orthographic units into whole words.

Specific Dyslexia deficits across the Reading System. Although Adams' reading system is theoretically present in those with dyslexia, deficits in the phonological

processor, orthographic processor, and the connection between the two are theorized to cause problems in readers with dyslexia (see Figure 3).

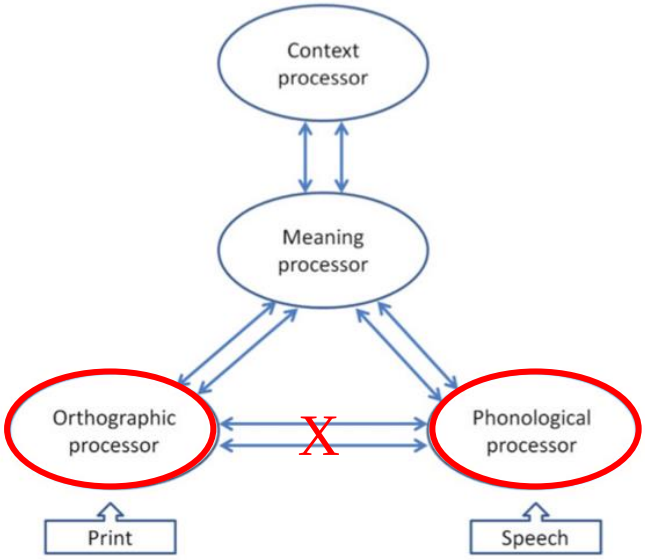

Figure 3. Adams's Model of the Reading System and Disruptions in Dyslexia. Based on Adams (2013).

Problems with the phonological processor in dyslexia are believed to weaken the ability to translate language to meaningful sounds and prevent the phonological processor from aiding in verbal memory (i.e., remembering individual sounds or sound units during analysis; Wagner & Torgesen, 1987). When individuals experience difficulty with multiple aspects of phonological processing, a more severe form of dyslexia emerges called Double Deficit Dyslexia (Wolf & Bowers, 1999).

In the orthographic processor, individuals with dyslexia fail to translate and secure word patterns, which causes labored decoding and spelling attempts (Adams, 2013; Ehri, 2017; Shaywitz, 2003). Steacy et al. (2017) explain that the "orthographic processor's magic presumes a deep and ready knowledge of the letterwise spellings of

words" (p. 792). Many with dyslexia never experience this magic because they cannot establish the deep knowledge of orthography that Steacy and colleagues discuss.

The theorized differences in the dyslexic Reading System have been proven with decades of brain imaging studies (see Figure 4). Dyslexics show decreased activation in the angular gyrus, supramarginal gyrus, and VWFA – the hubs for word analysis – during reading-related tasks (Shankweiler, Mencl, Braze, Tabor, Pugh, & Fulbright, 2008). Instead of reliance on the angular gyrus, supramarginal gyrus, and VWFA, dyslexic readers over-rely on Broca's area and over-activate right hemispheric regions, as if they are struggling with speech sounds while searching for novel visual patterns, not automatically recognizing words as complete visual units that have corresponding sounds (Shankweiler et al., 2008). When comparing adolescents with dyslexia to younger peers, both groups appear to have overactivation in Broca's area and the thalamus with under activation in the occipito-temporal VWFA, but dyslexics also have under-activation in parieto-temporal areas (e.g., angular gyrus, supramarginal gyrus, Wernicke's area; Deutsch, Dougherty, Bammer, Siok, Gabrieli, & Wandell, 2005; Hoeft et al., 2007; Linkersdorfer, Lonnemann, Lindberg, Hasselhorn, & Fiebach, 2012; Richlan, Kronbichler, & Wimmer, 2013). Taken together, these findings suggest that children with dyslexia have specific differences in posterior systems used for letter-sound integration and analysis.

Overall, the Reading Systems model suggests that inefficient and inaccurate representations from the orthographic and phonological processors coupled with disturbances in the communication between each processor are thought to cause decoding and spelling difficulties seen in dyslexia.

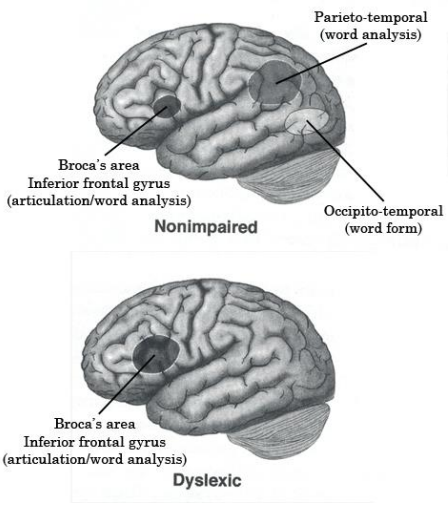

Figure 4. Brain Regions Associated with Reading and Dyslexia. Adapted from *Overcoming Dyslexia*, by Shaywitz, S., 2003, pp. 78-83, New York: Knopf.

Phonological Deficit Hypothesis. The most widely-accepted theory of dyslexia's origin that aligns with the Reading Systems model is Stanovich's (1988) Phonological Core Variable Model (also known as the Phonological Deficit Hypothesis). Stanovich offered a series of empirical studies (see Elliott & Grigorenko, 2014, for a review) to establish the Phonological Deficit Hypothesis, which suggests that all children – dyslexic or not – who struggle with word reading have core deficits in phonological processing. The original theory also specified that the variable differences between dyslexics and other poor readers were in the cognitive abilities outside the phonological, with dyslexics having fewer additional cognitive complexities than other poor readers (Stanovich, 1988).

Stanovich (1996) later revised the Phonological Deficit Hypothesis to reflect empirical evidence that some dyslexics showed co-occurring cognitive deficits as well (e.g., low-level visual difficulties, poor memory, imprecise auditory timing). That is, the Phonological Deficit Hypothesis names phonological processing as the main problem in dyslexia but allows for many other cognitive deficits as well. Unfortunately, Stanovich did not explain how or why so many other cognitive difficulties were present in some, but

not all of, the dyslexic population. Stanovich's theory also failed to specifically name the orthographic processor, though the author implied that difficulties with the phonological aspects of language could cause concomitant difficulties with translating and finding patterns in print. However, a specific deficit in orthographic processing was not part of the original Phonological Deficit Hypothesis.

Further, some opponents of the phonological view of dyslexia have demonstrated that some dyslexics do not face phonological difficulties (Frederickson & Frith, 1998; White et al., 2006). Other opponents have proven that individuals with dyslexia have underlying visual and auditory deficits that cause reading difficulties not explained by the phonological model (see Stein, 2018a). In sum, the Phonological Deficit Hypothesis describes a major aspect of dyslexia but does little to explain any common underlying biological cause for the spread of cognitive difficulties seen in dyslexia and omits the role of the orthographic processor.

Despite the theory's lack of precision, its impact was widespread and continues over 30 years later. In fact, the core phonological difference cited in Stanovich's (1996) theory was cited in the most recent NICHD definition of dyslexia and was adopted by the International Dyslexia Association (Lyon et al., 2003). In their comprehensive review of dyslexia, Elliott and Grigorenko (2014) stated that Stanovich convinced many that phonological deficits were to blame in dyslexia despite significant problems with the precision of the theory for understanding what dyslexia is and what dyslexia is not.

Magnocellular Processing

The major competing model and theory of dyslexia's origin outside of phonological processing lies in magnocellular processing. First, a brief overview and

model of the visual cortex will be offered. Following, the magnocellular theory of dyslexia will be presented. Empirical literature relevant to this dissertation will be discussed, including a critical analysis of the methodological design of most studies that test the magnocellular theory of dyslexia.

Hierarchy of visual processing. The visual cortex has a hierarchical structure with multiple feedback loops (Eagleman & Downer, 2016; Kolb & Whishaw, 2015). A simplistic overview of the hierarchy can be found in Figure 5. First, data from the visual world travels through the eye and back to the retina where retinal ganglion cells pass information on to the brain. The signals from the retinal ganglion cells are sent along the optic nerve to the lateral geniculate nucleus (LGN) in the thalamus. The thalamus works as a relay-station of sorts for various input (e.g., sensory, motor, emotional, hormonal) and sends information to the cerebral cortex, or higher-intellectual processing areas of the brain. Two major visual signals come into the LGN – parvocellular cells and magnocellular cells. Parvocellular cells (p-cells) are smaller cells that receive their input from cones in the retina and are specialized for color. Magnocellular cells (m-cells) are larger and more numerous and receive their input from rods in the retina specialized for contrast, not color. The LGN processes p-cells and m-cells separately and sends their signals on to the primary visual cortex (V1) of the occipital lobe.

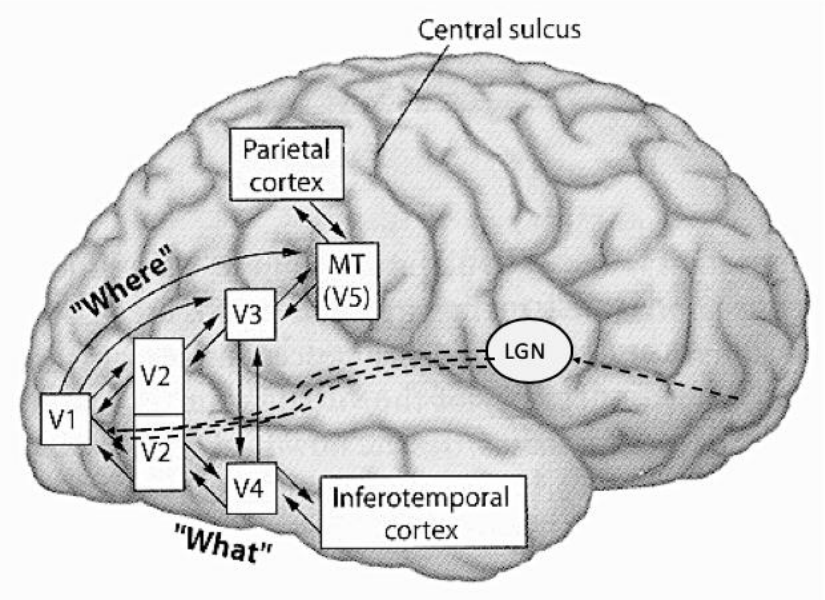

Figure 5. Overview of the Visual Cortex. Adapted from *Brain and behavior: A cognitive neuroscience perspective*, Eagleman, D. & Downer, J., 2016, New York: Oxford University Press.

V1 processes simple visual information (e.g., edges, degrees of tilt) and combines input from both eyes to construct a basic understanding of the visual field. Neurons in V1 send many smaller constructions (e.g., edges formed to a line) to the secondary visual cortex (V2) for further analysis. V1 also sends projections to other visual areas (e.g., visual areas 3, 4, 5). As the visual cortex operates as a hierarchy, neurons in V2 are more sophisticated than V1 and can construct a larger and more complex rendering of the stimuli (e.g., simple color, orientation, curves, angles, and borders). The hierarchical structure also includes feedback loops. Information sent forward to higher levels of the hierarchy is continuously sent back to lower levels to reinforce and recalibrate the perception of the outside visual world.

After V2, the visual cortex splits into two main pathways – the dorsal, "where" pathway, and the ventral, "what" pathway. The dorsal pathway establishes where objects are located and assists with the visual guidance of movements; this pathway is sometimes

called the magnocellular pathway (m-path) because of the large number of magnocellular cells that make up this path. Seen in Figure 5, the m-path travels from V1 to V2, V3, and then V5 in the medial-temporal (MT) area, which sends projections to the parietal cortex for further analysis. The ventral pathway assists with recognizing objects; this pathway is sometimes called the parvocellular pathway (p-path). This path travels from V1 to V2 to V4 near the inferior-temporal cortex (see Figure 5). There is a third pathway that travels along the superior temporal sulcus (STS) specialized for biological motion and facial and eye analysis, but the STS path is not relevant to this study.

Magnocellular theory of dyslexia. The magnocellular theory of dyslexia (m-theory) states that weak m-cells are the biological cause of the word-reading, spelling, and fluency difficulties seen in dyslexia (Stein, 2001, 2018a, 2018b, 2019; Stein & Walsh, 1997). Advocates of the m-theory argue that m-cell function in dyslexics is deviant from that of normal readers of the same age. Deviant performance is interpreted as a unique deficit to dyslexia that could potentially serve as a diagnostic sign during diagnosis. However, there are opponents to the m-theory and a significant flaw in the methodological design of many studies used to argue for the m-theory.

The following sections will begin with an overview of m-cell function and deficits specific to dyslexia. Then a brief yet relevant aside will acknowledge auditory "magnocellular" cells. Following, I will tie m-cell dysfunction to literacy performance. Next, the counterarguments to the m-theory will be presented, followed by a detailed review of the literature on motion detection and dyslexia, including a major flaw in their designs.

Visual m-cell function. Visual m-cells are important for visual temporal processing – the accurate timing and sequencing of visual stimuli coming into the visual cortex (Stein, 2001, 2018a, 2018b, 2019; Stein & Walsh, 1997). M-cells respond to rapid changes visual input, making them ideal for visual timing and sequencing. Temporal processing harnesses visual attention to boost visual memory for a target stimulus. Put another way, visual memory depends on the visual cortex's ability to focus and sequence incoming stimuli. Efficient m-cells enable precision of the *attentional spotlight*, or specific area of visual focus among distractors (Vidyasagar & Pammer, 2009; Vidyasagar 2019). The attentional spotlight is needed to make sense of the visual world in motion with rapidly incoming stimuli.

Visual m-cell dysfunction. Researchers have found unstable m-cell function in dyslexics at various levels of the brain from the retina to the cortical magnocellular pathway. Retinal m-cells are thought to suppress the noise created by the eye's constant saccadic shifting (i.e., the eyes are constantly making mini-movements and taking in large amounts of visual input). Without efficient suppression, we would perceive the world as a set of constant mini-shifts of our visual world. M-cells adapt to the constant shifting and reciprocally provide information about location and movement of objects in our environment, which enables the attentional spotlight. Dyslexic m-cells in the retina are believed to take longer to adapt to the mini-saccades, causing a delayed spotlight of attention on the target stimulus (Stein, 2018b). Impaired retinal m-cells have been found in dyslexics using measures of contrast sensitivity, or the ability to distinguish frequencies of moving white and black lines (Pammer & Wheatley, 2001).

Beyond the retina, m-cell signals to the cranial nerves are important for the control of eye movements. Specifically, m-cell signals to cranial nerves 3 (i.e., oculomotor), 4 (i.e., trochlear), and 6 (i.e., abducens) adjust and direct eye movement in response to visual motion (Kolb & Wishaw, 2015). Janita and Kapoula (2011) showed dyslexics had lower accuracy during the fixation phase of eye movements compared to normal peers. The LGN has also been implicated as a source of m-cell abnormality in dyslexics. Livingstone, Rosen, Drislane, and Galaburda (1991) found dyslexics had 30% fewer m-cells in the LGN, and Giraldo-Chica, Hegarty, and Schneider (2015) uncovered thinning of the LGN layers in dyslexic participants. Moving up into the cortex, aberrant m-cells in V1 have been found in dyslexics. M-cells in V1 help create central fixation points when viewing movement. Talcott, Hansen, Willis-Owen, McKinnell, Richardson, and Stein (1998) found that dyslexics take longer to create the central fusion point, suggesting damaged m-cells in V1.

Of particular interest to this study, researchers have also found m-cell disturbances in the magnocellular pathway of area V5/MT, which is saturated with m-cells. Eden, VanMeter, Rumsey, Maisog, Woods, and Zeffiro (1996) used functional imaging to show that dyslexics have reduced activity in V5/MT during tasks of visual attention and visually guided movements. Dyslexics are also less sensitive to coherent motion detection, the primary clinical measure of V5/MT function (see Benassi et al., 2010, for a review). Finally, m-cell abnormality is hypothesized to reduce the accuracy and efficiency of the posterior attentional system that controls serial visual search of targets in an array. Vidyasagar and Pammer (1999) found that dyslexics are slower at

serial visual search tasks and suggested that abnormal or inefficient m-cells projecting to the posterior attentional system can cause delays in visual scanning.

Taken together, reduced visual m-cell function from the retina to cortex has been demonstrated in dyslexic populations. Advocates for the m-theory of dyslexia point to these findings as support. However, it is important to include a short discussion of auditory m-cells to distinguish visual theories from less popular theories involving the auditory cortex.

Auditory m-cell function. A minority group of researchers have investigated cells in the auditory cortex hierarchy and their contributions to language analysis. Similar to the visual m-cells, auditory transient cells are hypothesized to impact temporal processing. A brief discussion of these cells is followed by the major theory related to dyslexia. When spoken language is heard, it is not heard as individual words or series of individual phonemes (e.g., *cat* is not heard as /kæt/ or /k/-/æ/-/t/). In reality, language is spoken and heard as co-articulated speech sounds (i.e., the sound signals are influenced by neighboring sounds). The co-articulated sounds are sent to the cochlea where they are translated into bands of frequency signals called formants, which signal the "sound shape" of the co-articulated sound. The formant signals travel from the cochlea through the brainstem and midbrain to the medial geniculate nucleus (MGN; Eagleman & Downar, 2016). These signals are relayed to the primary auditory cortex (A1) and up through the hierarchy of the auditory cortex (e.g., A2, A3) for analysis primarily concentrated in the left temporal lobe and inferior frontal gyrus (Gaab, Gabrieli, Deutsch, Tallal, & Temple, 2007). Specialized auditory transient cells signal formant changes, which are necessary for distinguishing phonemes and determining phonetic sequences.

Similar to visual magnocellular cells, transient auditory cells are large and involved in temporal processing.

Rapid auditory temporal processing has been examined in dyslexia. A small, yet consistent group of researchers argue that difficulties with various aspects of rapid auditory sequencing contribute to the phonological problems seen in dyslexics (Lorusso, Cantiani, & Molteni, 2014; Tallal, 1980; 2004; Gaab et al., 2007; also see Elliot & Grigorenko, 2014 for a review), but their evidence is not yet conclusive. A major criticism of this line of research is that not all readers who struggle with phonological processing have deficits in rapid auditory processing (Ramus et al., 2003; Tallal, 2004). However, brain imaging documented a measurable difference in dyslexic rapid auditory processing (Gaab et al., 2007), and a study of infants documented reduced responses to rapid auditory stimuli for babies with a family history of dyslexia (Benasich & Tallal, 2002). Though highly criticized for not applying to all children with dyslexia, it is important to consider that magnocellular abnormalities have been documented in the visual and auditory systems in dyslexics. However, for the remainder of this study, *m-cells* will refer to visual m-cells unless otherwise noted.

Visual m-cells and literacy. Researchers have demonstrated that dyslexics have impaired function on basic tasks requiring visual m-cells, but an important part of the m-theory describes the connection between compromised m-cells and literacy. M-cells are thought to focus the attentional spotlight on letters for their position and sequencing (Stein, 2001, 2018a, 2018b, 2019; Stein & Walsh, 1997). Stein (2001, 2018a, 2018b, 2019) theorizes that defective m-cells prevent the attentional spotlight from fixating on letters, causing crowding (i.e., letters on top of each other preventing clarity) that is

exacerbated by the serial nature of text reading. He also suggests that inefficient m-cells cause reduced precision during visually guided movements, increasing the timing of letter sequencing. Longer sequencing increases the load on visual memory, which means readers have longer inter-letter pauses during blending attempts of unknown words. Put another way, the longer it takes readers to receive quality input about the visual letter, the longer it takes to arrive at a whole-word attempt. When too much time lapses or too many unspecified signals are received, reading accuracy and speed diminish.

In addition, Stein hypothesizes that immature m-cells and the inability to suppress visual noise during saccadic movements cause letter movements and letters that persist longer than appropriate when moving along letter strings or words, known as the masking effect. Problems with letter sequences suggests ties to the orthographic processor (Adams, 2013); in fact, m-cell activity in V5/MT predicts orthographic knowledge in dyslexics and typical readers (Demb, Boynton, Best, & Heeger, 1998; Witton et al., 1998). In sum, the m-theory suggests that impaired m-cells result in lengthy and imprecise visual temporal judgments that reduce the efficiency and accuracy of readers with dyslexia.

Opposition to the visual m-theory. Despite the numerous empirical studies that support the m-theory of dyslexia, there is strong opposition to the theory. The most glaring critique of the m-theory is that not all dyslexics perform poorly on measures of m-pathway function and that typical readers can perform poorly on the same measures (Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Ramus et al., 2003). Another argument against the m-theory is that dyslexics' poor eye movement is caused by insufficient decoding skill instead of being the cause of the poor decoding skill (see

Rayner, 1998; Hutzler, Kronbichler, Jacobs, & Wimmer, 2006). Counter to this argument, Stein and Fowler (1981) showed that dyslexics have poor eye movements during linguistic and nonlinguistic tasks that require sequential visual processing, arguing that decoding skills would not have any effect on nonlinguistic visual task performance.

The last major criticism of the m-theory is that the m-path does not always respond during phonological tasks that cause problems for dyslexics. Paulesu, Danelli, and Berlingeri (2014) synthesized 53 imaging studies and concluded that dyslexics do not have reduced V5/MT activity during some phonological tasks. If m-cells in V5/MT caused the difficulties seen in dyslexia, then activity in area V5/MT should be reduced during those difficult tasks. M-theorists respond to this potential flaw with the counterargument that not all dyslexics have phonological weaknesses (Frederickson & Frith, 1998; White et al., 2006) and that phonological difficulties cannot explain the visual symptoms many dyslexics report (see Ramus et al., 2003).

Coherent Motion and the Visual M-Theory

The most robust line of research in favor of the m-theory includes studies that measure m-cell coherent motion detection in the magnocellular "where" pathway of the visual cortex. Coherent motion detection refers to the brain's ability to judge a percentage of dots moving in a similar direction among a noisy background. Functional magnetic resonance imaging confirms that random dot kinematograms (RDKs) can reliably measure m-cell ability to detect coherent motion in area V5/MT of the m-pathway (Eden et al., 1996; Olulade et al., 2013). Benassi et al. (2010) stated that RDK measures are the "gold standard for magnocellular processing" (p. 343).

An RDK uses randomly moving white dots against a black background to test the participant's ability to detect coherence in a portion of the moving dots. Almost all researchers use a one or two-panel RDK. One panel RDKs have one viewing area with a percentage of coherently moving dots among distractor dots; participants indicate the direction of the unified movement using keyboard arrows. Seen in Figure 6, two-panel RDKs have two separate yet identical areas on either side of a horizontal or vertical midline. One panel has random motion while the other has coherently moving dots among distractor dots. Instead of indicating the direction of the movement, participants choose the panel that has a percentage of motion coherence. The participant's coherent motion detection threshold refers to the percentage of moving dots needed to detect the subsection of dots moving in the same direction. For example, a threshold of 40% indicates the participant could detect coherent motion when 40% of the dots move in the same direction. Lower thresholds indicate better m-cell function as fewer unified dots are needed to detect coherence. Average m-cell function in typically developing children ranges from 17 to 20% coherence around age 12 to 13 (Cornelissen, 1998b; Hadad, Maurer, & Lewis, 2011; Joo, 2017; Raymond & Sorensen, 1998; Talcott, 2013).

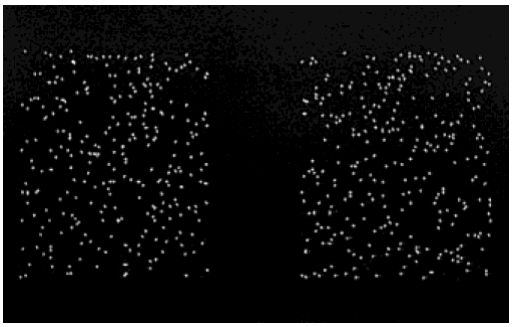

Figure 6. Two-panel Random Dot Kinematogram

The extant literature on coherent motion detection confirms that dyslexics have higher coherent motion thresholds (i.e., poorer m-cell function) than same-age peers (see

Table 1). The majority of findings come from English-speaking dyslexics and same-age controls (Conlon, Sanders, & Wright, 2009; Conlon, Sanders, & Zapart, 2004; Cornelissen et al., 1998a; Cornelissen et al., 1995; Dawes et al., 2009; Eden et al., 1996; Edwards et al., 2004; Everatt, Bradshaw, & Hibbard, 1999; Gibson, Hogben, & Fletcher, 2006; Kevan & Pammer, 2008; Pellicano & Gibson, 2008; Hansen, Stein, Orde, Winter, & Talcott, 2001; Pammer & Wheatley, 2001; Raymond & Sorensen, 1998; Ridder, Borsting, & Banton, 2001; Slaghuis & Ryan, 1999; Solan et al., 2007; Sperling, Lu, Manis, & Seidenberg, 2006; Talcott et al., 2002; Wilmer, Richardson, Chen, & Stein, 2004; Witton et al., 1998; Wright & Conlon, 2009), but there are also examples of higher thresholds in dyslexics across other languages (Bednarek & Grabowska, 2002; Bednarek, Saldaña, & García, 2009; Boets, Vandermosten, Cornelissen, Wouters, & Ghesquière, 2011; Boets, Wounters, van Wieringen, & Ghesquière, 2006; Menghini et al., 2010; Qian & Bi, 2015). Researchers have also documented that coherent motion threshold is related to orthographic skill ($r = .26$; Boets, Wouters, Van Wieringen, De Smedt, & Ghesquier, 2008) and broad reading skill ($r = -.44$; Joo, Donnelly, & Wheatman, 2017; Pammer & Wheatley, 2001). In addition, the coherent motion threshold of pre-readers reliably predicts first grade spelling skills (Boets et al., 2008).

Table 1

Summary of Coherent Motion Designs that Account for Age Differences

Author	n	Analysis	Control and/or matching variables
Significant difference between dyslexics and typical readers			
Significant relationship between dyslexia and coherent motion ability			
Bednarek et al. (2009)	43	ANOVA	age, FSIQ
Bednarek & Grabowska (2002)	49	MANOVA	age, FSIQ
Boets et al. (2006)	62	Mixed Model Analysis (MMA)	n/a
Conlon et al. (2004)	24	ANOVA	age
Conlon et al. (2009)	87	ANOVA	age
Cornelissen et al. (1995)	54	Regression	age, FSIQ
Cornelissen et al. (1998a)	58	ANOVA, Regression	age
Dawes et al. (2009)	19	ANOVA	age, auditory processing
Eden at al. (1996)	14	t-tests, Regression	age, FSIQ, SES, education
Edwards et al. (2004)	45	MANOVA	FSIQ
Everatt et al. (1999)	36	Mann-Whitney U	age, NVIQ
Gibson et al. (2006)	88	t-tests	age, NVIQ
Hansen et al. (2001)	49	Mann-Whitney U	age, NVIQ
Kevan & Pammer (2008)	62	ANCOVA	age, FSIQ
Menghini et al. (2010)	125	MANCOVA	age
Pammer & Wheatley (2001)	41	ANOVA, t-tests	age
Pellicano & Gibson (2008)	122	ANOVA	age, NVIQ
Qian & Bi (2015)	28	ANOVA	age
Raymond & Sorensen (1998)	29	ANOVA	age, FSIQ
Ridder et al. (2001)	40	t-tests	age, gender
Slaghuis & Ryan (1999)	30	ANOVA	age, FSIQ, gender
Solan et al. (2007)	42	MANOVA, ANCOVA	age, attention
Sperling et al. (2006)	55	ANCOVA, t-tests	age, FSIQ
Talcott et al. (2002)	350	ANOVA, Regression	NVIQ
Talcott et al. (2003)	41	Mann-Whitney U	n/a
Wilmer et al. (2004)	55	ANCOVA, Factor Analysis	FSIQ, visual form detection
Witton et al. (1998)	44	t-tests	age
Wright & Conlon (2009)	130	MANCOVA	age, NVIQ
Non-significant difference between dyslexics and typical readers			
Non-significant relationship between dyslexia and coherent motion ability			
Amitay et al. (2002)	28	Kolomogorov-Smirnov test	age, FSIQ
Hill & Raymond (2002)	14	ANOVA	age, gender
Hulslander et al. (2004)	73	ANCOVA, Regression	age
Kassaliete et al. (2015)	2027	Regression, t-tests	age
Kronbichler et al. (2002)	40	t-tests	age
Ramus et al. (2003)	32	Regression	gender, FSIQ, handedness
Tsermenseli et al. (2008)	41	Kruskall-Wallis	age, FSIQ
White et al. (2006)	55	t-tests	age, NVIQ, gender

Note: NVIQ: nonverbal intelligence; FSIQ: full-scale intelligence

Though these data are overwhelmingly in support of the m-theory, there are contradictions (Amitay, Ben-Yehudah, Banai, & Ahissar, 2002; Gori, Seitz, Ronconi, Franceschini, & Facoetti, 2016; Hill & Raymond, 2002; Hulslander et al., 2004; Kassaliete, Lacis, Fomins, & Krumina, 2015; Kronbichler, Hutzler, & Wimmer, 2002; Ramus et al., 2003; Tsermentseli, O'Brien, & Spencer, 2008; White et al., 2006). Amitay et al. (2002) argued against the m-theory because their sample of adult Hebrew dyslexics performed worse than same-age peers on a number of perceptual tasks including coherent motion detection. Given the spread of difficulties, Amitay and colleagues debated if dyslexics suffered from specific deficits in the m-pathway. Still, they found that dyslexics performed worse than same-age peers on an RDK task. Similarly, Ramus et al. (2003) concluded that a portion of their adult dyslexic population had coherent motion deficits but also had other concomitant visual perceptual difficulties. The authors added that not every dyslexic showed visual difficulties, which weakens the m-theory argument.

Hulslander et al. (2004) also found a range of visual difficulties in German dyslexic twins compared to a sibling when controlling for age in their model. The authors attributed wide visual difficulties to the m-pathway and other areas of the visual cortex, reducing the significance of the m-path's contribution to reading difficulties. In a large sample of Latvian children, Kassaliete et al. (2015) demonstrated that coherent motion thresholds lie on a continuum for all readers. The authors did not target dyslexics for their sample but affirmed that their poor readers included students who might fit the criteria for dyslexic (e.g., scoring less than the 10th percentile on measures of word reading). The poorest readers had the highest thresholds on an RDK task, but other readers who struggled with word reading performed similarly to same-age peers. Kassaliete et al.

concluded that m-path deficits are not present in all poor readers, suggesting the m-theory could not explain the source of all reading difficulties.

Several researchers simply found no difference in motion thresholds between dyslexics and same-age peers. Hill and Raymond (2002) tested a small sample of adult dyslexics and determined that dyslexic adults perform similarly to same-age controls. Even then, the authors noted that dyslexics had more difficulty when the coherent dots moved in opposite directions (e.g., rather than one coherent unit among random distractor dots, the RDK included two coherent units moving in opposite directions). Kronbichler et al. (2002) concluded that middle-school, German-speaking males with dyslexia had phonological deficits, not visual impairments. However, the authors did not include any form of individual data (e.g., graphs, tables), so it is impossible to know from a group mean if any of the dyslexics had poor motion detection.

Tsermentseli et al. (2008) failed to find threshold differences between English-speaking dyslexic adults and same-age controls. However, the RDK design used by the authors was unique compared to other researchers. Instead of dots moving linearly, Tsermentseli et al. asked participants to decide which side of a fixation cross a concentric swarm of dots was moving. It is possible that the differences between the dot projections could impact the findings. As the lone finding without serious limitations, White et al., (2006) used a common two-panel RDK with English-speaking dyslexic children and same-age peers and simply found no differences in detection threshold.

In sum, though there are some data to suggest dyslexics do not have singular m-path deficits, the majority of the findings support poor motion thresholds for dyslexics

compared to same-age peers; contradictory findings are limited by samples without diagnosed dyslexics and novel RDK tasks.

Methodological quality. Despite the preponderance of studies that demonstrate dyslexics have weaker m-cell function than same-age peers when detecting coherent motion, almost all of the designs lack methodological rigor. The problem with most of the designs is that researchers fail to control for confounding variables – specifically, almost all do not account for variance due to literacy maturity.

Literacy maturity. For the purposes of this study, literacy maturity refers to a reader's ability to read text effortlessly. This skill requires automaticity at the levels of the phonological and orthographic processor, specifically at the junction of the two processors (Adams, 2013). Mature readers connect phonemes and graphemes without pause and have consolidated orthographic analysis that leads to whole-word reading attempts. In contrast, immature readers may be labored during phoneme-grapheme translation and do not have larger orthographic consolidation (e.g., they sound out words as they read or chunk words rather than reading whole words automatically).

Given that reading is not innate, literacy maturity reflects the brain's ability to adapt existing structures for new uses (Kolb & Wishaw, 2015). Mature readers activate the inferior frontal gyrus, parieto-temporal region, and occipito-temporal region (see Figure 3) during reading tasks (Shaywitz, 2003). The maturity of the occiptio-temporal region reflects the storage of larger orthographic whole-word units, and the maturity of the parieto-temporal region reflects the analysis of these units (Kearns et al., 2019). However, other regions of the brain also mature as literacy skills grow. Specifically, regions of the visual cortex and m-pathway mature alongside reading development.

Huettig, Kolinsky, and Lachmann (2018) offered that "learning to read requires that basic visual processes are adapted" (p. 275), which implies that the visual skills needed for mature reading are not innate. Skeide et al. (2017) examined the maturity of the visual cortex in illiterate adults. They used fMRI to scan their brains before and after a reading and writing intervention. The authors were surprised to find that after only six weeks, participants had considerable and significant changes to the visual cortex, thalamus, and brainstem – all important for visual attention. These changes were not noticeable in an illiterate control group. Notably, Skeide et al. found the biggest changes in the tectopulvinar visual system, not the geniculostraiate visual system running from the LGN to V1 to the dorsal stream that involves m-cells (seen in Figure 5). However, Kaas and Lyon (2007) reviewed empirical imaging studies of the pulvinar visual system and found evidence that a portion of nuclei in the inferior pulvinar transmit and receive signals to the dorsal stream (i.e., m-stream). Taken together, these findings demonstrate that learning to read improves the visual cortex and the magnocellular pathway.

Fernandes, Coelho, Lima, and Castro (2018) found similar results when studying groups of pre-literate children and illiterate adults. One comparison focused on pre-literate children and age-matched beginning readers; the second comparison focused on illiterate adults and age-matched literate counterparts. The authors questioned if learning to read enhanced the brain's ability to process the orientation of objects. Fernandes et al. provided evidence that this ability depended on the dorsal stream (i.e., m-path) and its projection area, the posterior parietal cortex. They demonstrated that better readers made fewer orientation errors, suggesting that learning to read refines dorsal stream function and, as a result, function of the posterior parietal cortex.

Malik-Moraleda, Orihuela, Carrieras, and Dunabeitia (2018) also studied illiterate and literate adults. They measured visual scanning for target letters or symbols among nonword letter strings and random visual forms and found that literate adults were faster and more accurate than illiterate peers. The authors explained that literate adults were able to separate the nonword letter strings into units because of their literacy maturity, which boosted their visual scanning performance. Huettig et al. (2018) echoed this finding when he stated that "literacy acquisition promotes a type of analytic, part-based processing that does not seem to be inherent to the visual system as it is not observed in illiterate adults" (p. 276). In sum, learning to read results in changes to m-cell function in the visual cortex.

As learning to read enhances visual function, dyslexics are at a true disadvantage. Emerging literacy skills are developed reciprocally with practice (Share, 1995). Share (1995) described the phenomenon as the self-teaching mechanism of reading. The more beginning readers practice, the better they get; the better they get, the more they read. Dyslexia is characterized by difficulties with basic skills – phonological recoding and orthographic consolidation – needed for successful reading attempts (Ehri, 2017; Vellutino et al., 2004). Because dyslexics struggle to reach automaticity with basic skills, they read fewer words than peers. Reading fewer words leads to less practice, which limits basic skill growth, and so on. Therefore, reduced reading experiences may not lead to the same visual cortex maturity as skilled readers at similar ages.

Research designs and literacy maturity. Researchers should account for the cognitive-perceptual differences between dyslexics and same-age peers due to literacy maturity. Goswami (2003, 2015) suggested that researchers could account for brain

changes following literacy maturity by including a younger control group matched to dyslexics on reading-levels. She also argued that origin research depends on assigning causality, and the first step toward causality is confirmation that deficient performance on a basic, underlying skill (e.g., m-function) separates dyslexics from other children at similar levels of reading development (Goswami, 2003). In other words, researchers must demonstrate that the underlying difficulty could cause the reading problem rather than serve as a consequence of prolonged reduced reading experiences, and the first step is to establish differences between readers on the underlying skill. She follows that a significant finding in a comparison study should be followed with a training study, but she does not waver that the first step is establishing deficient performance after accounting for the impact of literacy maturity.

Accounting for literacy development provides the pre-requisites for causality arguments but also answers the question of deviance or delay for dyslexia m-cell function. Deviant m-function in dyslexics would be unique or atypical from the developmental progression of normal readers (Talcott et al., 2013). Delayed m-function would parallel the m-function of younger peers at similar levels of literacy maturity and similar levels of literacy-based brain changes. Determining deviant or delayed m-function in dyslexics impacts diagnosis. If a measure of m-cell function were to be deviant and not dependent on reading instruction and practice, then prereaders could be diagnosed with dyslexia, leading to earlier remedial efforts. The current state of dyslexia diagnosis requires formal instruction to occur before diagnosing and addressing the problem.

Researchers have explored dyslexics' m-cell function for over thirty years. Currently, researchers use data from age-matched comparisons to debate deviance or

delay in dyslexia. However, the debate requires knowledge of dyslexic performance compared to children with similar literacy maturity. At present, there are few empirical studies that examine the impact of literacy maturity on the cortical m-pathway.

Coherent motion and literacy maturity. When critically analyzing the existing literature on m-path function and coherent motion detection, most designs do not include reading level-matches. Seen in Table 1, 36 recent empirical designs comparing coherent motion detection in dyslexics to controls account for age differences in their models or match participants on age prior to data analysis. In contrast, Table 2 shows six studies that collectively account for differences in literacy maturity.

When examining the results from designs that control for literacy maturity, the results are mixed. Out of six studies, four included group comparisons. Two supported deviance in m-cell functioning for dyslexics, meaning that after accounting for reading ability and age, dyslexics had impaired motion thresholds (Cornelissen et al., 1998b, Gori et al., 2016). The other two comparison studies supported delay in m-cell functioning for dyslexics, meaning that dyslexics' m-cell function was like younger peers with similar literacy maturity (Olulade et al., 2013; Talcott et al., 2013). The final two did not include comparisons but found that motion thresholds did not improve as reading ability improved (Joo et al., 2017; Taylor et al., 2018). The following discussion will summarize and critique the six studies.

Table 2

Summary of Coherent Motion Designs that Account for Reading Level Differences

Author	n	Analysis	Control and/or matching variables
Group comparisons			
Cornelissen et al. (1998b)	58	Regression	age, FSIQ, single word reading, phonological awareness
Talcott et al. (2013)	350	ANOVA, t-tests	age, NVIQ, reading level
Intervention studies			
Gori et al. (2016)*	46	ANOVA, Regression	age, IQ, reading level
Joo et al. (2017)	48	Wilcox-rank Sum Regression	age
Olulade et al. (2013)*	78	t-tests ANOVA	age, NVIQ, reading level
Longitudinal descriptive			
Taylor et al. (2018)	12	ROI analysis	one sample of typical readers

Note: *Gori et al. (2016) and Olulade et al. (2013) also included group comparisons.

Support for deviance. Gori et al. (2016) compared coherent motion detection in three groups of Italian children – poor readers, age-matched controls, and reading-level matched controls. Interestingly, the authors chose a unique RDK task that measured accuracy at randomly assigned levels of coherence (e.g., 20%, 60%, 80%, 40%) rather than calculating an overall coherence threshold like researchers in same-age comparison studies. Gori and colleagues demonstrated that dyslexics had lower motion detection accuracy than age and reading-level matched peers.

The authors also measured motion detection accuracy in pre-readers at age five and again at the end of first grade and found that poor readers at the end of first grade had poorer motion accuracy as pre-readers (Gori et al., 2016). Importantly, the authors argued that their data proved m-function was established prior to reading experience and that literacy maturity did not impact m-function. However, they failed to acknowledge that only 10% of the variance in first grade reading skill was explained by pre-reading m-

function. Further, they measured phonological segmentation in pre-readers but did not report if the groups were significantly different on either measure as pre-readers. Certainly, phonological weakness as a pre-reader could explain more variance in first grade reading performance than m-function, yet their argument was that m-function was driving the differences in reading ability.

Finally, Gori et al. (2016) designed a training study to improve m-function in dyslexics using video games. They found that video game playing in Italian dyslexics improved both reading skill and motion accuracy. However, Gori et al. did not include a control group for comparison to know if m-cell function improved because of status-quo school-based instruction or because of the video game. Taken together, Gori et al. found that dyslexics have weaker m-function than same-age and younger readers, that pre-readers' weakened m-function predicts future reading failure, and that video game playing improves reading ability and motion thresholds. The authors used their data to argue that dyslexics' m-function was deviant, or atypical, of other peers and that improving m-function could also improve reading ability.

Cornelissen et al. (1998b) did not use a group comparison design but controlled for reading ability in a logistic regression model. Corenlissen et al. examined letter errors across all readers, including poor readers who might qualify as dyslexic. Letter errors were defined as reading attempts that included sounds outside of the printed word. For example, *vikim* would be a letter error for *victim* and *subpact* would be a letter error for *suspect*. In contrast, *blanket* for *banker* (i.e., whole word substitution that carries meaning) and *fevver* for *fever* (i.e., phonological substitution that preserves letter type) were not counted as letter errors (p. 474).

In Cornelissen and colleagues' (1998b) logistic regression model, they examined the relationship between the proportion of letter errors and motion thresholds, controlling for IQ, age, phonological awareness, and reading ability (i.e., single word reading). They concluded that the letter errors made by poor readers were expressions of orthographic weakness caused by reduced letter sequencing and positioning skills housed in the magnocellular pathway. Because they found a significant relationship between the m-path and letter errors even after accounting for literacy maturity, their argument supports deviant magnocellular function in dyslexics. Yet Cornelissen and colleagues cautioned that not all dyslexics may have m-cell weaknesses and offered that phonological and magnocellular difficulties were not mutually exclusive.

Support for delay. Talcott et al. (2013) examined sensory (i.e., visual and auditory) and phonological skills in 69 children with reading disability assigned to a phonological-deficit group, an orthographic-deficit group, or a combined-deficit group. Each group was assigned age-matched controls and reading-level matched controls. Age-matched controls were required to have reading skills within 12 months of their ages, but this timeframe is too wide. Talcott et al. sampled 7 to 12 year old children, which meant that the seven-year-old dyslexic required an age-matched peer with reading skills within 12 months of his or her age. The age-matched seven-year-old reading 11.5 months behind could be a pre-reader focused on building basic alphabet skills just like the dyslexic counterpart. Therefore, Talcott et al.'s design is limited by possible overlap in the dyslexic and control groups.

Despite the limitation, Talcott et al. (2013) concluded that the combined deficit group had higher thresholds for coherent motion detection compared to age-matched

controls. Neither the phonological only nor orthographic groups had significantly different thresholds when compared to age-matched controls. When the combined deficit group was compared to the reading-level controls, the combined group was no longer significantly impaired on coherent motion. Taken together, these findings suggest that m-function in dyslexics is delayed.

Olulade et al. (2013) provides the only imaging evidence that m-function is delayed in English-speaking dyslexic children, but the authors' RDK task was unique compared to most designs. Rather than finding a coherence threshold in participants, Olulade and colleagues set the coherence level at 40% for high accuracy and measured brain activation during responses. This constraint was necessary so the analysis of fMRI data did not include obscure group differences due to performance variability across multiple levels of coherence.

In the first phase of a three-phase study, the authors found moderate correlations between reading ability (i.e., real and nonword reading) and V5/MT activity in typically reading children and adults (real words – left V5/MT: $r = 0.46$; right V5/MT: $r = 0.52$; nonwords – left V5/MT: $r = 0.41$; right V5/MT: $r = 0.61$). In the second phase, Olulade et al. (2013) compared motion accuracy in dyslexic children to age-matched and reading-level matched peers and found that dyslexics performed worse than same-age peers but similar to peers with comparable literacy maturity. In the final phase, 22 dyslexic children participated in an intervention period that targeted phonological/orthographic knowledge, a control period that targeted mathematics, and a control period with no intervention. The authors used fMRI to measure participants' motion accuracy before, during, and after intervention periods. Olulade et al. found that the intervention improved

reading ability and increased activation in right area V5/MT during the motion task. The authors concluded that learning to read has a positive influence on m-function and stated that "the magnocellular visual deficit is a consequence and not the cause of impoverished reading" (Olulade et al., 2013, p. 7).

The heterogeneity of the dyslexic group limits these results – dyslexics had standard scores ranging 41 to 91 on a measure of real word reading and scores ranging 47 to 98 on a measure of nonword reading. This means some dyslexics had average word reading abilities. Vellutino et al. (2004) questioned the inclusion of these participants in dyslexia studies and suggested that wide ranges of ability in research complicate the findings.

Improved reading not related to improved motion threshold. Joo et al. (2017) cited Olulade et al.'s (2013) findings and designed an intervention study to determine if improving reading ability led to concomitant improvements in coherent motion detection. Joo et al.'s intervention was identical to Olulade et al., and Joo et al. also used a unique RDK task with black and white pixels rather than only white pixels. Joo and colleagues demonstrated that throughout the intervention period, both good and poor motion detectors improved their basic reading skills but not their motion thresholds. That is, improving the literacy maturity of good and poor motion detectors did not result in significant improvements to their motion detection. The authors suggested that "motion processing deficits are among a collection of correlated risk factors for reading difficulty" (Joo et al., p. 4). Overall, Joo and colleagues' findings imply that weaker m-function in dyslexics is not caused by literacy immaturity.

Finally, Taylor et al. (2018) measured V5/MT activation during a motion task longitudinally from the end of second grade to the winter of third grade. Their goal was to observe any changes in motion detection as reading experience grows (e.g., from the end of second grade to third grade). The RDK task was identical to Olulade et al.'s (2013), meaning the task did not measure coherence threshold but was designed for high accuracy to locate brain regions associated with motion detection. Taylor et al. stated that their 12 participants were "typical readers" (p. 224), but average word reading and nonword reading performance was reported as standard scores of 118 and 117, respectively. These scores are in the upper end of the high average range, meaning their participants were not typical readers – they were excellent readers.

During the RDK task, the 12 children showed bilateral activation of area V5/MT, with greater activation on the right side, confirming that motion is detected in area V5/MT in children. After 10 months of daily living, the children returned for a second scan, again showing bilateral activation of V5/MT. In contrast to Olulade et al.'s (2013) argument that reading experience improves motion detection, Taylor et al. (2018) found no differences in activation strength after 10 months. Taylor and colleagues concluded that reading experience may not impact motion detection like previously hypothesized and that the m-pathway may not be related to reading improvement.

The problem with Taylor and colleagues' (2018) conclusions are twofold. First, their conclusions were not representative of all readers and surely not dyslexic readers. Second, a longitudinal study from the end of second grade to the winter of third grade does not capture the learning to read process. Their second graders were already proficient readers (e.g., above average word and nonword reading, fluency, and

phonological awareness) with basic reading skills. Therefore, when Taylor et al. (2018) stated that "reading development did not appear to be driving" (p. 237) motion detection skill, they did not consider that basic reading skills were already developed in their participants. It is possible that the m-pathway reaches a ceiling level of efficiency as readers master basic skills. In other words, it is possible that m-cells are trained during the learning to read process; once basic skills are mastered and fluent reading begins, m-cells can function efficiently during reading. The findings from Taylor and colleagues would be strengthened with similar results in a longitudinal study from the end of Kindergarten to the end of first grade when learning to read occurs.

Overall, most researchers approach the debate of m-cell deviance or delay in dyslexia with age-matched designs. Most of these researchers demonstrated significant differences in motion threshold between dyslexics and same-age peers or found significant relationships between motion threshold and dyslexia when controlling for age. A small number of researchers had contradictory findings, but the majority of age-matched data supports differences in the m-pathway between dyslexics and same-age peers. However, age-matched designs do not account for an important confounding variable – literacy maturity.

Few researchers have examined coherent motion detection with respect to literacy maturity, and their findings have limitations. Gori et al. (2016), Olulade et al. (2013), Joo et al. (2017), and Taylor et al. (2018) used unique RDK tasks not seen in age-matched studies. Other studies had sampling variations that did not include diagnosed dyslexics (Cornelissen et al., 1998b; Taylor et al., 2018) or included dyslexics with average reading ability (Talcott et al., 2013). Despite the need for studies that consider literacy maturity

(Goswami, 2015), the existing body of empirical literature adds little to the debate over m-cell deviance or delay in dyslexia.

Conceptual Framework

To frame this study, I built a model of reading to illustrate how readers build literacy maturity, or consolidated, automatic phoneme-grapheme units and what can go awry in dyslexia.

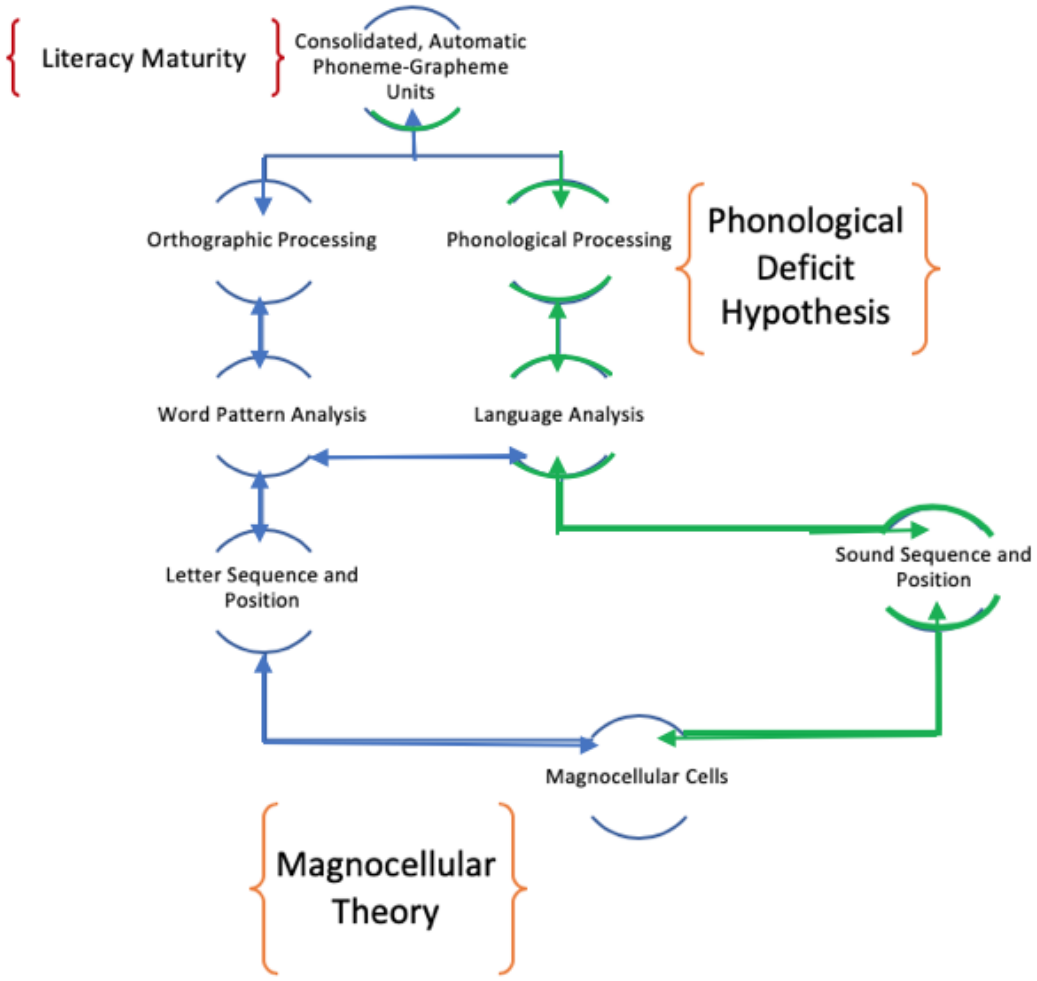

Figure 7. Conceptual Model of Reading and Dyslexia

Building Literacy Maturity

This model illustrates the intrinsic pathways that build consolidated, automatic phoneme-grapheme units in mature readers. The blue path represents the pathway that involves print and is most germane to this dissertation. However, it is important to acknowledge that analysis of spoken language overlaps with analysis for written language. The green path shows how auditory cells similar to visual magnocellular cells impact language analysis during phonological tasks without printed text (e.g., phonological deletion and segmentation tasks, rhyming tasks).

Importantly, for children with knowledge of phoneme-grapheme correspondences, oral tasks that begin on the green path will still activate part of the blue path because of the associations between the phonological and orthographic processors (Adams, 2013; Ehri, 2017). Even children with beginning letter-sound knowledge (e.g., *pat* starts with /p/, which corresponds to the letter *p*) have already begun wedding orthographic knowledge to phonological knowledge such that sounds automatically activate the corresponding visual forms, and vice versa (Ehri, 2005, 2017). A second critical feature of this model is the bidirectional nature of the relationships. The brain uses feedforward and feedback loops to continually refine perception and action (Kolb & Whishaw, 2015), so signals are sent up and down the model for refinement, accuracy, and efficiency.

I will describe the model from the bottom of the blue reading path for ease; however, the current literature base has not established if the top or bottom of the proposed model would more heavily influence the other. Visual m-cells are at the most basic level of the model and enable readers to sequence letter strings, encode letter

positions, and do these tasks efficiently (see Stein, 2019). Accurate and efficient letter sequences and positions enable word pattern analysis by the orthographic processor, which applies "overlearned knowledge about likely and unlikely sequences of letters" (Steacy et al., 2017, p. 792). Moving horizontally from word pattern analysis, the letter sequences activate the pattern's associated sounds (Ehri, 2017) in the phonological processor. These processes happen simultaneously and instantly in the skilled reader. The letter patterns from the orthographic processor are combined with the letter sounds from the phonological processor to build phoneme-grapheme units. These units vary in size, from one-to-one correspondences (*b* = /b/) to rimes (*ate* = /et/) to syllables (*ra-don* = /re-dɑn/) to whole words (*splat* = /splæt/). Literacy maturity reflects the consolidation and automaticity of larger units.

Dyslexia in the Conceptual Model

Referring to Figure 7, researchers have established that children with dyslexia have meaningful differences in the ability to automatically consolidate phoneme-grapheme units, which can be traced back to weakeness in phonological processing (Vellutino et al., 2004). In fact, the Phonological Deficit Hypothesis (Stanovich, 1988) and recent imaging studies (see Pugh et al., 2013) have highlighted the role of phonological processing in dyslexia. However, a full understanding of the cortical underpinnigns of dyslexia requires that Figure 7 extend beyond the phonological processor and Phonological Deficit Hypothesis. Even Stanovich stated that "the more fundamental processes that might underpin the phonological processing problem is a source of much contention" (Stanovich, 1996, p. 155), thereby acknowledging that other cortical processes – which are still debatable today – impact phonological processing.

Orthographic processing difficulties do not receive the same attention as phonological processing difficulties in the dyslexia literature, but a recent focus on orthographic knowledge has revealed meaningful differences between children with dyselxia and same-age peers on measures of word pattern analysis and consolidation (see Ehri, 2017; see also Steacy et al., 2019).

Moving down the model in Figure 7, researchers have also demonstrated that children with dyslexia have weaker accuracy and slower speed during inter-letter sequence recognition and during sound sequence tasks; however, other researchers have found similar difficulties in other poor readers (see Elliott & Grigorenko, 2014 for a review of this debate). Consequently, and most relevant to this study, researchers are now debating the role of m-cells in dyslexia. Researchers have documented differences in m-cell function in children with dyslexia compared to peers (see Stein, 2019). However, the comparisons made between dyslexics and peers have been critcized for failing to include other poor readers (Goswami, 2015), which may give a false positive result. That is, deviant m-cell function should separate children with dyslexia from other poor readers, not just typical readers the same age as dyslexic readers (Goswami, 2015). Therefore, researchers are still debating if children with dyslexia even have meaningful differences in m-cell function when compared to other strong and weak readers.

Hypotheses based on the Model

Based on the existing literature and my current conceptual model, there are two likely hypotheses for m-cell function in children with dyslexia:

First, children with dyslexia have delayed m-cell function, or comparable m-cell function to younger peers at similar levels of literacy. Taking a top-down interpretation of

Figure 7, this hypothesis suggests that children with dyslexia and younger peers with similar levels of literacy maturity have weakened phoneme-grapheme units, which results in fewer reading opportunities (Vellutino et al., 2004). Reduced reading experiences lead to fewer opportunities for statistical and analogical learning of word pattern analysis (Steacy et al., 2017), which leads to fewer known letter strings or patterns. Readers who know fewer sequences of likely and unlikely letter strings take longer to confirm letter sequences (McClelland & Rumelhart, 1981). Longer inter-letter processing may not efficiently train m-cells to perform quickly, which may result in weaker m-cell performance on clinical measures. Put another way, as younger children and children with dyslexia are unable to use larger phoneme-grapheme units during word and sound analysis, their m-cells may or may not be capable of faster speeds, but the necessity for speed has not been established.

In contrast, readers with strong literacy maturity who consolidate word patterns may need m-cells to signal quickly because letter order and position are reinforced by neighboring letters (Adams, 2013), speeding up the movement to subsequent letters and words. In this way, m-cells may be trained to "keep up" with the orthographic processor as it learns likely letter patterns. Given that m-cells continue to develop and become more efficient during the school-age years (Boden & Giaschi, 2007; Crewther et al., 1999; Goswami, 2003), it logically follows that a child with significantly fewer opportunites for transient spatial analysis of print may not train or refine m-cells similarly to typically developing peers who encounter vast amounts of print that requires transient spatial analysis. As a result, children with dyslexia will have weaker m-cell function than peers who have performed more transient spatial analysis of print; however, children with

dyslexia will have comparable m-cell function to peers with similar levels of spatial analysis during print reading.

A second, alternate hypothesis of m-cell function in dyslexia takes a bottom-up approach to Figure 7. In this hypothesis, m-cell function in dyslexia is deviant, or unique, even after considering the impact that literacy maturity may have on the visual cortex. Put another way, children with dyslexia face difficulties in m-cell function that cannot be explained by the learning to read process and the impact that learning to read may have on training the visual cortex. This hypothesis suggests that unique m-cell differences increase inter-letter and inter-word timing, which reduce the precision of letter strings, which causes disruptions in word pattern analysis and orthographic processing, which in turn causes problems communicating with the phonological processor and ultimately establishing automatic, consolidated phoneme-grapheme units.

Critically, each hypothesis offers a theorized version of m-cell function and development in children with dyslexia. Establishing causality from m-cells to reading difficulties is not the goal of this study; rather, this study aims to explore if children with dyslexia have a unique pattern of m-cell function even after the impact of literacy maturity has been accounted for that deserves future attention to establish pathways of causality. As Goswami (2015) suggested, future studies would attempt to establish causality once the literature base accurately describes the neurological problems that dyslexics face.

Gaps in Existing Literature

This study builds on existing research on the magnocellular theory of dyslexia to understand if magnocellular function in dyslexics is deviant or delayed. The vast majority

of researchers who address the role of magnocellular function assume deviance but do not account for possible confounding variables in their designs. Specifically, most designs fail to account for the differences in literacy maturity (i.e., automatic, consolidated phoneme-grapheme units) between dyslexics and same-age peers.

A very small number of researchers have explored magnocellular function with respect to literacy maturity (Cornelissen et al., 1998b; Gori et al., 2016; Joo et al., 2017; Olulade et al., 2016; Talcott et al., 2013; Taylor et al., 2018). However, three of the six researchers did not sample dyslexics (Cornelissen et al., 1998b; Talcott et al., 2013; Taylor et al., 2018). The other three used novel tasks to measure magnocellular function compared to previous designs (Gori et al., 2016; Joo et al., 2017; Olulade et al., 2016). When discussing the importance of additional studies that use similar procedures as the established literature base, Kim, Davis, Burnham, and Luksaneeyanawin (2004) stated that "the use of the same method and procedures increases confidence that the findings have not been affected by differences in methodology" (p. 327). Therefore, a review of the previous literature presents a gap in the knowledge base. Presently, there are no studies of dyslexic magnocellular function with respect to literacy maturity under conditions that mirror previous age-matched designs.

Research Questions

The overarching goal of this study is to examine if magnocellular function in dyslexics is deviant or delayed and if m-cell function could be used as a diagnostic sign of dyslexia. To accomplish this goal, my objectives are to compare magnocellular function in dyslexics to same-age peers and to peers with similar literacy maturity. The following research questions address my objectives.

1. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar ages, controlling for nonverbal ability?
2. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar levels of literacy maturity, controlling for nonverbal ability?

CHAPTER III

METHODOLOGY

In this observational study, I compared the magnocellular (m-cell) function of children with dyslexia to age-matched and reading level-matched peers. The purpose of this study was to determine if weakened m-cell function is unique to children with dyslexia or if m-cell function is delayed in children with dyslexia and more characteristic of younger peers at similar literacy levels. I administered a range of visual and literacy measures in one-hour sessions to confirm qualification, assign participants to groups, match peers on age and reading level, and determine m-cell function.

Study Design

I used a non-experimental, group comparative design for this study. Previous researchers have compared m-cell function between dyslexics and same-age controls (see Benassi et al., 2010). However, some have argued that comparing magnocellular (m-cell) function in dyslexics and same-age controls does not account for the confound of reading experience (i.e., literacy maturity; Goswami, 2015; Olulade et al., 2013). Olulade and colleagues (2013) hypothesized that learning to read may bootstrap the development of the magnocellular pathway. To account for the possible confounding variable of literacy maturity, I compared m-cell function in children with dyslexia to younger peers matched on automatic, phoneme-grapheme knowledge (i.e., literacy maturity). I also compared m-cell function in children with dyslexia to same-age, typically developing peers, which

replicated previous findings. Both comparisons accounted for pre-existing differences in nonverbal ability, visual discrimination, visual acuity, and line orientation.

Participants and Setting

Participants

The sample comprised 90 children between 6 and 14 years of age [*M*(SD) = 9.42(2.28)] from Virginia. Thirty children between 8 and 14 years of age [*M*(SD) = 10.63(1.78)], had a prior diagnosis of dyslexia, including 14 males and 16 females. An additional 30 children between 8 and 14 years of age [*M*(SD) = 10.63(1.78)], including 17 males and 13 females, served as age-matched controls to the dyslexia group. A final group of 30 children between 6 and 8 years of age [*M*(SD) = 7.02(.75)], including 13 males and 17 females, served as reading-level matched controls to the dyslexia group.

All 90 children spoke English as their first language. Information on race, ethnicity, or socioeconomic status was not collected to protect the anonymity of children and families. Despite the fact that neurological imaging shows similarities in dyslexics across racial and ethnic boundaries, dyslexia has historically been a diagnosis of privilege that is typically less available to children from racial/ethnic minority backgrounds or low-income statuses (Hoyles & Hoyles, 2010; Robinson & Thompson, 2019; Wolf, 2019). Therefore, African American and Hispanic children in the United States who struggle to acquire literacy are significantly under-identified with dyslexia (Robinson & Thompson, 2019). In a sample of only 30 children with dyslexia, it was very likely that a child could be identified by his/her race if such information was included. As a result, information on race and ethnicity, while important to understanding the population of dyslexics, was

omitted from this study due to low sample size and the possibility of breaching anonymity.

Geographic information. Participants selected a public library, church, or community center as a location for the testing. Seen in Table 3, participants requested to meet across Virginia, with a high number of children from Central and Southwest Virginia.

Table 3

Geographic Regions Where Participants Requested to Meet

Region	Dyslexia	Controls
Southwest		
Montgomery County/Carroll	0	8
Roanoke/Salem	3	16
Northwest		
Augusta/Harrisonburg/Waynesboro	2	7
North		
Loudoun/Fairfax/Arlington	5	3
Central/Piedmont		
Gordonsville/Orange/Charlottesville	6	6
Louisa/Fredericksburg/Richmond/Chester	7	10
Lynchburg/Danville	4	9
East/Tidewater		
Chesapeake/Newport News/Norfolk	3	1

Examiner

I conducted all assessments individually with participants. My training as a Reading Specialist and Doctoral Clinician in the McGuffey Reading Center has included specific teaching, observation, and feedback on the measures used in this study. I have also been trained on neuropsychological measures and taken courses focused on neuropsychological systems and assessment. In addition, the software developer for the magnocellular task (i.e., the outcome variable) provided me with one-on-one training in the clinical use of the software program. In total, my training and experience with the measures exceeds 100 hours.

Setting

This study took place at libraries, community centers, and churches across Virginia. The vast majority of participants asked to meet at local libraries; four participants asked to meet at a local community center and two asked to meet a local church due to scheduling conflicts during library operating hours. Data collection occurred in the following regions: Northern (e.g., Loudoun, Fairfax, Arlington), Central/Piedmont (e.g., Orange, Charlottesville, Fredericksburg, Richmond, Louisa, Lynchburg, Forest, Danville), Eastern/Tidewater (e.g., Newport News, Norfolk, Chesapeake), Northwest (e.g., Harrisonburg, Augusta, Waynesboro), and Southwest (Roanoke, Salem, Blacksburg, Carroll). I conducted the assessments in a private room with no other individuals present. Three participants asked a parent/guardian to sit in the room during the testing session.

Procedures

The procedures for this study occurred in phases, with multiple decision points in each phase (see Figure 8). The phases were recruitment, assessment, grouping, and matching. A detailed description of each phase can be found below.

Recruitment

With approval from the University of Virginia Institutional Review Board (IRB), I recruited elementary and middle school children in Virginia with and without dyslexia. I recruited children with dyslexia before typically-developing peers because of the matching procedures in this study. During the first phase of recruitment, I created an IRB approved advertisement for children with dyslexia (see Appendix A). The advertisement described the study as an investigation of neurological reading processes for children in

elementary and middle school and specifically asked for volunteers with diagnosed dyslexia. I provided my phone number for interested families to call or text for more information.

I placed dyslexia flyers at assenting libraries, psychological assessment centers, tutoring centers, churches, gyms, grocery stores, and pediatrician offices across the following regions in Virginia: Northern (e.g., Loudoun, Fairfax, Arlington), Central/Piedmont (e.g., Orange, Charlottesville, Fredericksburg, Richmond, Louisa, Lynchburg, Forest), Eastern/Tidewater (e.g., Newport News, Norfolk, Chesapeake), Northwest (e.g., Harrisonburg, Augusta, Waynesboro), and Southwest (Roanoke, Salem, Blacksburg, Christiansburg, Carroll). An anonymous individual also posted an electronic version of the flyer to the Facebook page of the parent group, Decoding Dyslexia-Virginia; parents operate this page and posted the flyer voluntarily. I ensured the posting met the guidelines given by the IRB.

In addition, I asked school districts and private schools to send flyers home with children identified as dyslexic. A total of 81 districts and/or private schools received a maximum of three emails and two calls. Of the 81 districts and schools contacted, 9 public school systems and 5 private schools agreed to send home dyslexia flyers to parents. The flyers included information to contact the researcher if families wanted to participate; consent occurred in person and not via school letters.

Once I secured 75% of the participants for the dyslexia group, I recruited two control groups. First, I created a flyer seeking typically-developing elementary and middle school children for a study on reading and the brain; the IRB approved the flyer

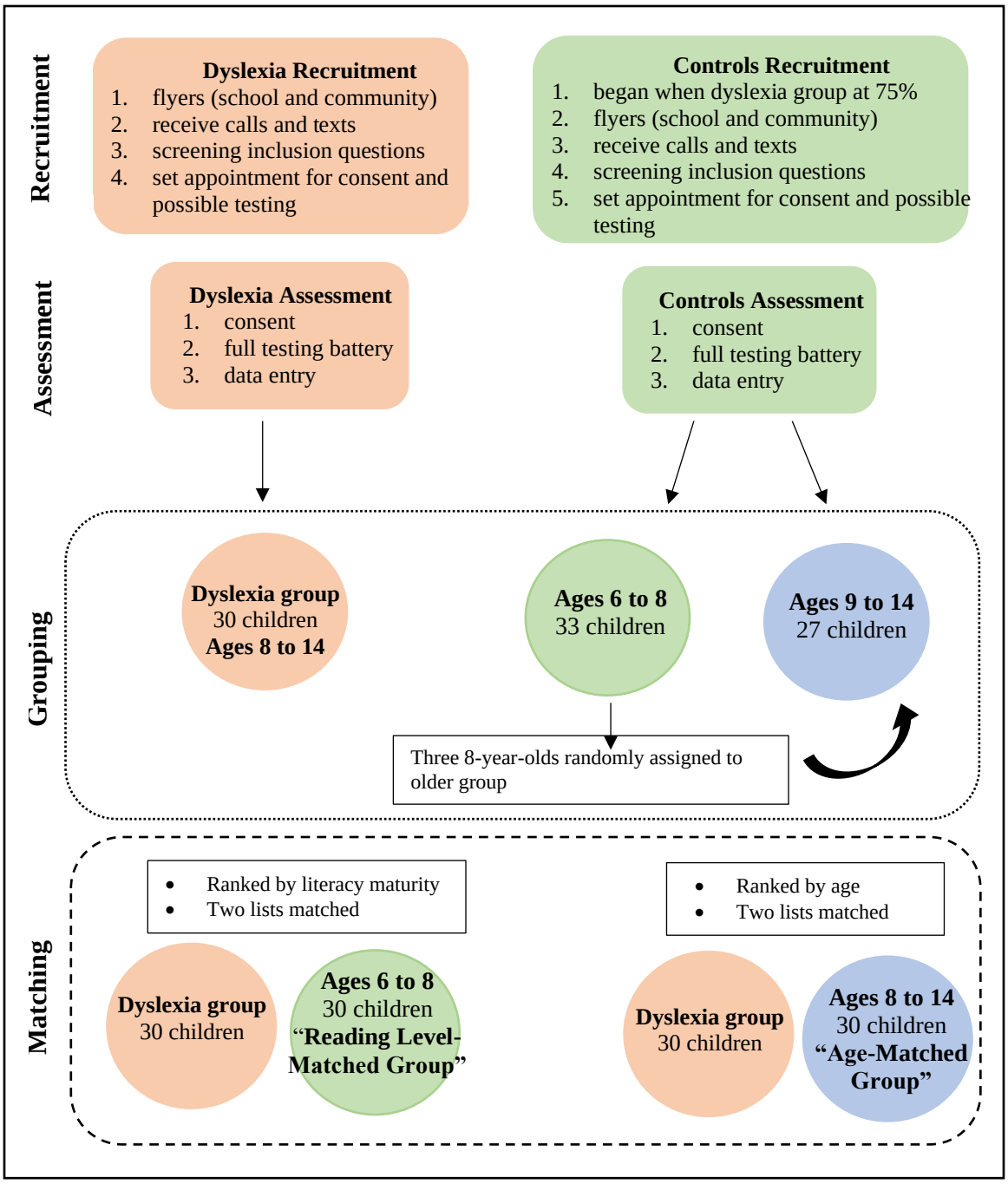

Figure 8. Four Phases of Procedures

(see Appendix B). Second, I placed flyers at assenting libraries, churches, gyms, grocery stores, and pediatrician offices across Virginia. Many owners who initially posted the dyslexia flyer allowed me to post the control flyer as well; however, the control group recruitment included 35 new locations.

Seventy-six of the original 81 school districts were contacted to assist with control group recruitment. The districts knew of the follow-up procedure during initial recruitment for the dyslexia group. I did not contact five private schools that sent dyslexia flyers because they specialized in teaching children with various behavioral or academic difficulties. Each district received a maximum of three emails and two calls. Six school districts agreed to send home a flyer to all students in Kindergarten through Ninth Grade. One district sent home flyers for both the dyslexia and control groups; however, nesting is unlikely because the district enrolls over 10,000 students.

Recruitment occurred simultaneously for the two control groups (e.g., age-matched and reading-level matched). I did not assign participants to a specific control group at the recruitment stage.

Screening. Families who called or texted the phone number listed on the recruitment flyer received a phone call or text to conduct a basic screening before moving through informed consent procedures. To screen potential participants, I read through scripted statements, which varied across groups. For the dyslexia group: (1) Your child has a diagnosis of dyslexia; (2) Your child is between the ages of 8 to 14; (3) Your child has reading difficulty; (4) Your child's primary language is English. For the control groups: (1) Your child has not received any prior educational labels or diagnoses related to learning; (2) Your child is between the ages 6 to 14; (3) Your child is meeting or exceeding school-based reading benchmarks; (4) Your child's primary language is English. I recruited control group participants at a slightly younger age than the dyslexia group because the design required older children with dyslexia matched to younger peers reading at similar levels. A family member confirmed the statements before scheduling a

date, time, and library location to discuss consent in person. If the family member could not confirm all of the statements, the conversation did not continue.

As anticipated, recruitment and screening for the control participants became more stringent after testing the entire dyslexia group. For example, the dyslexia group included two 14 year-old participants; therefore, I did not move past the screening stage of recruitment if a control family called with a 14 year-old after those two spots were filled. This situation occurred six times, including twice for age 9, three times for age 10, and once for age 12. For younger controls, I continued to recruit children between the ages of 6 to 8 until 33 participants qualified (30 going to the reading-level group and 3 going to the age group).

Assessment Procedures

Following consent, I conducted all assessments in a single, individual session. Each assessment took approximately 1 hour and every participant completed the entire assessment battery. I administered the measures in the same order: three visual rule-out measures, five literacy measures, the measure of m-cell function, and finally the nonverbal ability measure (see descriptions below). Two 5 minute breaks were offered – one after the visual measures and one after the measure of m-cell function.

Parent participation. Parents or guardians of participating children with and without dyslexia completed a short participant demographic questionnaire. After obtaining consent, parents/guardians completed the questionnaire in less than 5 minutes. All participants provided the following demographic information: biological gender, birthdate, known disabilities, current grade level, intervention history, and retention history.

Qualification inclusion. After each assessment, I scored the protocols to determine qualification. To qualify for the study, participants had to meet the following score minimums. Children in the dyslexia group needed to score in the average range on each of the three visual rule-out measures and score in the bottom 10th percentile on any of the literacy measures. Children in the control group needed to score in the average range on each of the three visual rule-out measures and score above the bottom quartile (i.e., above 25th percentile) on four out of five of the literacy measures. Detailed descriptions of the measures and required scores can be found below. I tested 112 children for the study; 90 children qualified for participation. Information on the 22 children tested who did not qualify for the study can be found in Table 4.

Table 4

Participants Tested Who Failed to Qualify

Reason for disqualification	n
Did not meet dyslexia diagnosis reading criteria	2
Control group reading abilities too low	6
Failed Visual Acuity screener	2
Did not meet Directionality score minimum	2
Did not meet Object Discrimination score minimum	3
Did not meet Directionality + Object Discrimination score minimums	4
Undisclosed diagnosis during recruitment screening	3

Note. Descriptions of the measures can be found below.

Half of the potential participants failed to qualify because of low scores on the three visual rule-out measures (e.g., Visual Acuity, Directionality, Object Discrimination). Eight more failed to qualify because of their literacy scores, with two potential participants scoring too high for qualification in the dyslexia group and six scoring too low for membership in the control groups. Participants who scored too low

for the study lacked basic knowledge of the alphabet or failed to read enough words on the literacy measures to continue beyond practice trials. The final three potential participants had additional diagnoses (e.g., Autism, ADHD) not disclosed during the initial recruitment screening conversation.

Participant Grouping

I assigned qualified participants to one of three groups for analysis: (1) children with dyslexia, (2) typically-developing younger children matched to the reading-levels of the dyslexia group (i.e., reading-level matched group), and (3) typically-developing children matched to the ages of the dyslexia group (i.e., age-matched group). Participants with dyslexia were assigned to the dyslexia group. Once I tested all dyslexia participants, I analyzed the dyslexia ages and assigned control participants. The dyslexia group spanned 8 to 14 years old but included only three 8 year-old children. Therefore, I assigned all 9- to 14-year-old control participants to the age-matched group. As the reading-level matches to the dyslexia group needed to be younger peers, I assigned all 6- to 8-year-old control participants to the reading-level matched group.

After 27 participants qualified for the age-matched group (i.e., leaving only the three 8-year-old spots open), I randomly assigned three 8-year-old participants from the reading-level group to complete the age-matched group, leaving 30 independent participants in each control group. Parents, guardians, and participating children remained naive to group assignment and the study analysis plan.

Participant Matching

After group assignment, I ranked participants in the dyslexia group by age (i.e., year : month, with days as a tiebreaker) in ascending order. Then, I ranked participants in

the age-matched group by age and matched the lists. The ranked scores and matched pairs by age can be found in Table 5.

Table 5

Age-Matched Rank and Pairs

Dyslexia participant ages	Age-matched participant ages
8.05	8.01
8.05	8.04
8.07	8.09
9.00	9.01
9.02	9.02
9.05	9.03
9.05	9.04
9.07	9.06
9.08	9.09
9.11	9.11
10.00	10.03
10.01	10.06
10.02	10.07
10.05	10.07
10.07	10.08
10.07	10.08
10.11	10.10
11.02	11.04
11.08	11.05
11.09	11.05
12.05	12.01
12.07	12.08
12.08	12.10
12.10	12.10
12.11	12.11
13.04	13.03
13.05	13.08
13.10	13.08
14.01	14.02
14.11	14.09

Note: 10.02 refers to 10 years, 2 months

Next, I ranked participants in the dyslexia group by literacy maturity, defined as the sum of raw scores on the Sight Word Efficiency (SWE) and Phonemic Decoding Efficiency (PDE) subtests from the Test of Word Reading Efficiency-2 (TOWRE-2; Torgesen, Wagner, & Rashotte, 2012). When two participants had identical scores, I ranked the participant with the lower raw score on the Letter Word Identification (LWI) subtest of the Woodcock Johnson Tests of Achievement-IV (Schrank, Mather, & McGrew, 2014a) first to indicate weaker reading performance. After ranking children in the reading-level matched group with the same method, I matched the two ranked lists. The ranked scores and matched pairs by literacy maturity can be found in Table 6.

Measures

I administered three visual measures to rule out significant visual problems; one visuospatial measure served as a proxy for nonverbal ability. I also facilitated assessment for the outcome measure, m-cell function. In addition, I administered five literacy measures to confirm and rule-out dyslexia, determine reading ability for group matches, and determine literacy maturity. A summary of measures can be found in Table 7.

Visual Rule-Out Measures

Measures of visual acuity, spatial locations, and object discrimination ruled out visual difficulties. The visual measures ensured that participants had normal or corrected-to-normal vision, the ability to judge line orientation or directionality, and the ability to discriminate visual forms.

Table 6

Reading Level-Matched Ranked Scores and Pairs

Dyslexia SWE + PDE raw scores	Reading-level SWE + PDE raw scores
20	24
37	31
38	36
39	37
39	41
41	41
43	44
52	45
53	50
53	50
54	53
58	55
60	56
61	62
62	65
65	67
68	68
68	72
71	73
73	73
75	75
76	79
80	80
81	83
86	91
95	92
100	94
101	99
104	106
112	113

Visual acuity. I measured visual acuity with the Dean Woodcock Sensory Motor Battery (DWSB; Dean & Woodcock, 2003). The Near-Point Visual Acuity subtest is one of 18 subtests that measure overall sensory and motoric function. Visual acuity measures the clarity of vision. Participants read an adapted Snellen chart with each eye to determine corrected visual acuity. This measure ruled out students with uncorrected visual difficulties, such as a child who has glasses but does not wear them or a child who may need to visit an optometrist. Average scores are 500, indicating 20/20 vision and "normal" visual acuity. Reliability for the Near-Point Visual Acuity subtest is .80. Participants had to score in the Average range or better to continue in the study; I recorded scores as pass or fail.

Line orientation. I administered line orientation, which measures directionality, as a second rule-out measure. This rule-out measure was necessary because the dependent variable (i.e., coherent motion detection) requires directionality. The Arrows subtest from the NEPSY II is a nonmotor measure of a child's ability to judge the directionality of lines (Korkman, Kirk, & Kemp, 2007). This measure of visuoperception asked children to decide which arrow among choices points to the center of a target; it is appropriate for ages 5 to 16. The reliability of the NEPSY II Arrows subtest for typically developing students is .92 for ages 5 to 6 and .75 for ages 7 to 16. For students with known disabilities, the reliability is .92 for ages 5 to 16. Participants had to score in the Average range or better to continue in the study.

Table 7

Summary of Measures

Domain	Name	Subtests	Description
Visual	Dean-Woodcock Sensory Motor Battery	Near-Point Visual Acuity	measures visual clarity
	NEPSY-II	Arrows	measures directionality and spatial locations
	Test of Visual Perceptual Skills-4	Object Discrimination	measures ventral stream object discrimination
Nonverbal ability	NEPSY-II	Block Construction	measures spatial visualization and analysis; offers a measure of nonverbal ability
M-path function (DV)	Random Dot Kinematogram	n/a	measures dorsal stream motion detection
Dyslexia diagnosis	Comprehensive Test of Phonological Processing-2	Elision	measures phonological awareness – the ability to manipulate the sounds of language
	Comprehensive Test of Phonological Processing-2	Rapid Digit and Letter Naming	measures rapid symbolic naming – the quick access to and retrieval of alphanumeric visual-verbal information
	Woodcock Johnson Test of Oral Language	Rapid Picture Naming	measures quick access to and retrieval of non-alphanumeric visual-verbal information
	Test of Word Reading Efficiency-2	Sight Word Efficiency	measures automatic real word reading
		Phonemic Decoding Efficiency	measures automatic nonword reading
	Woodcock Johnson-IV Tests of Achievement	Letter Word Identification	measures untimed single word reading
Literacy maturity (reading level matching)	Test of Word Reading Efficiency-2	Sight Word Efficiency	measures automatic real word reading; a measure of familiar automatic, consolidated phoneme-grapheme units
		Phonemic Decoding Efficiency	measures automatic nonword reading; a measure of automatic, consolidated phoneme-grapheme units without the influence of meaning

Object discrimination. Though this study focused on differences in magnocellular processing, it was critical to ensure that participants did not have deficient parvocellular processing. As the alternate projection from the visual cortex, the parvocellular pathway assists in object perception and recognition. Poor performance on object discrimination would question if parvocellular processing was an additional confound to visual and/or reading difficulty. I administered the Visual Discrimination subtest from the Test of Visual Perception Skills (TVPS-4; Martin, 2017) to confirm appropriate object discrimination. This untimed subtest assessed the ability to distinguish similarities and differences in objects. Students looked at a target picture and selected (e.g., point or say corresponding number) the matching picture below among five options. Standard scores are reported for children ages 5 to 21 years. Reliability for Object Discrimination is .91. Students had to score in the Average range or better to continue in the study and ensure that potential visual difficulties on the outcome measure were isolated to the m-cell pathway and not characteristic of a larger occipital lobe (i.e., visual lobe) disturbance.

Nonverbal Ability

In line with previous studies (see Benassi et al., 2010), I included a measure of nonverbal ability as a covariate in the group comparisons. The NEPSY II Block Construction served as a measure of performance or nonverbal ability. This measure of visuoconstruction asked children to "reproduce three-dimensional constructions from models or two-dimensional drawings" (Korkman et al., 2007, p. 23) and is appropriate for ages 3 to 16. The reliability of Block Construction is .85 for ages 5 to 6 and .78 for ages 7 to 16. During norming, the mean performance of students with reading disorders

compared to matched controls was nonsignificant ($p > .10$), suggesting that reading ability is not a significant factor in block construction.

Outcome Measure

Coherent motion detection. A coherent motion detection task measured magnocellular function. Coherent motion detection refers to the brain's ability to judge a percentage of dots moving in a similar direction among a noisy background (i.e., other dots placed randomly). Coherent motion was measured using a random-dot kinematogram (RDK). Motion detection via RDKs are the "gold standard for magnocellular processing" (Benassi et al., 2010, p. 343). The Okazo Lab built the RDK software, part of the EventIDE suite, for the purposes of this study. The following parameters were adapted from previous empirical studies that used the same algorithm for calculating coherence percentage (Talcott et al., 2002, 2003, 2013; White et al., 2006; Wilmer et al., 2004; Witton et al., 1998).

Participants sat 60 cm from a Dell Laptop screen in a darkened room. Stimuli were presented as two square patches on a Dell laptop computer. Three hundred high-luminance white dots (1 pixel) moved in each square against a black background. One of the squares had dots moving randomly in a Brownian manner. The target square had a portion of dots moving in an up/down or left/right manner at 7 degrees/second. To avoid following one dot continuously, the coherently moving dots had a lifetime of five frames.

The percentage of coherently moving dots in the target patch began at 40% but varied according to the subject's performance. The weighted up-down method was used for adaptive student response (Kaernbach, 1991). Participants responded on a touchscreen by selecting the patch that contained the moving dots with their finger. Correct responses

led to slightly lower coherence on the next trial (reduced by 1dB); incorrect responses led to slightly higher coherence on the next trial (increased by 3dB). Coherence thresholds were estimated as the geometric mean of the coherence levels from the last six of eight reversal points. The algorithm produced a staircase threshold, which estimated the lowest percentage of coherence a participant could reliably detect. Talcott et al. (2002) report inter-trial reliability of these procedures at 0.71.

Literacy Measures

The literacy measures served two purposes: (1) they confirmed and ruled out a dyslexia diagnosis; (2) they measured literacy maturity, the matching variable for the reading-level control group and dyslexia group.

Dyslexia diagnosis confirmation. I used measures of phonological awareness, rapid symbolic naming, untimed word reading, and automatic word reading to confirm and rule out dyslexia. In line with many previous researchers, I confirmed dyslexia with performance at the 10th percentile or lower on any of the measures below (Elliott & Grigorenko, 2014; Vellutino et al., 2004). I accepted performance at or below the 10th percentile on *any* measure because students with dyslexia are characterized by "difficulties with accurate and/or fluent word recognition" (Lyon, Shaywitz, & Shaywitz, 2003, p. 2). A measure of untimed isolated word reading captured accuracy difficulties; measures of rapid naming and automatic word reading captured fluency difficulties. To qualify for the control groups and rule out dyslexia, participants had to perform at or above the bottom quartile, or the 25th percentile, on three of the four measures. I also required parent report of one-to-one or small group intervention for at least six months

given the literature on the persistence of difficulties in dyslexia (Shaywitz, 2003; Vellutino et al., 2004).

Phonological awareness. I measured phonological awareness with the Elision subtest from the Comprehensive Test of Phonological Processing-2 (CTOPP-2; Wagner, Torgesen, Rashotte, & Pearson, 2013). The Elision subtest asked children to delete target sounds in 34 words with increasing difficulty. Age-normed scaled scores below the 10th percentile are 6 or lower; scaled scores above the bottom quartile are 8 or higher. Reliability for the Elision subtest is .91; the test-retest coefficient is .82.

Rapid symbolic naming. Rapid symbolic naming refers to the quick access and retrieval of verbal information, which is related to reading fluency (Wagner et al., 2013). I assessed picture names, numbers, and letter names with two subtests – the Rapid Picture Naming subtest from the Woodcock Johnson Test of Oral Language (WJ OL; Shrank, Mather, & McGrew, 2014b) subtest and the Rapid Digit Naming subtests from the CTOPP-2 (Wagner et al., 2013). Each subtest asked children to name pictures, numbers, or letters in an array as fast as possible; each array contained only one type of stimulus. Using the test developed norms, I converted total time to an age-normed standard score. Scores at or below the 10th percentile are 81 or lower; scores above the bottom quartile are 90 or higher. Reliability for the CTOPP-2 Rapid Symbolic Naming composite is .85 and the test-rest coefficient is .91. Reliability for the WJ OL Rapid Picture Naming subtest is .90.

Untimed word reading. I measured untimed, single-word reading with the Letter Word Identification subtest from the Woodcock Johnson-IV Test of Achievement (WJ-IV ACH; Schrank et al., 2014a). In this test, lists of words are presented for participants

to read aloud. Participants continued to read lists of words with increasing difficulty until they read six words in a row incorrectly. The test software converted word reading to an age-normed standard score. Scores at or below the 10th percentile are 81 or lower; scores above the bottom quartile are 90 or higher. Reliability for Letter Word Identification ranges from .88 to .96 for ages 6 to 14.

Timed word reading. I measured timed, single-word reading with the Sight Word Efficiency (SWE) subtest from the Test of Word Reading Efficiency (TOWRE-2; Torgesen, Wagner, & Rashotte, 2012). I measured timed, single-nonword reading with the Phonemic Decoding Efficiency (PDE) subtest from the TOWRE-2. Students read real words or nonwords as fast as possible for 45 seconds. Using test developed norms, I converted scores to age-normed standard scores. Scores at or below the 10th percentile are 81 or lower; scores above the bottom quartile are 90 or higher. As the test is timed, the authors state that split-half coefficients are not appropriate. Test-retest for SWE is .91, for PDE is .92, and for TWRE is .95.

Reading-level matching. I matched participants with dyslexia to younger peers on literacy maturity, or automatic, consolidated knowledge of phoneme-grapheme units (i.e., sound-letter units). I chose the TOWRE-2 as a measure of literacy maturity for three reasons. First, the TOWRE-2 provides a measure of familiar phoneme-grapheme units in the Sight Word Efficiency (SWE) subtest, which serves as a proxy of reading experience (i.e., many reading experiences will build a sight word vocabulary). Second, the Phonemic Decoding Efficiency (PDE) subtest reflects knowledge of unfamiliar phoneme-grapheme units without the influence of vocabulary. Third, the SWE and PDE measure automaticity, which is a reflection of maturity with phoneme-grapheme units. I used the

sum of raw scores from the PDE and SWE subtests to match participants with dyslexia to younger peers. To confirm matches when two participants scored identically on the TOWRE, I used raw scores from the WJ-IV ACH Letter Word Identification subtest. Therefore, the TOWRE-2 and WJ-IV ACH served two purposes – I used standard scores to confirm or rule out dyslexia diagnosis and I used raw scores to match participants in each group.

Reliability

With IRB approval (see Appendix C), I audio recorded each assessment session on a SONY ICD-PX470 Stereo Digital Voice Recorder. During data collection, I stored files on a password-protected USB drive. Within 48 hours of testing a participant, I listened to the recording and checked scores for the TOWRE-2, chosen because children often read words very fast and have phonetically similar substitutions for the nonwords (e.g., *mib* or *nip* for *mip*). In addition, the matching design relied on accurate scores from the TOWRE-2, so listening to each participant twice captured the correct responses as often as possible.

Following data collection, an independent scorer with doctoral-level training administering assessments re-scored 20% of the protocols (18 participants). I assigned participants in each group (e.g., dyslexia, age-matched, and reading level-matched) a number 1 to 30; then I randomly selected six participants in each group for re-scoring using a random number generator. During re-scoring, the second scorer listened to the selected audio recordings and marked correct scores for each subtest; we then compared the second set of protocols to the original scoring sheets. Two subtests could not be re-scored using audio recording because they required looking at the participant: (1)

DWSMB Near-Point Visual Acuity and (2) NEPSY II Block Construction. A software program scored the dependent variable, m-cell function, making it inherently free from examiner bias. Therefore, the measures available for re-scoring included: (1) CTOPP-2, (2) TOWRE-2, (3) NEPSY-II Arrows, (4) TVPS-4, (5) WJ-IV ACH, (6) WJ OL.

I measured inter-rater reliability with percent agreement, defined as the number of ratings that were in agreement divided by the total number of ratings (Lange, 2011). This study had 99% agreement. A total of nine disagreements were found – five on the TOWRE-2 nonsense word reading (i.e., PDE), two on the Elision subtest from the CTOPP-2, one on the TVPS-4, and one on the WJ-IV LWI subtest. I replayed each instance of disagreement on the audio device until scorers reached consensus. Importantly, none of the errors resulted in a participant failing to meet inclusionary criteria.

Analytic Plan

In this study, I explored differences in m-cell function between children with dyslexia and typically-developing peers. The following research questions guided this study:

1. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar ages, controlling for nonverbal ability?
2. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar levels of literacy maturity, controlling for nonverbal ability?

After exploring group performance via descriptive statistics with appropriate visuals, I used multiple regression to quantify differences in m-cell function across

groups, accounting for nonverbal ability. Multiple regression is an appropriate method for research questions that investigate relationships between dependent and independent variables while accounting for omitted variable bias (Gordon, 2015).

The final additive regression model was:

$$Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(nonverbal\ ability)_i + \varepsilon_i,$$

where m-cell function served as a continuous dependent variable (Y_i). Group membership served a categorical independent variable with three levels – dyslexia, age-matched, or reading-level matched; dummy codes classified membership across the groups. For example, I coded participants in the age-matched group [1 0 0] to signal membership in the age-matched group. Likewise, I coded participants in the dyslexia group as [0 1 0] to specify membership in the dyslexia group but not the age-matched group or the reading level-matched group. The dyslexia group served as the reference category or omitted group in the model because the research questions focused on comparing both the age-matched and reading level-matched group to the dyslexia group. Previous investigations have unequivocally found a relationship between m-cell function and age in typically developing children (see Taylor et al., 2018), so I did not compare the age-matched group to reading-level matched group in this study. The models for each group are below:

Dyslexia: $Y_i = \beta_0 + \beta_3(nonverbal\ ability)_i + \varepsilon_i$

Age matched: $Y_i = (\beta_0 + \beta_1) + \beta_3(nonverbal\ ability)_i + \varepsilon_i$

Reading level: $Y_i = (\beta_0 + \beta_2) + \beta_3(nonverbal\ ability)_i + \varepsilon_i$

Nonverbal ability served as an additional independent variable in the model to account for any variance in m-cell function due to differences in nonverbal ability. I explored biological gender as a potential predictor, but this study confirmed previous

findings that gender did not significantly predict m-cell function (see Stein, 2019). I also calculated measures of fit, including adjusted R-squared and Root Mean Square Error, to determine how well the data fit the model. Finally, I conducted hypothesis tests to identify any statistically significant differences among group m-cell performance.

Hypotheses

Dyslexia vs age-matched controls. The first individual slopes test explored the effect of group membership (dyslexia versus age-matched controls) on m-cell function, controlling for nonverbal ability. The null hypothesis, $H_0 = \beta_1 = 0$, states there is no difference in m-cell function between the dyslexia and age-matched groups, holding nonverbal ability constant. The alternative hypothesis, $H_1 = \beta_1 \neq 0$, states there is a significant difference in mean m-cell function between the dyslexia and age-matched groups, holding nonverbal ability constant. Practically, a rejection of the null hypothesis suggests that individuals with dyslexia have a significant difference in m-cell ability compared to typically-developing peers of the same age. Many researchers have found this difference but have used results of this one comparison to suggest that dyslexics have deviant m-cell function that could be captured as a diagnostic sign during diagnosis (i.e., there is something unique about the m-cell function in dyslexics that could point directly to a diagnosis).

Dyslexia vs reading level-matched controls. The second individual slopes test also explored the effect of group membership (dyslexia versus reading level-matched controls) on m-cell function, controlling for nonverbal ability. The null hypothesis, $H_0 = \beta_2 = 0$, states there is no difference in m-cell function between the dyslexia and reading level-matched groups, holding nonverbal ability constant. The alternative hypothesis,

$H_1 = \beta_2 \neq 0$, states there is a significant difference in mean m-cell function between the dyslexia and reading level-matched groups, holding nonverbal ability constant. Practically, a rejection of the null hypothesis suggests that m-cell function in children with dyslexia is significantly different than typically developing peers with similar levels of literacy maturity (i.e., automatic analysis of letter-sound units). In other words, rejecting the null hypothesis means that dyslexics have a unique pattern of m-cell function that cannot be explained by their levels of literacy acquisition.

Nonverbal ability. A third individual slopes test explored the effect of nonverbal ability on m-cell function, controlling for group membership. The null hypothesis, $H_0 = \beta_3 = 0$, states there is no relationship between nonverbal ability and m-cell function, accounting for group membership. The alternative hypothesis, $H_1 = \beta_3 \neq 0$, states there is a significant relationship between nonverbal ability and m-cell function, accounting for group membership. This test is not directly related to the research questions for this study, but it could be important to consider a possible relationship for future study design.

Summary. Rejecting both the age-matched and reading-level matched null hypotheses suggests that m-cell function may be a way to differentiate those with dyslexia from peers, pointing to a specific and sensitive diagnostic sign. Such a sign would require that dyslexic children have unique m-cell function unlike typically developing children at similar stages of maturation – including maturation by age and maturation by literacy development. Failing to reject both the age and reading level null hypotheses questions the inclusion of m-cell function in a diagnostic model because it may not add any precision to a dyslexia diagnosis beyond the current literacy-based model.

Limitations

This analytic plan answered the research questions posed in this study, but the plan had limitations. First, I did not include information on the socioeconomic and race/ethnicity of participants. Second, the analysis included small sample sizes (n=30) in each group. Finally, the results of this analytic plan could not infer causality. Multiple regression models quantified the relationship between group membership and m-cell performance while controlling for nonverbal ability, but this analysis could not provide evidence for what causes various levels of m-cell function. Rather, in this observational study, my goal was to explore and gather important variables related to a population of interest. Specifically, I aimed to explore if m-cell function was related to membership as dyslexic.

CHAPTER IV

RESULTS

In this observational study I investigated magnocellular (m-cell) function in children with dyslexia and typically-developing peers. Group assignment methods accounted for possible pre-existing group differences by matching each dyslexic child to a peer of the same age and to a younger peer with similar literacy maturity (i.e., similar automaticity and consolidation of letter-sound units). Multiple regression quantified differences in m-cell function of children with dyslexia compared to age-matched peers and reading-level matched peers. In addition, the regression model explored possible differences in m-cell function by group due to nonverbal ability and gender. Results are presented after a short discussion of data inspection procedures and descriptive statistics.

Data Inspection

I checked for data entry errors by scanning each participant's protocols line by line and checking scores against the entered data. Three errors were found and corrected. Then I inspected the data for missing cases; every participant had scores for each measure.

Descriptive Statistics

Group Characteristics

Characteristics of each group and performance on the visual and literacy measures can be found in Table 8. Data for the rule-out measure Visual Acuity are not presented

because scoring followed a pass/fail dichotomy; participants who failed did not qualify for inclusion in the final analysis. The dyslexia group and typically-developing age-matched controls had nearly identical ages and mean grades; per the design, the reading-matched controls were younger, typically-developing children ages six to eight. Multiple one-way ANOVA comparisons with post hoc contrasts and Bonferroni adjustments revealed significant literacy differences among the groups.

The age-matched older children and reading-matched younger children had similar performance when comparing age-normed standard scores on the following measures: Phonemic Decoding Efficiency (PDE), Sight Word Efficiency (SWE), Test of Word Reading Efficiency Composite (TWRE), Elision, Rapid Symbolic Naming (RAN), Rapid Picture Naming, and Letter Word Identification (LWI). Both typically-developing control groups (e.g., age-matched and reading-matched) had significantly better literacy performance when comparing age-normed standard scores to the dyslexia group. However, the reading-matched group and dyslexia group had similar literacy performance when comparing z-scores on the following tasks: Elision (i.e., phonological deletion), LWI (i.e., untimed word reading), SWE (i.e., timed sight word reading), and Literacy Maturity (i.e., sum of raw PDE and SWE). Overall, the dyslexia and reading-matched groups had significantly different performance on three literacy measures: (1) PDE z-score, $t(58) = -2.80$, $p = .007$; (2) RAN standard score, $t(58) = -7.14$, $p < .001$; and (3) Rapid Picture Naming standard score, $t(58) = -5.52$, $p < .001$, with the younger, typically developing children scoring higher in all cases. Notably, the dyslexia group still scored in the Average range on Rapid Picture Naming despite a significant difference compared to all controls. None of the groups had significant differences on the visual

measures or nonverbal ability measures. Overall, each group's scores aligned with the greater population of typically-developing children ages 6 to 14 and dyslexic children ages 8 to 14, with all children's nonverbal abilities slightly above the expected mean.

Table 8

Descriptive Statistics by Group

Variables	Dyslexia	Age-Matched Controls	Reading-Matched Controls
Demographic Characteristics			
Age	10.63 (1.78)	10.63 (1.78)	7.02 (0.75)***
Grade	5.27 (1.87)	5.20 (1.99)	1.50 (0.68)***
Female	16	13	17
Male	14	17	13
Visual-Spatial Abilities			
Line Orientation	9.63 (1.78)	10.57 (2.16)	10.43 (1.78)
Object Discrimination	10.57 (1.74)	11.63 (2.03)	10.93 (2.15)
Nonverbal Ability	11.93 (2.26)	12.47 (2.52)	12.30 (2.63)
Literacy Abilities			
PDE	69.60 (6.48)***	104.50 (8.89)	100.30 (10.38)
SWE	78.00 (12.55)***	104.23 (9.13)	104.70 (11.05)
TWRE	72.37 (9.30)***	104.63 (8.55)	102.80 (10.64)
Literacy Maturity	-0.52 (0.71)	1.05 (0.58)***	-0.53 (0.73)
Elision	6.63 (1.99)***	10.37 (1.56)	10.90 (1.88)
Rapid Symbolic Naming	81.70 (12.43)***	101.40 (12.29)	101.67 (8.94)
Rapid Picture Naming	92.73 (9.90)***	103.73 (8.72)	106.20 (8.98)
LWI	78.90 (9.26)***	109.60 (8.61)	104.50 (10.32)
N	30	30	30

Note. M(SD) reported for each measure; PDE = Phonemic Decoding Efficiency; SWE = Sight Word Efficiency; TWRE = Total Word Reading Efficiency; Literacy Maturity = z-score of raw PDE + SWE; Rapid Symbolic Naming = composite standard score of rapid letter and digit naming; LWI = Letter Word Identification

***$p < .001$.

Dyslexia group literacy performance. To better understand the target sample, detailed testing results for the dyslexia group are presented in Figure 9. Out of 30

children, 21 scored in the below average range or worse on a measure of phonological deletion (i.e., Elision); almost half scored in the poor or very poor range. Sixteen children qualified as having a double deficit in phonological processing, or at least below average performance in both phonological awareness (i.e., Elision) and Rapid Symbolic Naming. Only one child demonstrated no phonological processing difficulty by scoring in the average range on both Elision and Rapid Symbolic Naming; this child performed in the poor range on a measure of timed nonword reading (i.e., PDE).

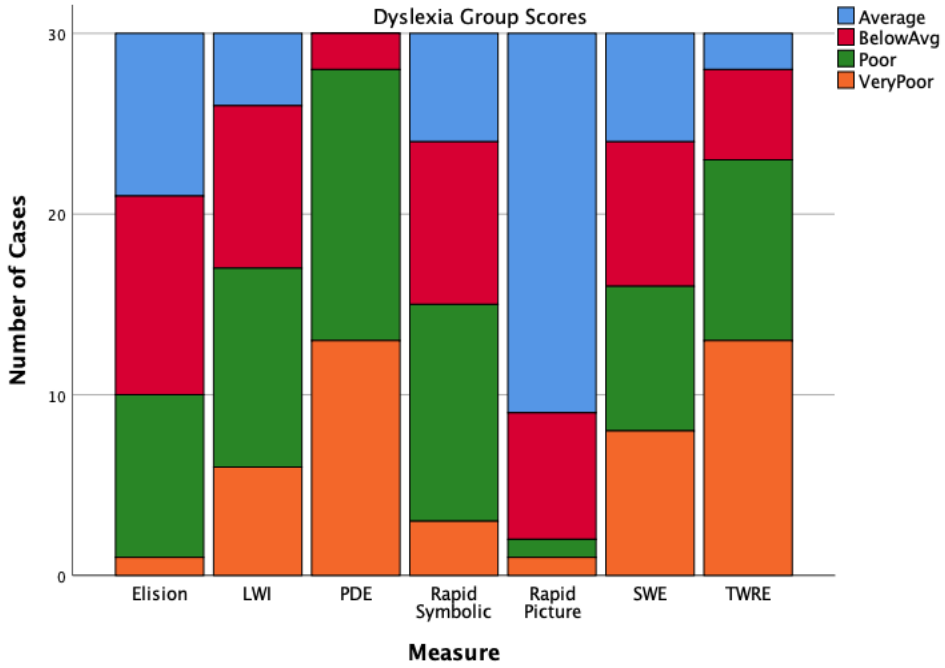

Figure 9. Dyslexia Group Scores
Note. LWI = Letter-Word Identification; PDE = Phonemic Decoding Efficiency; SWE = Sight Word Efficiency; TWRE = Total Word Reading Efficiency

Interestingly, children with dyslexia performed differently across measures of rapid naming. Twenty-four children scored in the below average range or worse on Rapid Symbolic Naming (i.e., Rapid Digit Naming; Rapid Letter Naming), whereas 21 children scored in the average range on Rapid Picture Naming.

When reading real words without timing constraints (i.e., LWI), six children with dyslexia scored in the average range. When timing pressures were added to real word reading (i.e., SWE), four children scored in the average range. In contrast, zero children scored in the average range on timed nonword reading (i.e., PDE), and 28 out of 30 children scored in the poor or very poor range. The two children who scored in the below average range for PDE scored at the 9th and 10th percentiles. The composite standard score for timed real and nonword reading (i.e., TWRE) reflected the difficulty with timed nonword reading, with 24 children scoring in the poor or very poor range.

Outcome Variable

Descriptive statistics for the outcome variable are reported by group in Table 11. A side-by-side box plot shows average m-cell function by group (Figure 10).

Table 9

M-Cell Descriptive Statistics by Group

Variables	N	*M(SD)*	Min.	Max.
Dyslexia Group	30	23.96 (11.55)	9.50	42.67
Age-matched Group	30	17.46 (6.12)	5.00	32.67
Reading Level-matched Group	30	25.52 (11.15)	5.33	44.67

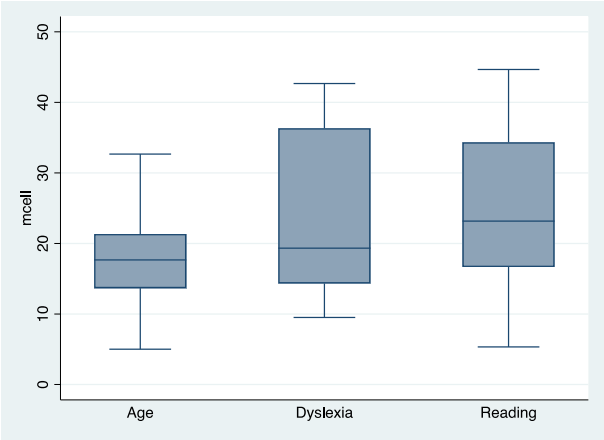

Figure 10. Box Plot of M-Cell Function by Group

M-cell function at lower coherence percentages indicates better performance, and coherence at higher levels indicates weaker performance. Coherence levels describe the percentage of moving dots among distractor dots needed to reliably detect any dots moving together. The age-matched group had the lowest mean coherence at 17.46% (*SD*=6.12), followed by the dyslexia group at 23.96% (*SD*=11.55), and finally the reading level-matched group at 25.52% (*SD*=11.15). Notably, each group had a wide range of scores, from coherence in the single digits to six to eight times that on the upper end, with the age-matched group having the least variability from the 25th to 75th percentile.

I also examined m-cell function across the school-age years. Average coherence percentages by grade for all typically-developing children (i.e., age-matched and reading-matched groups) can be found in Figure 11. Group trajectories through the school-aged years can be examined in Figure 12.

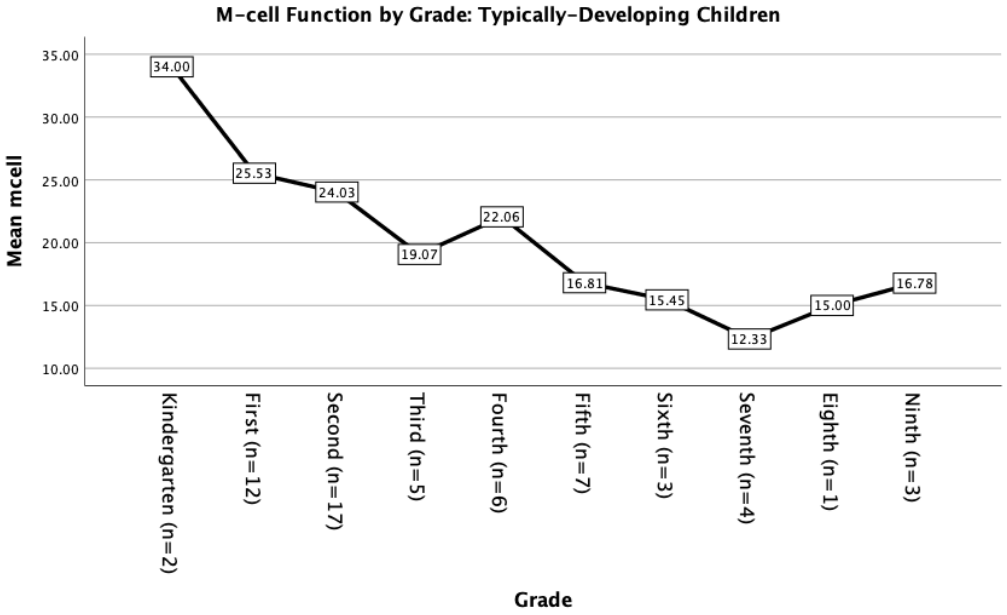

Figure 11. M-cell Function Trajectory: Typically-Developing Children (*N* = 60)

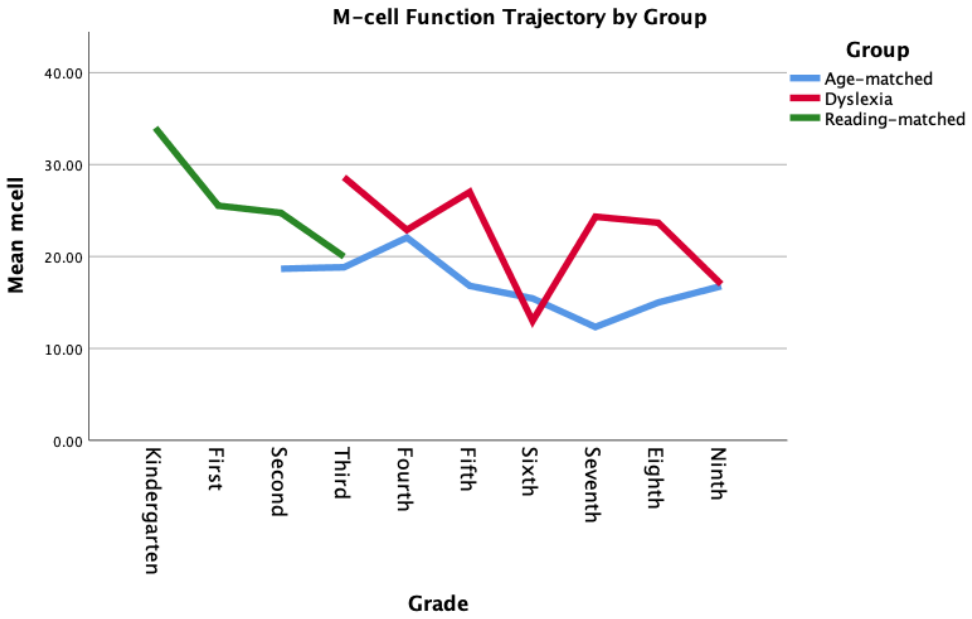

Figure 12. M-cell Trajectory by Group (*N* = 90)

In typically-developing children, a decreasing trend marking improvement in m-cell function can be seen across the school-age years. There also appears to be considerable improvement in m-cell function from kindergarten to third grade compared to the improvement from third grade to ninth grade. In children with dyslexia, there appears to be inconsistent m-cell performance across the school-age years.

Correlations

Correlations among the measures are presented in Table 10. M-cell function had small yet significant negative associations with Age (r = -.27) and Object Discrimination (r = -.23). Importantly, Literacy Maturity and m-cell function had a small yet significant negative association (r = -.31). M-cell function and nonverbal ability had a weak, nonsignificant association (r = -.18).

In addition, the visual rule-out measures, Object Discrimination and Line Orientation, were significantly associated with the measure of Nonverbal Ability (r = .44 and r = .39, respectively). Interestingly, the visual measures Object Discrimination and

Line Orientation had small yet significant associations with the phonological measure Elision, a test of sound deletion (r = .30 and r = .28, respectively). The literacy measures – Elision, Rapid Symbolic Naming, Rapid Picture Naming, LWI, SWE, PDE, Literacy Maturity, and TWRE, had significant intercorrelations, ranging from small to large.

Summary of Descriptive Statistics

Overall, the dyslexia group, age-matched group, and reading level-matched group had similar visual and nonverbal abilities. Similar in age, the dyslexia and age-matched groups differed among reading abilities. On the other hand, the dyslexia and reading level-matched groups were similar in literacy maturity, single word reading, and timed single word reading but different in age, timed nonword reading z-score, and rapid naming (e.g., symbolic naming and picture naming). All but one participant in the dyslexia group showed phonological processing (i.e., Elision and/or Rapid Symbolic Naming) difficulties and every participant in the dyslexia group scored outside of the average range on a measure of timed nonword reading (i.e., PDE). Finally, typically-developing children's m-cell function appeared to improve over the school-age years with a sharp improvement from the kindergarten to third grade years. In contrast, the dyslexia group appeared to have inconsistent m-cell growth over time.

Table 10

Correlations Among Measures (N = 90)

Variables	1	2	3	4	5	6	7	8	9	10	11	12
1. M-cell function	–											
2. Age	-.27*	–										
3. Nonverbal Ability	-.18	.05	–									
4. PDE	-.12	-.22*	.15	–								
5. SWE	-.08	-.19	.04	.89**	–							
6. TWRE	-.10	-.22*	.10	.97**	.97**	–						
7. Literacy Maturity	-.31**	.57**	.12	.58**	.59**	.61**	–					
8. Elision	-.04	-.28**	.21*	.76**	.64**	.72**	.33**	–				
9. Rapid Symbolic Naming	-.02	-.24*	-.09	.70**	.75**	.75**	.39**	.44**	–			
10. Rapid Picture Naming	-.09	-.25*	.07	.53**	.58**	.57**	.23*	.31**	.68**	–		
11. LWI	-.16	-.14	.17	.90**	.84**	.90**	.62**	.74**	.61**	.47**	–	
12. Line Orientation	-.08	-.18	.39**	.22*	.14	.19	.03	.28**	.05	.08	.24*	–
13. Object Discrimination	-.23*	.02	.44**	.19	.10	.15	.10	.30**	-.01	.00	.14	.45**

Note. PDE = Phonemic Decoding Efficiency; SWE = Sight Word Efficiency; TWRE = Total Word Reading Efficiency; Literacy Maturity = z-score of raw PDE + SWE; Rapid Symbolic Naming = composite standard score of rapid letter and digit naming; LWI = Letter Word Identification

*$p < .05$. **$p < .01$. ***$p < .00$

Inferential Statistics

Testing Assumptions

To confirm multiple regression as an appropriate analysis for this study, I analyzed the five assumptions of multiple regression prior to running the final analysis – linearity, homoscedasticity of the residuals, independence, normality of the residuals, and the absence of multicollinearity. I investigated linearity with scatterplots of the *Y* regressed on the *X* axis at each level of the grouping variable; the plots showed weak linear relationships without curvilinear trending. A plot of predicted m-cell values and studentized residuals suggested a slight violation of the homoscedasticity assumption, but the Breusch-Pagan test contradicted a violation ($p = .095$). To further investigate a possible violation, I ran White's test for heteroskedasticity, which confirmed a violation ($p < .05$). Therefore, robust standard errors were used during the regression analysis.

Although the design did not include random sampling, observations did not impact one another, confirming the assumption of independence. A residual Q-Q plot suggested slight deviation from normality; however, the residuals plot of predicted m-cell values and studentized residuals confirmed normality. Finally, I examined variance inflation factors to rule out the presence of multicollinearity with all values less than four.

A Priori Model Selection

Multiple regression explored the relationship between m-cell function and group membership, holding nonverbal ability constant. Nonverbal ability was centered around its mean to allow for better interpretation of the constant term. As specified a priori, the initial model was extended to explore biological gender as an additional categorical predictor variable (i.e., "female"), where male = 0 and female = 1. The two models were

1. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(nonverbal\ ability)_i + \varepsilon_i$

2. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(nonverbal\ ability)_i + \beta_4(female)_i + \varepsilon_i$

where Y_i represented m-cell function, measured by percent coherence. The results of each model can be seen in Table 11.

Table 11

Model Selection: M-cell Function

	Model 1			Model 2		
Variable	*b*	*SE b*	*b**	*b*	*SE b*	*b**
Age-Matched	-6.13*	2.43	-.28	-6.29*	2.42	-.29
Reading-Matched	1.82	2.88	.08	1.90	2.89	.08
Nonverbal Ability	-0.70	0.42	-.16	-0.75	0.42	-.18
Female				-1.92	2.06	-.09
Dyslexia [Constant]	23.68***	2.11		24.68***	2.35	
adjusted R^2		.11			.11	
RMSE		9.83			9.84	
F		6.56			5.65	
N		90			90	

*$p < .05$. **$p < .01$

Model two, which includes the categorical variable to represent biological gender, did not significantly change the standard errors, coefficients, individual slopes tests, overall F-test, or the measures of fit (e.g., adjusted R-squared, Root Mean Squared Error). Therefore, model one was selected as the final model to answer the research questions as biological gender did not appear to explain additional variance in m-cell function across groups. Results of model one were also robust to alternative models, including a model that removed suspected outliers and a robust regression model (see Table D1).

Research Questions

The final model (i.e., model one in Table 11) addressed the following research questions:

1. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar ages, controlling for nonverbal ability?
2. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar levels of literacy maturity, controlling for nonverbal ability?

Individual slopes tests quantified the relationship between m-cell function and group membership, controlling for nonverbal ability. Seen in model one of Table 11, the average m-cell score for dyslexics with average nonverbal ability was 23.68% coherence. Coherence for the age-matched group averaged 6.13 percentage points lower (i.e., better) than the dyslexia group, holding nonverbal ability constant, $p = .013$. Coherence for the reading-matched group averaged 1.82 percentage points higher (i.e., worse) than the dyslexia group, holding nonverbal ability constant, $p = .530$. Therefore, there was a significant difference in m-cell function between the dyslexia and age-matched groups but not the dyslexia and reading level-matched groups when accounting for nonverbal ability. Though the individual slopes test for nonverbal ability was nonsignificant, the model predicted that increasing nonverbal ability by one scaled score is associated with a 0.70 decrease (i.e., improvement) in m-cell coherence, holding group membership constant.

Measures of fit included adjusted R-squared and Root Mean Squared Error. The final model explained 11% of the variance in m-cell function, and the average size of a residual was approximately one standard deviation of m-cell coherence.

Exploratory Analyses

Multiple regression examined group differences, but wide variability among lower literacy maturity children warranted further analyses. As a result, I also explored variation within the dyslexia group and examined m-cell performance in all lower literacy children (i.e., dyslexia and reading-matched children). Finally, I explored the surprising possibility of timed nonsense word reading as a potential diagnostic sign of dyslexia.

Dyslexic variation and phonological skill. In the first analysis, I explored the variability in m-cell function within the dyslexia group. Twelve children from the dyslexia group scored the same as or better than the typically-developing average (17.46% coherence), which was somewhat surprising given the previous literature on m-cell function. In this analysis, I explored phonological skill as a potential contributor to the variability in m-cell function across the dyslexia group. I questioned if dyslexics who scored the same as or better than the typically-developing average (17.46%) had better phonological ability than the dyslexics who scored worse on m-cell function. After sorting children with dyslexia by phonological ability (e.g., Average, Below Average, Poor), I further separated them by Average or Below Average m-cell function, using 17.46% as the threshold. Within the dyslexia group, children with strengths in m-cell function did not necessarily have similar strengths in phonological ability, measured as phonological deletion (i.e., Elision). In fact, phonological abilities were equally distributed across ability levels within the 12 children with dyslexia who had better than

average m-cell function. Similarly, phonological ability varied among the less skilled m-cell performers with dyslexia. A detailed description of m-cell function by phonological ability can be found in Table D2. An independent samples t-test confirmed a nonsignificant difference in phonological ability between dyslexic children with average or better m-cell ability and dyslexic children with below average m-cell ability, $t(28) = .630$, $p = .534$, suggesting an independence of m-cell function and phonological ability in children with dyslexia.

Within group variability. Although the results of the multiple regression model suggested that the dyslexia and reading-matched groups had similar m-cell ability, a closer visual inspection of participant performance within each group revealed considerable variability. Figure 13 shows m-cell function and literacy maturity by group, with each quadrant of the plot labeled (e.g., I, II, III, IV). M-cell function was centered at the mean of all participants ($M = 22.31\%$ coherence); literacy maturity was already centered as a z-score. As a reminder, lower m-cell coherence indicates better performance and higher literacy maturity indicates better performance. The scatterplot revealed four patterns. First, quadrant I was mostly empty, meaning very few children had poor m-cell function and good literacy maturity. Second, quadrant II had only children with dyslexia and younger, typically-developing children matched on reading level, meaning children with lower literacy ability had poor m-cell function. Third, quadrant III had nearly all children with dyslexia and younger typically-developing children matched on reading level, meaning that children with lower literacy ability could also have good m-cell ability. Fourth, quadrant IV had primarily typically-developing children matched to the

dyslexia group on age, meaning that children with higher literacy maturity had better m-cell function.

Visual inspection of the plot suggests that a divide in the plot occurs at approximately m-cell = 5.5 (i.e., 27.81% coherence). Each side of the divide appears to house a cluster of children with dyslexia and younger typically-developing children matched on reading level – one cluster mostly in the upper bounds of quadrant II and one cluster in quadrant III that barely extends into quadrant II. Quadrants I and IV did not appear to have a similar dividing line because there was less variability within the age-matched group.

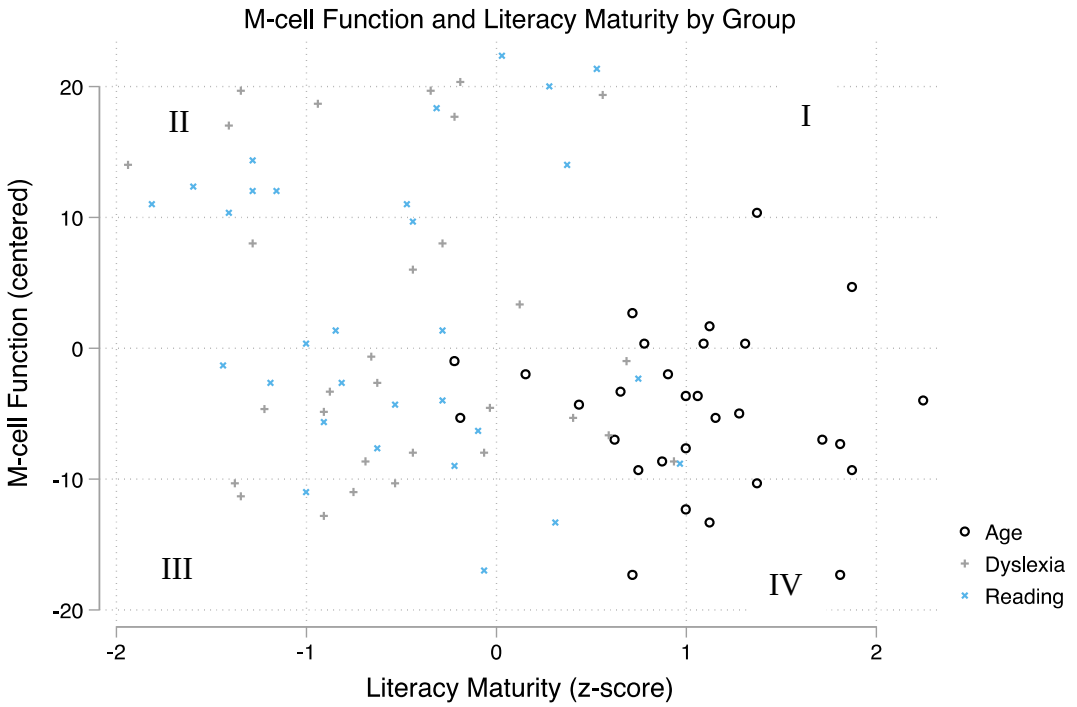

Figure 13. M-cell Function and Literacy Maturity by Group. M-cell function is centered around the sample mean at 27.81% coherence.

Using 28% coherence (i.e., approximately m-cell = 5.5 in Figure 13) as a threshold, the "good" m-cell cluster (*M* = 16.42%; mostly quadrant III) included 24 children from the dyslexia and reading-matched groups; the "poor" m-cell cluster (*M* = 37.21%; quadrant

II) included 36 children from the dyslexia and reading-matched groups. Within the "good" m-cell cluster, 19 of 24 children were from the dyslexia group; within the "poor" m-cell cluster, 25 of 36 children were from the reading-matched group. Gender did not appear to be a significant factor within or between the clusters. The "good" and "poor" m-cell clusters significantly differed on m-cell ability, t (58) = 16.72, $p < .001$.

Independent samples t-tests investigated if the "poor" cluster above the hypothetical threshold ($n = 36$) significantly differed from the "good" cluster below the threshold ($n = 24$) on any of the following variables: Age (years, months), Nonverbal Ability, Directionality, Object Discrimination, Elision, Rapid Symbolic Naming, Rapid Picture Naming, Letter Word Identification, Sight Word Efficiency, Phonemic Decoding Efficiency, Total Word Reading Efficiency, and Literacy Maturity (z-score). Results indicated that the two clusters scored similarly across all literacy measures in the study ($p > .05$) despite a significant difference in m-cell function. The two clusters differed on z-scores for visual and nonverbal abilities (e.g., Directionality, Object Discrimination, and Nonverbal Ability), with the "good" cluster scoring significantly higher. However, results of an ANCOVA for m-cell function by cluster, controlling for Directionality, Object Discrimination, and Nonverbal Ability revealed that m-cell function significantly differed between groups, even after accounting for visual and nonverbal abilities, F (1, 55) = 234.766, $p < 0.001$, $\eta_p^2 = 0.81$. It appeared that something outside of the visual and literacy measures collected in this study was driving the differences in m-cell function between children at lower literacy levels.

Timed nonword reading. Due to the surprising universal difficulty with timed nonword reading in the dyslexia group, a final exploratory analysis investigated timed

nonword reading as a potential diagnostic sign of dyslexia, asking whether differences between the dyslexia group and reading-matched group were meaningful even after accounting for other literacy skills (e.g., speeded sight word knowledge, phonological knowledge). A significant finding when comparing the dyslexia to age-matched and dyslexia to reading-matched groups on timed nonword reading would suggest that children with dyslexia score worse than all typically-developing peers, pointing to a potential diagnostic sign of dyslexia. That is, timed nonword ability may be a way to differentiate children with dyslexia from other poor readers. This analysis process mirrored the previous m-cell analysis which evaluated the usefulness of m-cell function as a diagnostic sign of dyslexia.

Descriptive statistics. Descriptive statistics for the outcome variable (timed nonword reading) are reported by group in Table 12.

Table 12

Timed Nonword Descriptive Statistics by Group

Variables	N	*M(SD)*	Min.	Max.
Dyslexia Group	30	-0.76 (0.47)	-0.32	2.52
Age-matched Group	30	1.12 (0.65)	-1.61	0.22
Reading Level-matched Group	30	-0.36 (0.62)	-1.14	1.24

Note. Scores reported are z-scores, or the number of nonwords read correctly in 45 s. Standard scores for each group are reported in Table 8 as PDE. The dyslexia group read 13.57 words on average (*SD* = 6.96). The age-matched group read 41.30 words on average (*SD* = 9.62). The reading-matched group read 19.47 words on average (*SD* = 9.20).

On average, the age-matched group read 41.30 nonwords accurately in 45 s (*SD*=9.62), which equates to a z-score average of 1.12 (0.65). The reading-matched group averaged 19.47 (*SD*=9.20) nonwords in 45 s, equivalent to a z-score of -0.36 (*SD*=0.62). The dyslexia group averaged 13.57 (*SD*=6.96) nonwords in 45 s, equivalent to a z-score of -0.76 (*SD*=0.47). The age-matched group had the widest range of scores despite having

an identical age range as the dyslexia group. Timed nonword reading trajectories by group can be seen in Figure 14.

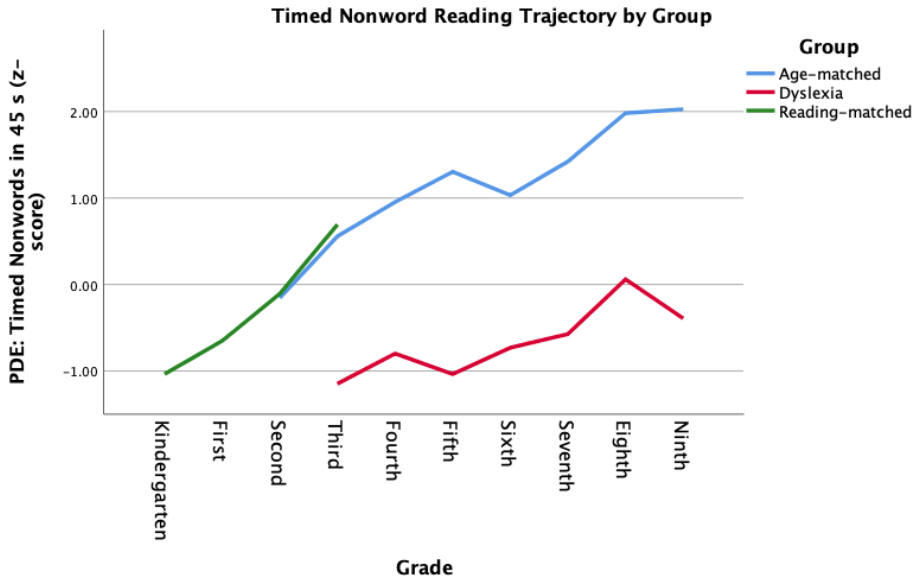

Figure 14. Timed Nonword Reading Trajectory by Group (N = 90)

In typically-developing children, an increasing trend marking improvement in timed nonword reading can be seen across the school-age years. In children with dyslexia, there appears to be an increase in timed nonword reading across the school-age years, but the rate of progress from third to ninth grade did not approximate the rate of progress for typically-developing children. In addition, the gap between dyslexics and typically-developing peers does not appear to close over time.

Testing assumptions. I checked the multiple regression assumptions prior to running the exploratory analysis for timed nonword reading and confirmed the assumptions after selecting the final model. I investigated linearity with scatterplots of the *Y* regressed on the *X* axis at each level of the grouping variable; the plots showed mostly linear relationships, with the reading-matched group showing a possible, very slight trend toward curving at the lowest values of SWE and PDE. However, this trend could be an artifact of the very small sample size at this end of the continuum. A plot of

predicted timed nonword reading values and studentized residuals did not indicate a violation of linearity or the homoscedasticity assumption, and the Breusch-Pagan test confirmed homoscedasticity (p = .30). Although the design did not include random sampling, observations did not impact one another, confirming the assumption of independence. A residual Q-Q plot suggested slight deviation from normality; however, the residuals plot of predicted timed nonword reading values and studentized residuals confirmed normality. Finally, I examined variance inflation factors to rule out the presence of multicollinearity with all values less than 4.25.

Model building. Multiple regression models explored the differences in timed nonword reading (z-scores) between the dyslexia and age-matched group and the dyslexia and reading-matched group, controlling for various literacy skills. The a priori progression of adding predictor variables was: (1) include grouping variables Age Matched and Reading Matched, which are identical to the groups in the m-cell analysis; (2) add Rapid Symbolic Naming (RAN) to account for temporal speed; (3) add Letter Word Identification z-score (zLWI) plus RAN to account for untimed sight word reading ability and speed; (4) add Sight Word Efficiency z-score (zSWE) instead of RAN + zLWI to test a difference in RAN + zLWI and zSWE as both measure timed sight word reading; (5) add Elision z-score to either RAN + zLWI or zSWE to account for untimed phonological deletion ability. The dyslexia group served as the reference group in all models. The outcome variable, timed nonword reading, was measured with Phonemic Decoding Efficiency z-score (zPDE). The equations for each model were:

1. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \varepsilon_i$
2. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(RAN)_i + \varepsilon_i$
3. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(RAN)_i + \beta_4(zLWI)_i + \varepsilon_i$

4. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(zSWE)_i + \varepsilon_i$
5. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(zSWE)_i + \beta_4(zElision)_i + \varepsilon_i$

Model five was chosen as the best model in the first phase of model building (see Table D3) – the predictor variables appeared significantly related to timed nonword reading, the standard errors remained consistent across the models, and zSWE provided simplicity compared to RAN + zLWI when including a term to account for timed sight word reading ability. After choosing model five, I explored if the relationship between timed nonword reading and timed sight word reading differed by group; this seemed plausible given the variability in SWE raw scores across the control groups. I also explored if phonological deletion (i.e., zElision) slopes differed by group; this seemed less likely given the similarities in raw scores across the control groups. The second model building exploration allowed me to quantify the relationships between timed nonword reading and other literacy skills such as timed sight word reading and phonological deletion (i.e., Elision) while also asking if there were differences between groups on timed nonword reading, holding the other potentially important literacy skills constant. Finding a significant difference between the dyslexia and reading-matched group on timed nonword reading after holding other important variables constant would provide evidence that timed nonword reading could be used as a diagnostic sign of dyslexia. That is, timed nonword reading ability may develop uniquely in children with dyslexia compared to other children with novice literacy ability (i.e., younger reading-matched peers). The interactive regression models for the second model building phase were:

6. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(zSWE)_i + \beta_4(agematched * zSWE)_i + \beta_5(readingmatched * zSWE)_i + \beta_6(zElision)_i + \varepsilon_i$

7. $Y_i = \beta_0 + \beta_1(age\ matched)_i + \beta_2(reading\ matched)_i + \beta_3(zSWE)_i + \beta_4(agematched * zSWE)_i + \beta_5(readingmatched * zSWE)_i + \beta_6(zElision)_i + \beta_7(agematched * zElision)_i + \beta_7(readingmatched * zElision)_i + \varepsilon_i$

Table 13 shows model five from the first phase of model building and the two interactive models from the second phase of model building. Independent slopes tests suggested that the slopes for zSWE and zElision could vary by group, especially when comparing the age-matched and dyslexia groups. Incremental F-tests confirmed a significant difference in timed sight word reading slopes by group (i.e., zSWE), F (2, 81) = 9.08, $p < .001$. However, an incremental F-test for phonological deletion (i.e., zElision) questioned if there were differences in slopes by group, F (2, 81) = 2.81, $p < .07$. Therefore, the interaction terms for phonological deletion (i.e., zElision) were dropped. Dropping the elision interaction terms did not significantly change the measures of fit (e.g., adjusted R-squared or Root Mean Square Error) or the standard errors. To complete model selection, models five (no interaction terms) and six (interaction terms for zSWE) were compared; model six did not significantly inflate the standard errors, change the significance levels, or change the direction or magnitude of the coefficients. In fact, model six showed a slight improvement to adjusted r-squared, though this could be an artifact of adding two more predictor variables. Ultimately, model six was chosen as the final model.

Final model results. Seen in model six of Table 13, the model was a good fit for the data and accounted for 90% of the variance in timed nonword reading. In addition, the average size of a residual in this model was approximately one-third of a standard deviation for timed nonword reading. Results of model six were robust to alternative models, including a model that removed suspected outliers and a robust regression model

(see Table D4). The final model provided four results relevant to a discussion on timed nonsense word reading as a potential diagnostic sign of dyslexia – two related to comparisons and two related to non-causal relationships.

Timed nonword comparisons. First, the most important finding was that the dyslexia group had significant differences in timed nonword reading when compared to both age-matched and reading-matched peers, $p < 0.001$. As a reference, the model predicted that on average, dyslexics would score 0.58 (z-score) standard deviations below the sample mean on timed nonword reading when scoring exactly at the mean on timed sight word reading and phonological deletion. Put another way, the model predicted that the dyslexia group reads an average of 16.23 nonwords in 45 s when they also read 57.33 sight words in 45 s and score 23.66 (out of 34) on phonological deletion. In comparison, age-matched peers average 25.67 nonwords in 45 s under identical timed sight word and phonological deletion conditions. Reading-matched peers average 24.78 nonwords in 45 s under the same timed sight word and phonological deletion conditions. Taken together, the control groups were predicted to read significantly more nonwords in 45 s than the dyslexia group when performance on timed sight word reading and phonological deletion were average. In addition, the standardized beta coefficients indicated that the predictors Age-matched ($b^* = .30$) and Reading-matched ($b^* = .27$) had similar importance to the model, contributing to the high adjusted R-squared value for the model (adj-$R^2 = .90$). Overall, these findings suggested that the dyslexia group had a unique pattern of timed nonword scoring that was statistically different than children at similar ages and children with similar overall literacy maturity.

Table 13

Exploratory Analysis of Timed Nonword Reading (z PDE)

	Model 5 zSWE + zElision			Model 6 zSWE interaction terms			Model 7 All interaction terms		
Variable	*b*	*SE b*	*b**	*b*	*SE b*	*b**	*b*	*SE b*	*b**
Age-Matched	1.08***	0.11	.51	0.64***	0.14	.30	0.56***	0.14	.27
Reading-Matched	0.53***	0.10	.25	0.58***	0.10	.27	0.61***	0.10	.29
z SWE	0.51***	0.06	.51	0.33***	0.07	.33	0.36***	0.70	.36
zSWE*AgeMatch				0.70***	0.13	.37	0.58***	0.14	.30
zSWE*ReadingMatch				0.18	0.10	.10	0.19	0.12	.10
z Elision	0.16***	0.05	.16	0.16***	0.05	.16	0.11	0.07	.11
zElision*AgeMatch							0.30*	0.07	.14
zElision*ReadingMatch							0.01	0.10	.01
Dyslexia [Constant]	-0.05***	0.07		-0.58***	0.06		-1.22***	0.29	
adjusted R^2		.87			.90			.91	
RMSE		0.36			0.31			0.30	
F		154.09			141.12			111.16	
N		90			90			90	

Note. z PDE = Phonemic Decoding Efficiency z-score; RAN = Rapid Symbolic Naming; z LWI = Letter Word Identification z-score; z SWE = Sight Word Efficiency z-score. See Table D3 for models 1 – 4.
*$p < .05$. **$p < .01$ ***$p < .001$

Slope comparisons. Second, the model suggested that there was a significant interaction between group membership and timed sight word reading when predicting timed nonword reading. The timed sight word reading (i.e., zSWE) slope for the age-matched group ($b^* = 1.03$) was stronger than the slope for the reading-matched group ($b^* = 0.51$), indicating that a one-unit increase in timed sight word ability for the age-matched group would produce a two-fold increase in timed nonwords compared to the reading-matched group. This finding implies that the relationship between timed sight word and timed nonword reading in more skilled readers is different than unskilled readers. A close inspection of the added variable plots for each interaction term revealed that in the age-matched group, children with higher timed sight word scores were more likely to also have higher timed nonword scores. In the reading-matched group, children with higher timed sight word scores had a small lag in timed nonword scores, indicating that on average, younger children's timed sight word ability may develop slightly ahead of timed nonword ability but will likely even out over time.

When comparing the zSWE slopes of the two control groups to the dyslexia group slope, the model predicted that the difference between the slopes of the dyslexia and age-matched groups was significant at 0.70 standard deviations ($p < 0.001$), but the difference between the slopes of the dyslexia and reading-matched groups was nonsignificant at 0.18 standard deviations. Taken together, these findings suggest that the relative rate of growth between timed nonword ability and timed sight word ability for children with dyslexia does not rapidly improve over time like typically-developing children.

Non-causal relationships. Third, the results indicated a significant, yet non-causal relationship between timed sight word reading and timed nonword reading, accounting

for group membership and phonological deletion ability, $p < 0.001$. However, this relationship appeared to improve differently for older children compared to younger and dyslexic children, as previously discussed. Finally, the results supported a significant, yet non-causal relationship between timed nonword reading and phonological deletion ability ($p < 0.001$), accounting for group membership and timed sight word reading, and the relationship between improvements in phonological ability and timed nonword reading appeared consistent across groups.

Summary of Results

Overall, the results support the study design – (1) group performance was similar on visual and nonverbal ability; (2) the dyslexia and age-matched groups had similar ages yet different reading abilities; and (3) the dyslexia and reading-matched groups had similar z-scores for Literacy Maturity yet different ages. Children with dyslexia and age-matched peers (controlling for nonverbal ability) had a statistically significant difference in m-cell function, while children with dyslexia and reading-matched peers had comparable m-cell function (controlling for nonverbal ability). Typically-developing children appeared to have a rapid improvement in m-cell function during the early school-years (i.e., kindergarten to third grade), while children with dyslexia appeared to have inconsistent m-cell growth over the school-age years.

An exploratory analysis revealed that not every child with dyslexia had weakened m-cell function, and phonological ability appeared independent of dyslexic m-cell function. Similarly, children with lower literacy performance overall (i.e., dyslexic and reading-matched peers) had wide variability in m-cell function despite statistically similar group means. A cluster of “good” m-cell performers with lower literacy had similar

literacy and visual abilities as a cluster of "poor" m-cell performers with lower literacy, yet the groups had a statistically significant difference in m-cell function. None of the measures from this study could explain the variability in m-cell function among the "good" and "poor" m-cell performers at lower literacy levels. A final analysis explored timed nonword reading as a potential diagnostic sign of dyslexia because every member of the dyslexia group had difficulty with timed nonword reading. Interestingly, dyslexics' timed nonword reading was significantly different than both the age- and reading-matched groups, even when controlling for timed sight word reading by group and phonological deletion ability.

CHAPTER V

DISCUSSION

Dyslexia diagnosis suffers from low diagnostic power, which makes it difficult to identify those with dyslexia (i.e., diagnostic sensitivity) and rule out dyslexia in others (i.e., diagnostic specificity). Researchers disagree over the biological systems to investigate for a true diagnostic sign of dyslexia (i.e., a characteristic that points directly to the disorder). Many researchers argue for a phonological approach, citing brain imaging showing that language areas (e.g., Broca's area, Wernicke's area, the angular gyrus, the supramarginal gyrus) operate differently in children and adults with dyslexia compared to typically-developing peers (see Shaywitz, 2003). However, not all dyslexics demonstrate phonological difficulty (Frederickson & Frith, 1998; White et al., 2006), which weakens the phonological approach to diagnosis.

In contrast to the phonological approach to diagnosis, some researchers argue for a visual systems approach, citing imaging and structural evidence that weaknesses in magnocellular cells (i.e., m-cells) along the dorsal visual pathway (e.g., retina, thalamic LGN, area V5/MT of the occipital lobe) disrupt the efficiency needed for accurate and automatic literacy learning (see Stein, 2018a for a review). The majority of magnocellular theorists have focused on area V5/MT in the occipital lobe, but these designs suffer from a serious methodological flaw, which limits their conclusion that m-cells could serve as a deviant, or unique, diagnostic sign of dyslexia. Specifically, researchers ignore how

learning to read changes the occipital cortex (Fernandes et al., 2018; Huettig et al., 2018; Kaas & Lyon, 2007; Malik-Moraleda et al., 2018; Skeide et al., 2017), and how children with dyslexia may be unable to sharpen the occipital lobe in the same way as same-age peers due to blunted literacy acquisition (Olulade et al., 2013).

In this study, I measured m-cell function in children with dyslexia and included comparisons to age-matched and reading-matched peers to account for possible differences in occipital lobe refinery due to literacy acquisition. This study addressed the following research questions to determine if m-cells develop along a deviant or delayed path and to determine the usefulness of m-cells and the m-theory to dyslexia diagnosis.

1. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar ages, controlling for nonverbal ability?
2. To what extent does magnocellular function differ between school-age children with and without dyslexia at similar levels of literacy maturity (skilled automaticity with consolidated phoneme-grapheme units) controlling for nonverbal ability?

A multiple regression model that accounted for possible differences in nonverbal ability compared m-cell function among groups. Additional exploratory analyses examined m-cell variability within the dyslexia group, examined m-cell variability among children at lower levels of literacy maturity, and investigated timed nonword reading as a potential diagnostic sign of dyslexia. Similar to previous studies, m-cell function was measured with a coherent motion task that calculated a coherence threshold, or the percentage of moving dots needed among distractor dots to accurately identify coherence. In this study, 8- to 14-year old typically-developing children averaged 17.46% coherence, which aligns

with previous studies (Cornelissen et al., 1998b; Hadad et al., 2011; Joo, 2017; Raymond & Sorensen, 1998; Talcott et al., 2013).

The results support a difference in m-cell function between children with dyslexia and same-age peers; however, the difference becomes statistically insignificant when comparing the same children with dyslexia to younger peers at similar levels of literacy maturity. Though this outcome appears straightforward, additional exploratory analyses of the data suggest a complicated picture of m-cell function at lower literacy levels and questions both the m-theory and the argument against the m-theory. The following sections will discuss the deviance versus delay debate among m-theorists and their opponents, raise three questions that need to be answered in the m-cell debate, and offer possible explanations for each question given the results of this study and the extant literature, culminating with an integrated approach to dyslexia diagnosis to guide future research.

Magnocellular Theory of Dyslexia

The Magnocellular Theory of Dyslexia (m-theory) posits that children with dyslexia have deviant m-cells that cause literacy difficulty (Stein, 2019). In contrast, other researchers contend that children with dyslexia have delayed m-cells because of delayed literacy acquisition and that literacy growth will improve m-cell function (Olulade et al., 2013). The results of this study will be discussed within the deviance versus delay m-cell debate.

M-cell Deviance

The major evidence for m-cell deviance comes from replicated comparisons of children with dyslexia and same-age peers (see Benassi et al., 2010). M-theorists cite the

replication rate of this difference as support for a universal difficulty with m-cells in dyslexia. M-theorists also contend that the pervasive phonological problems present in most dyslexics can be attributed to a primary weakness in orthographic processing, which is caused by m-cell deviance (Stein, 2018a). The results of the m-cell comparison between dyslexics and age-matched peers in this study support a deviant view of m-cells within the m-theory – on average, children with dyslexia had significant weaknesses in V5/MT m-cell function compared to peers. However, the within-group m-cell variability for dyslexic children questions a universal deviance in dyslexic m-cells. Twelve out of 30 children with dyslexia scored the same as or better than the typically-developing m-cell average (17.46%), yet all 12 children had literacy difficulties that m-theorists argue originate with poor m-cell function. Other researchers have documented inconsistencies in the prevalence of m-cell function in dyslexic adults (Conlon et al, 2004; Hill & Raymond, 2002; Ramus et al., 2003; Tsermentseli et al., 2008) and in children with reading problems (Kassaliete et al., 2015; Wright & Conlon, 2009), though Kassaliete et al. did not sample diagnosed dyslexics.

In addition, the results of this study do not support the explanation that m-cell weaknesses cause secondary phonological difficulties (Stein, 2018a). In the current sample of children with dyslexia, phonological deletion (i.e., Elision) and m-cell function appeared to be independent. Dyslexic children with average or better m-cell function (i.e., greater than or equal to 17.46%) scored evenly across the phonological ability levels (e.g., Average, Below Average, Poor); similarly, dyslexic children with poor m-cell ability scored similarly across the phonological ability levels (see Table D4). The worst m-cell performers were not necessarily the worst phonological performers, and some of

the best m-cell performers still struggled with the phonological task. Cornelissen et al. (1998b) suggested a hypothetical independence in m-cell function and phonological ability in their study of typically-developing children, but the authors noted that in their sample, the worst m-cell performers were also the worst phonological performers. It appears that in children with dyslexia, m-cell function and phonological ability may be more independent than in typically-developing children.

In sum, the results of the initial regression comparison of dyslexics and same-age peers support the m-theory argument, but the wide m-cell variability within the dyslexia group and the independence of phonological ability and m-cell function question the m-theorists' argument that all children with dyslexia have deviant m-cells that cause literacy difficulty.

M-Theory Opposition

In contrast to possible deviance in m-cell function, other researchers have suggested a delayed m-cell trajectory in children with dyslexia (Olulade et al., 2013; Taylor et al., 2018). In the opposing view to the m-theory, children with dyslexia show m-cell weakness because of delayed literacy acquisition, meaning that as dyslexics learn to read like same-age peers, they will train their m-cells to keep up with the visual demands of text reading. In this study, two results support a delayed view of m-cell function in dyslexia.

First, the regression results for the dyslexia and reading-matched comparison suggest that on average, children with dyslexia have similar m-cell function as younger, typically-developing peers matched on literacy maturity. That is, the unique difference in dyslexic m-cell function when compared to same-age peers is no longer significant

when accounting for literacy maturity. Second, the developmental trajectory of m-cells documented in this study supports a delayed view of m-cell function. Though this study was not longitudinal, a cross-sectional view of m-cell development over the school-age years (see Figure 10) suggests that, on average, typically-developing children improve m-cell function over time. It can be assumed that barring any diagnoses, the younger, typically-developing children in this study will, on average, go on to have average m-cell ability. Age cannot be the determining factor in m-cell development, though, because children with dyslexia performed worse than same-age counterparts. Whereas typically-developing children appear to improve over time, children with dyslexia continue to have poor m-cell ability and poor literacy ability into the older school years, supporting the view that literacy acquisition and m-cell function may develop in tandem.

Further supporting a delayed view of m-cell development, m-cell improvement for typically-developing children does not appear consistent over time, with a rapid improvement in m-cell function from kindergarten to third grade (see Figure 10). Though children go through many physical and emotional changes from kindergarten to third grade, one of the most prominent changes is learning to read. Taylor et al. (2018) documented negligible m-cell improvements in readers from second to third grade and took this finding to possibly contradict the delayed m-cell view that literacy bootstraps m-cells to develop. However, Taylor et al. measured m-cell improvements across very advanced second-grade readers in a one-year period; it is possible that these children had already bootstrapped m-cells to develop before second grade because of their early advancements in literacy acquisition. Measurements of one-year increases in m-cell function as they relate to literacy acquisition may be more meaningful during the

kindergarten to second-grade years compared to the second to fifth-grade years, especially in more advanced readers.

Despite the logic in the argument that literacy bootstraps m-cells to develop, a closer inspection of m-cell function within the dyslexia and reading-matched groups raises questions about Olulade et al.'s (2013) theory of literacy-driven m-cell development. The association between literacy maturity and m-cell function in this study ($r = -.31$, $p < .01$) replicates previous literature (Gibson et al., 2006; Joo et al., 2017; Pammer & Wheatly, 2001; Olulade et al., 2013; Wilmer et al., 2004), but m-cell function and literacy ability did not appear to develop the same way for all children as suggested by Olulade and colleagues. In this study, lower levels of literacy maturity were characterized by more variability than higher levels of literacy maturity (see Figure 13). Among children with lower literacy maturity (i.e., children from the dyslexic and reading-matched groups), some children had significantly better m-cell function than others. Other literacy and visual abilities could not account for the significant difference in m-cell function between "good" and "poor" m-cell performers with low literacy. If gains in literacy drive m-cell function as Olulade and colleagues suggested, children at similar levels of literacy should have comparable m-cell function. At the group level, the regression comparison supported the claim that children at similar levels of literacy have comparable m-cell function. However, this was not the case when children with lower literacy were grouped together (i.e., removing the dyslexic or reading-matched label) and the "good" m-cell performers were compared to the "poor" m-cell performers. At least one additional researcher has questioned Olulade et al.'s theory that literacy ability and

m-cells develop together; Joo et al. (2017) demonstrated that an eight-week reading intervention improved literacy ability but did not improve to m-cell function in dyslexics.

In sum, the comparison of m-cell function between dyslexics and reading-matched peers and the trajectory of m-cell development over the school-age years support a delayed view of m-cell development in dyslexia. However, a closer analysis of m-cell function within the dyslexia and reading-matched groups question if m-cell function and literacy maturity develop in tandem in all children.

Summary

I predicted the following results would point to delayed m-cell function in dyslexia: (1) a significant difference between dyslexics and same-age peers; (2) a nonsignificant difference between dyslexics and reading-matched peers. That is, children with dyslexia could not have deviant m-cell function if younger, typically-developing children with comparable literacy maturity had similar m-cell function. However, the results do not support a delayed or deviant understanding of m-cell function in dyslexia at this time because of the m-cell variability within the dyslexia group and the m-cell variability among lower literacy performers.

M-cells appear to operate differently in some but not all dyslexics, but this finding does not support the larger m-theory because m-theorists claim that m-cells are responsible for literacy difficulties in all dyslexics. Opponents to the m-theory argue that literacy ability and m-cell function mature together, with literacy ability driving m-cells to develop (Olulade et al., 2013). But in this study, within the lower literacy performers (i.e., the combined dyslexia and reading-matched group), m-cell function and literacy

ability did not develop similarly. Therefore, neither the m-theory nor its opposing theory of literacy driven m-cell change can be fully supported with the current data.

Important M-Cell Questions and Hypotheses

Given the inconclusive m-cell results of this study, the following three questions remain unanswered in the m-cell and m-theory debate. The importance of answering each question will be argued, followed by possible hypotheses to provide future direction and thought.

How Can Some Children with Dyslexia Have no M-cell Weakness?

This question is important to address because the m-theory assumes that all children with dyslexia have m-cell weaknesses; however, in this study, 12 out of 30 children did not have m-cell weakness. Four scenarios could explain the finding that 12 children with dyslexia exhibited no m-cell disturbance, though the likelihood of each varies.

First, the m-cell location could be explanatory. The task in this study measured m-cell activity in area V5/MT, so it is possible that individuals with dyslexia have deviant m-cells at a different point along the visual pathway from the retina to area V5/MT in the occipital lobe. A very small number of researchers have investigated m-cell function in the retina (Pammer & Wheatley, 2001), thalamic LGN (Livingstone et al., 1991; Giraldo-Chica et al., 2015), and area V1 (Talcott et al., 1998), but their findings point to differences when compared to same-aged peers, so it is unknown if those differences remain significant when compared to reading level-matched peers. The 12 individuals with dyslexia who did not have difficulty with the coherence task in this study could have m-cell disturbances in the retina, thalamic LGN, or visual area 1 (i.e., V1) in the occipital

lobe not captured by the coherence task in this study. It is unlikely that lower levels of analysis (e.g., retina, LGN, V1) with disturbances great enough to interact with literacy ability would not also impact projections to area V5/MT, but a disassociation between higher and lower areas of the dorsal pathway has been found in one study of English-speaking dyslexics when compared to same-age peers (Pellicano & Gibson, 2008).

Another explanation for the high number of dyslexic children with average m-cell ability could be the sampling technique in this study. About 90% of the cells in area V5/MT are m-cells, but the other 10% are smaller parvocellular cells (p-cells; Kolb & Wishaw, 2015). The inclusion criteria for this study required average or better p-cell function (measured with an object discrimination task), which has not been used in previous studies for inclusion. It was chosen for this study to rule out any interaction between the magnocellular pathway and parvocellular pathway that could cause visual difficulty. Therefore, it is possible that previous findings where almost all children with dyslexia had poor m-cell function could be capturing a joint disturbance in parvocellular and magnocellular cells, leading to an assumption that almost all children with dyslexia have poor m-cell function in area V5/MT. One group of researchers have reported joint m-cell and p-cell disturbances in Iranian children with dyslexia compared to same-age peers (Ahmadi, Pouretemad, Esfandiari, Yoonessi, & Yoonessi, 2015), but these results have not been reported in English-speaking children to date.

A third scenario to explain average m-cell function in dyslexia could be temporal cell disturbance in another system, namely the auditory system. Large cells in the primary and secondary auditory cortex and the inferior frontal gyrus work similarly to visual m-cells; these auditory cells respond to rapid temporal elements of sound, both speech and

non-speech sounds (Lorusso et al., 2014; Eagleman & Downar, 2016; Gaab et al., 2007; Tallal, 1980; 2004). Researchers have documented disturbances in rapid auditory processing in children with dyslexia in behavioral studies (Lorusso et al., 2014; Tallal, 1980; 2004) and brain imaging studies (Gaab et al., 2007). This difficulty has been observed in children as young as infants who have a familial risk for dyslexia (Beasich & Tallal, 2002), suggesting that rapid auditory processing deficits may be present before literacy instruction begins. Similar to the m-theory, not all children with dyslexia have performed poorly on measures of rapid auditory processing. Collectively, it appears that auditory temporal cells may be present in some dyslexics, offering a possible explanation for the dyslexics without m-cell difficulty who have low literacy ability.

A final scenario could be that almost half of the greater population of dyslexic children have no difficulty with m-cell function. This scenario would question the use of m-cells as a single diagnostic sign of dyslexia.

How Can Children with Lower Literacy Maturity Have Such M-cell Variability if Literacy and M-cells Develop Together?

Though similar to the question about variability within the dyslexia group, this question looks at variability within lower literacy performers. This question is important to the larger debate over m-cell function in children because it challenges a crucial argument against the m-theory. Opponents to the m-theory contend that children at similar levels of literacy ability have similar m-cell function, arguing that literacy ability drives m-cell development (Olulade et al., 2013). In this study, children at similar literacy levels had similar m-cell function at the group-level (i.e., dyslexia group compared to reading-matched group). However, when group assignment was not considered, children

at lower levels of literacy formed distinct clusters of "good" and "poor" m-cell performers who had statistical differences in m-cell function. This finding implies that literacy maturity may not explain m-cell function in all children at lower literacy levels. Asking this question challenges the predominate argument against the m-theory and leaves room for other explanations outside of literacy acquisition for poor m-cell function among lower literacy achievers.

First, and most importantly, m-cell task differences likely explain why children with lower literacy levels had statistical differences in m-cell function in this study but not the study by Olulade et al. (2013). The opposing m-theory argument that literacy drives m-cell development came from an fMRI study by Olulade and colleagues. In the study, m-cell function was measured as accuracy at 40% coherence. Most m-cell studies (including this one) measured an m-cell coherence threshold, or the lowest percentage of moving dots needed to detect coherence among any of the dots. The task used by Olulade et al. was necessary to find differences in cortical activation – participants needed to perform a repetitive task to firmly establish a pattern of activation in area V5/MT and subtract the "noise" (e.g., head movement, wandering thoughts, etc.). But a study of m-cell accuracy at a very high level of coherence (i.e., 40%) may not capture the variability seen among lower literacy performers in this study. Using Figure 13, a task such as Olulade et al.'s would likely fail to capture the entire "good" m-cell cluster among lower literacy performers. Given that Olulade et al.'s results formed the major opposition to the m-theory, future researchers may consider retesting the theory that literacy drives m-cell development by using m-cell tasks that allow for variability.

As previously discussed, another possible explanation for the variability in m-cell function at lower literacy levels could be m-cell weakness in another location outside of area V5/MT. Possibly the "good" m-cell cluster with lower literacy has difficulty with m-cells in the retina, thalamic LGN, or V1 not captured by the task in this study.

It is tempting to suggest that attentional systems (e.g., posterior parietal or prefrontal) could explain differences in m-cell ability among low literacy children – possibly the cluster of "poor" m-cell performers with lower literacy had trouble paying attention to the task, explaining their low m-cell scores. After all, m-cells have been shown as a basic pre-requisite to visual attention (Vidyasagar, 2019; Vidyasagar & Pammer, 2009), so the likelihood of the "poor" m-cell cluster having visual attention problems may be greater than the "good" m-cell cluster. However, none of the 24 children in the "poor" m-cell cluster had reported attentional difficulties, and it is statistically unlikely that all 24 children in the "poor" m-cell cluster had undiagnosed attentional system deficits.

If visual projections to area V5/MT (e.g., retina, thalamic LGN, V1) and visual projections from area V5/MT (e.g., posterior parietal attentional system) may not explain why children at similar levels of literacy maturity have differing m-cell function, possibly a projection area outside of the visual system may explain why children at lower levels of literacy maturity have discrepant m-cell function. The most obvious system to blame given the ties to reading would be the language systems, specifically the phonological part of the language system that has historically been implicated in dyslexia (Shaywitz, 2003). However, the clusters of "good" and "poor" m-cell performers at lower literacy levels had nonsignificant differences when comparing z-scores for Elision and Rapid

Naming, both phonological processing tasks. In other words, children in the "good" m-cell cluster had comparable phonological ability to children in the "poor" m-cell cluster, suggesting that phonological prowess cannot explain the differences observed in m-cell function among lower literacy performers.

It appears that the task in the Olulade et al. (2013) study, which formed the opposing m-theory argument that literacy drives m-cell development, may be the most likely explanation for the variability within lower literacy performers. However, a critical assumption in explaining the variability among lower literacy performers is that lower literacy performers are supposed to have similar m-cell function. That is, this discussion centers around ways to explain the variability under a delayed view of m-cell function. If m-cells are truly deviant in dyslexics, it may be that the variability among lower literacy performers is not caused by the same mechanism. Put another way, it is possible that whatever factor determines which younger, typically-developing children have "good" versus "poor" m-cell function may not be what determines which children with dyslexia have "good" versus "poor" m-cell function. Removing the labels "dyslexic" and "typically-developing" to analyze all lower literacy performers illuminates potential problems in the way literacy maturity and m-cell function have been previously discussed. But grouping them together and investigating factors for their varied m-cell performance assumes that the unknown factor operates similarly across both groups. Grouping dyslexics and typically-developing peers together also assumes that their m-cells have the same potential and prognosis for growth, which may not be true.

Is There Another Way to Approach Dyslexia Diagnosis and Research?

The preceding discussions have illuminated potential problems with the m-theory and the opposing m-theory argument. I have previously argued that the m-theory suffers from a serious methodological flaw by neglecting to consider how learning to read impacts the brain. I have also now suggested that the opposing m-theory argument (i.e., literacy drives m-cells) suffers from a task-design limitation by neglecting to allow for variability among lower literacy performers. Each scenario significantly limits our ability to make conclusions about m-cells in dyslexia. Though finding conclusive answers about m-cells in dyslexia is important, we are no closer to identifying a diagnostic sign.

The major contenders for a potential diagnostic sign of dyslexia are difficulties with phonological aspects of language (Ramus et al., 2003) and visual m-cells (Stein, 2019), though rapid auditory cells (Gaab et al., 2007) have a growing number of supporters. Each contender's argument is limited by cases of dyslexics who seem to contradict the idea of a universal difficulty in either the phonological, visual, or auditory system (see Elliott & Grigorenko, 2014, for a review). In this study, not every diagnosed dyslexic had phonological difficulty; visual difficulty was also not ubiquitous.

However, every child with dyslexia had difficulty with timed nonword reading, and the majority had extreme difficulty. This outcome adds to previous findings of nonword reading and/or timed nonword reading difficulty for dyslexics compared to same-age peers (Gallagher, Laxon, Armstrong, & Frith, 1996; Hatcher, Snowling, & Griffiths; Norton et al., 2014; Witton et al., 1998) and compared to reading-matched peers (van Ijzendoorn & Bus, 1994). However, the use of timed nonword reading as a diagnostic sign has received little traction because researchers have used timed nonword

reading results to support a phonological-only view of dyslexia (see van Ijzendoorn & Bus, 1994). Consequently, advocates of other approaches to dyslexia's origin (e.g., visual or auditory advocates) may not accept a universal difficulty in timed nonword reading because they have demonstrated that not every dyslexic has phonological difficulties. However, timed nonword reading requires more than phonology. In reality, timed nonword reading requires the orthographic processor, the phonological processor, the auditory system, and the visual system, including contributions from m-cells. Moving forward, it may be beneficial to reconceptualize how the field of dyslexia diagnosis has historically categorized timed nonword reading given its potential as a diagnostic sign of dyslexia.

Timed nonword reading. The following sections will briefly describe timed nonword reading and present timed nonword reading as an alternative approach to dyslexia diagnosis given the inconsistencies with the m-cell literature and the boom in biological imaging studies since timed nonword reading was labeled a phonological-dominant skill. I will also offer a cohesive way forward for dyslexia diagnosis research within the framework of timed nonword reading.

***What is timed nonword reading*?** On a practical level, timed nonword reading in this study required children to accurately read as many nonwords as possible in 45 s (e.g., *zarp, bemhun*). Within the larger understanding of literacy abilities, timed nonword reading requires the automatic consolidation of phoneme-grapheme units. Literacy ability matures after smaller units of varying size that correspond to regularly occurring patterns in the reader's orthography (e.g., *-ain* in English) grow to whole units (i.e., *rain*) automatically available during text reading (Adams, 2013; Ehri, 2017; Perfetti, 1992;

Steacy et al., 2019). Even after whole units become automatic, skilled readers retain smaller units to automatically apply when faced with novel words (e.g., *legerdemain*; Ehri, 2017). Timed sight word reading measures the integrity and efficiency of whole-word units. Timed nonword reading measures the integrity and efficiency of smaller units. Whereas whole-word units benefit from the reinforcement of meaning (Adams, 1990), smaller units necessary for nonword reading require reinforcement from the statistical and analogical properties of an orthography (Steacy et al., 2017; Steacy, Compton et al., 2019). For example, the sound /e/ in *-ain* becomes stable and reinforced because of the statistical occurrence of *-ain* in English, allowing *-ain* to be analogically applied to novel words (Steacy et al., 2017; Steacy, Compton, et al., 2019).

Therefore, timed nonword reading relies on mastery of an orthography for statistical and analogical application as well as mastery of phonology to map phonemes onto graphemes. In the Reading Systems model (Adams, 1990, 2013), timed nonword reading relies on the orthographic and phonological processors. Previous researchers have discussed timed nonword reading as a measure of phonological awareness or phonological decoding (Torgesen et al., 2012; van Ijzendoorn & Bus, 1994; Wagner & Torgesen, 1987; Witton et al., 1998), but that term may not capture the true nature of a timed nonword task. In reality, timed nonword reading relies on the automatic ability to map sounds onto printed letters and combine the sounds in a way that is sensitive to the letter combinations of the specific orthography. Therefore, timed nonword reading may be better seen as an equally phonological and orthographic task that relies on auditory and visual input.

Timed nonword reading as a diagnostic sign. In this study, an exploratory regression analysis for timed nonword reading documented significant differences between dyslexics and typically-developing peers of the same age and reading level. The final model explained 90% of the variance in timed nonword reading, accounting for group status (e.g., age-matched, dyslexic, reading-matched), differences in timed sight word ability by group, and differences in phonological deletion ability. Specifically, the model predicted that on measures of timed nonword reading, children with dyslexia would score approximately 0.58 *SD* below reading-matched peers and 0.64 *SD* below age-matched peers, even if all children (e.g., dyslexics, age-matched, and reading-matched) had identical timed sight word ability and identical phonological deletion ability. This finding aligns with van Ijzendoorn and Bus's (1994) meta-analysis that the difference in nonword reading between dyslexics and typically-developing peers was approximately half of a standard deviation, though the inclusion of a timed version in the current study may account for additional explained magnitude. Conceptually, a difference in timed nonword reading compared to age- and reading-matched peers implies that children with dyslexia have unique differences in the integrity and/or efficiency of smaller phoneme-grapheme units compared to typically-developing children at any age. Future studies may include an additional measure of untimed nonword reading to parse out the influence of integrity versus efficiency.

Critically, the regression results also imply that phonological deletion difficulty may not be a unique difficulty in dyslexia. Previous researchers have argued for a phonological approach to dyslexia diagnosis (Ramus et al., 2003; Vellutino et al., 2004). However, the results of this study suggest that a phonological-only approach to dyslexia

diagnosis may not be as sensitive or specific as an approach that centers around timed nonword reading ability. Other researchers have used dyslexic differences in timed nonword reading as support for a universal phonological deviance in dyslexia (see van Ijzendoorn & Bus, 1994). However, the results of this study suggest that differences in timed nonword reading between dyslexics and typically-developing peers cannot be explained by phonological differences, positioning timed nonword reading as more than phonological and more ubiquitous than phonological ability in dyslexics.

Timed nonword reading appears an even more likely diagnostic sign when inspecting its trajectory across the school-age years. A cross-sectional trajectory of timed nonword reading by group suggests a deviant course of development for children with dyslexia compared to typically-developing peers – the gap between typical and dyslexic does not appear narrow over time (see Figure 15). This potentially deviant trajectory for timed nonword reading can be compared to the trajectory for timed sight word reading (see Figure 16). In contrast to timed nonword reading, the trajectory for timed sight word reading suggests a delayed path of development – the gap between dyslexic and typical children appears to be closing over time.

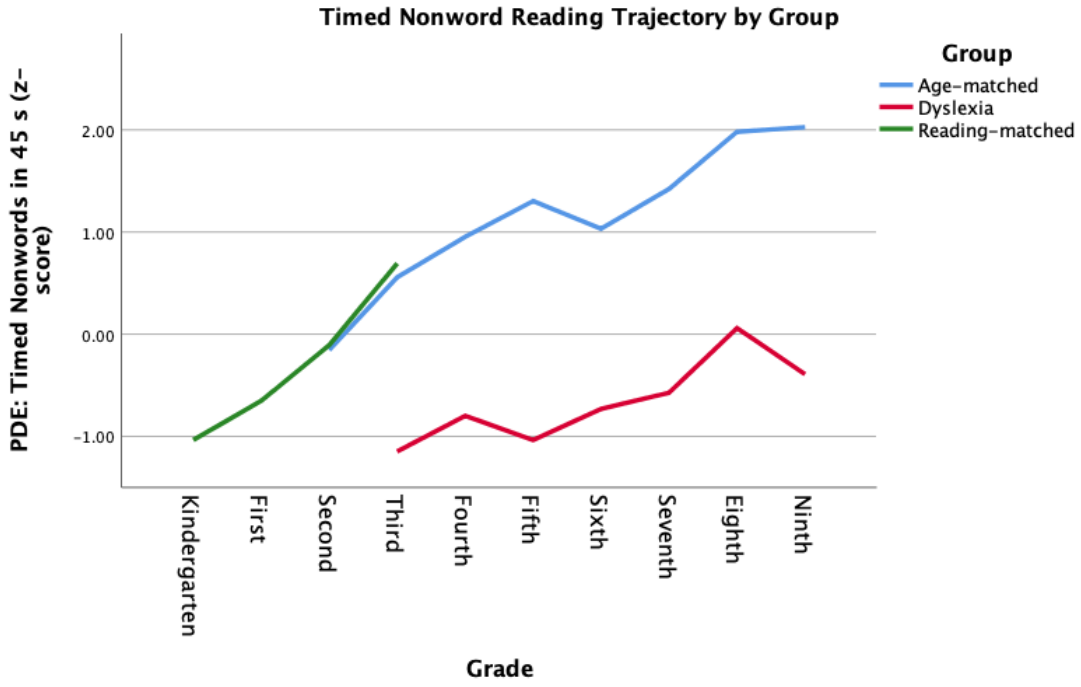

Figure 15. Timed Nonword Word Reading Trajectory by Group (N = 90)

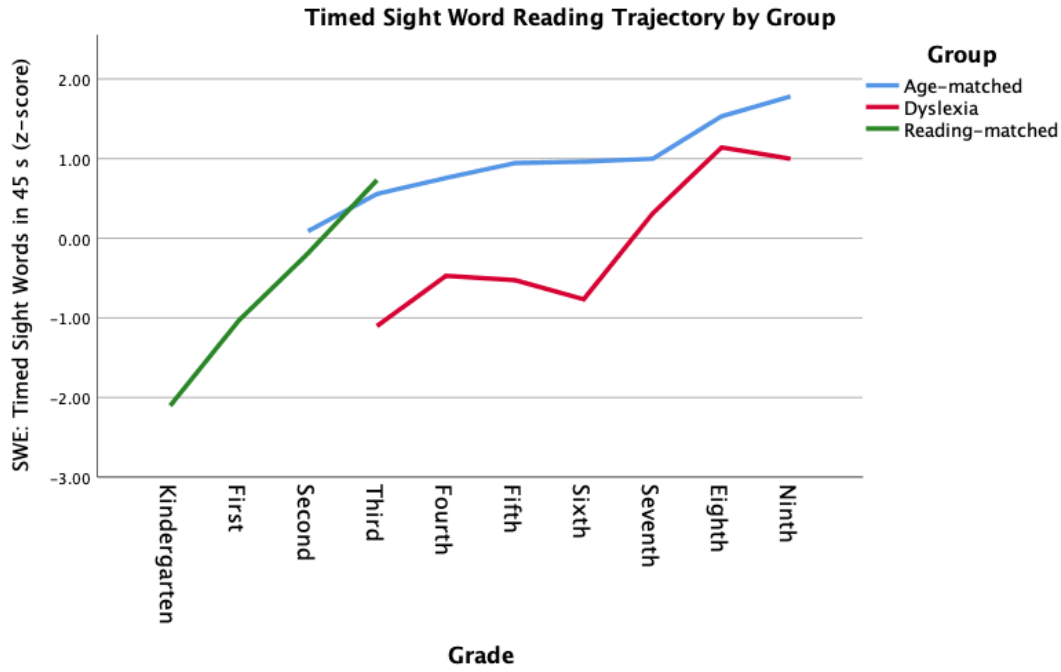

Figure 16. Timed Sight Word Reading Trajectory by Group (N = 90)

Taken together, the trajectories suggest that children with dyslexia may increase their bank of whole-word units over time without improving their bank of smaller units at the same rate; it could also be that they have a bank of smaller units but are unable to apply them under timing pressures when the meaning processor cannot assist the phonological and orthographic processors (Adams, 1990) during nonword reading tasks. The trajectories are supported by the differences in timed nonword reading slopes among the groups in the regression model. The model predicted that over time, typically-developing children improve timed sight word ability and timed nonword ability at comparable rates, whereas dyslexic children improve timed sight word ability without similar improvements in timed nonword ability. The trajectories for other skills measured in this study can be referenced in Appendix E.

In sum, timed nonword reading may provide a measure of unique dyslexic difficulty during dyslexia diagnosis. Though younger peers also demonstrate lower timed nonword reading scores, it is likely that inexperience drives this difficulty because typically-developing children improve over time. In contrast, children with dyslexia continue to struggle with timed nonword reading despite targeted instruction and intervention; every child with dyslexia in this study had one-to-one or small group intervention for at least six months prior to the assessment. The prevalence of nonword deviance in dyslexia has historically been interpreted as support for a phonological view of dyslexia; however, phonological ability cannot account for the differences in timed nonword reading between children with dyslexia and typically-developing peers, implying that timed nonword reading requires more than the phonological aspects of language.

Biological basis of timed nonword reading. Researchers have shown that nonwords recruit more than phonological areas of the brain, but the imaging evidence came after nonword reading became synonymous with a phonological view of dyslexia diagnosis. In reality, timed nonword reading requires the synthesis of visual, auditory, language, and motor networks. Nonwords are analyzed in the left inferior frontal gyrus and left parieto-temporal junction (e.g., angular gyrus, supramarginal gyrus), not primarily in the VWFA where real words can be processed (see Figure 17; Herbster, Mintun, Nebes, & Becker, 1999; Mattheis, Levinson, & Graves, 2018; Rissman, Eliassen, & Blumstein, 2003). Whether nonwords are read silently or orally, the auditory cortex must send sound information to the inferior frontal gyrus and the angular gyrus for further phonological analysis (Gaab et al., 2007). Similarly, the visual cortex must send visual information to the angular gyrus, and there is evidence that the visual cortex is still involved during oral-decision nonword tasks (i.e., nonword repetition) in readers who have learned phoneme-grapheme combinations (Rissman et al., 2003). The finding that the visual cortex may still activate during oral-decision nonword tasks strengthens the theory that the phonological and orthographic processors are never turned off during tasks that require either letters or letter sounds (Adams, 1990, 2013), implying that once phonemes are wedded to graphemes, the brain activates information for both simultaneously.

The left superior temporal gyrus, including Wernicke's area, also remains active during nonword reading to assist with verbal short-term memory (Perrachione et al., 2016). The temporal nature of timed nonword reading requires efficiency in the right cerebellar lobule, important for receiving and integrating sensory input for motor

planning (Norton et al., 2014; Saygin et al., 2013). Taken together, timed nonword reading appears to rely on left tempo-parietal regions, the left superior temporal gyrus, and the inferior frontal gyrus, with a small yet important contribution from the right cerebellar lobule.

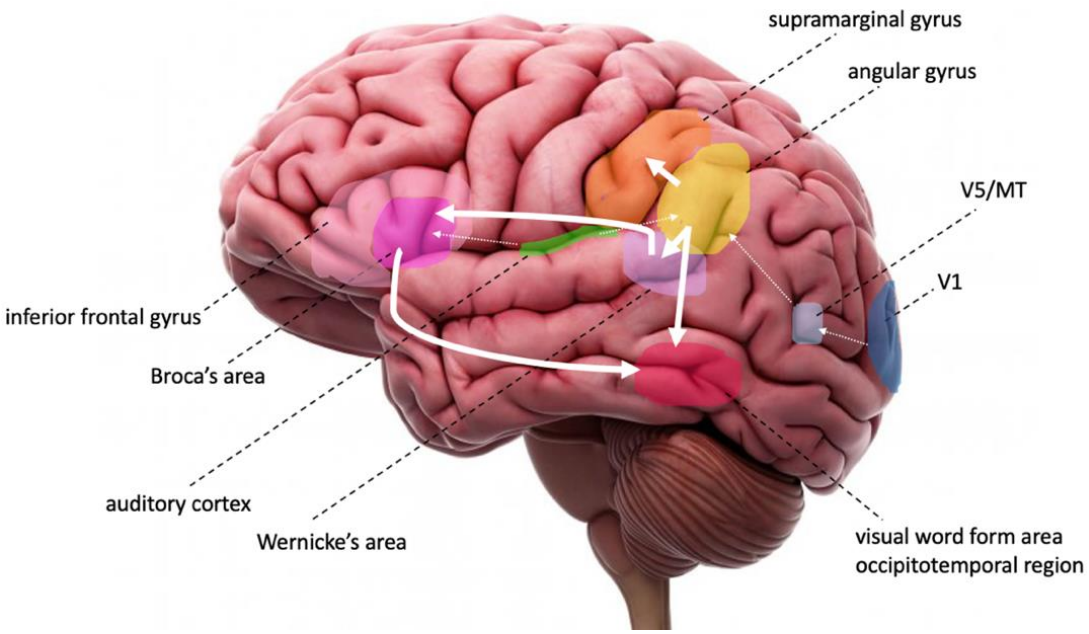

Figure 17. Model of Reading Networks

Dyslexic children show general reductions in gray matter volume (Linkersdorfer et al., 2012; Richlan et al., 2013) and white matter tracts in the left tempo-parietal region (Deutsch et al., 2005), including the angular gyrus, supramarginal gyrus, and Wernicke's area (see Figure 17), all regions necessary for nonword reading analysis. Importantly, decreased gray matter volume and decreased white matter tracts in areas necessary for nonword reading have been found in a comparison of dyslexic children to younger reading-matched peers (Hoeft et al., 2007), providing evidence of deviant development. Less gray matter means fewer nerve cells in the cortical area responsible for receiving visual and sound signals, integrating and analyzing the signals, and providing a hub to project the signals for further analysis (Horwitz et al., 1998; Kolb & Wishaw, 2015). Less

white matter means a reduced ability for communication in the area (Deutsch et al., 2005; Kolb & Wishaw, 2015). Researchers have also found that dyslexics have decreased activation in the right cerebellar lobule (Nicolson & Fawcett, 2006; Norton et al., 2014; Saygin et al., 2013), though recent findings suggest that not every child exhibits cerebellar deficits (Ashburn, Flowers, Napoliello, & Eden, 2020).

Timed nonword reading as cohesive. The brain basis for timed nonword reading includes the major contenders in the dyslexia diagnosis debate: (1) timed nonword reading requires areas necessary for phonological processing, including the inferior frontal gyrus, angular gyrus, and Wernicke's area; (2) timed nonword reading requires that m-cells in V5/MT send letter sequence and position information to the angular gyrus; (3) timed nonword reading requires that rapid auditory cells send sound sequence and position information to the inferior frontal gyrus and angular gyrus. Given that visual, auditory, and phonological difficulties are observed in many dyslexics, yet none can account for all, timed nonword reading may be a cohesive solution. Whereas phonological tasks require primarily auditory and phonological input, timed nonword reading requires auditory, phonological, and visual input, possibly accounting for all of the proposed difficulties in dyslexia. Further, timed nonword reading requires heavy orthographic processing, which may be the missing link in previous attempts to identify a diagnostic sign that captures all dyslexics. That is, previous measures of m-cell function, rapid auditory processing, and phonological processing may not tax the orthographic processor enough to separate dyslexics from other readers.

During timed nonword reading, the orthographic processor is essential because it mitigates the quantity problem in the visual cortex when trying to process letter sequence

and position (Adams, 2013). That is, the brain uses the orthographic processor as a higher-order analysis "center" to filter the amount of visual information coming into the system during reading attempts. Without knowing how the orthography of a written language system works (i.e., the likely and unlikely letter combinations), the orthographic processor does not know what to pay attention to beyond single letters and therefore cannot filter for efficiency. It may be that deviant m-cells prevent the orthographic processor from working efficiently. In fact, researchers have shown that V5/MT function predicts orthographic knowledge (Demb et al., 1998; Witton et al., 1998). Or, it may be that the orthographic processor receives quality information but cannot act on it because orthographic processing is outside of the innate brain network and requires a code-specific system, something dyslexics struggle to master (Shaywitz, 2003). Either way, the role of m-cells in dyslexia cannot be ruled out at this time.

Though the orthographic processor and visual cortex are essential to timed nonword reading, the phonological processor and auditory cortex are essential as well. In contrast to the orthographic processor, the phonological processor is innately wired for processing the individual formants (i.e., "sound shapes") received by the auditory cortex. However, it takes instruction to bring this process to a level of explicit awareness where readers can act on the sound information. That is, instruction in how to separate the sounds of words can allow a reader to manipulate the formants (that we perceive as phonemes). If the auditory cortex cannot accurately perceive changes in sound frequencies (e.g., problems with rapid auditory processing), the phonological processor cannot make sense of the formants, especially during a task like timed nonword reading

that requires the phonological processor to make sense of formants without the influence of meaning or context.

In dyslexia, it may be that the auditory cortex sends incorrect signals; or, it may be that the phonological processor receives quality information but struggles to blend or manipulate formants. Even if the signals from the visual and auditory cortices reach the orthographic and phonological processors without disturbances, and each processor is able to analyze the information accurately and efficiently, the brain still has to integrate orthographic and phonological information to construct automatic and consolidated phoneme-grapheme units used in nonword reading. Therefore, it appears that many things could go wrong and cause dyslexia, but the potential causes appear to be captured by timed nonword reading.

Moving forward with timed nonword reading. Using timed nonword reading as a diagnostic sign may improve previous efforts to identify a biological cause for two reasons. First, using timed nonword reading as a diagnostic sign may improve prevalence rates and the homogeneity of the dyslexic population. Second, the possible underlying causes of dyslexia could be systematically tested according to the requirements of timed nonword reading. The current approach is to test underlying causes across heterogenous samples of dyslexics (i.e., not all have similar difficulties) without a systematic approach to move from lower to higher levels of analysis (or vice-versa). Using timed nonword reading as an anchor measure to begin testing the possible underlying causes may provide structure and consistency for future researchers.

In reality, it is likely that the exact pattern of disruption varies among individuals because of treatment and aptitude interactions. As a result, researchers may consider

exploring the usefulness of timed nonword reading plus one or more related measures, such as m-cell function, rapid auditory processing, or rapid symbolic naming, to capture all children diagnosed with dyslexia across the school-age years. Finding a combination of related measures that serve as diagnostic signs could provide earlier risk-identification and intervention as m-cell function, rapid auditory processing, and rapid symbolic naming can be measured in pre-reading children.

Moving forward, it may also be beneficial for the collective dyslexia community to establish a diagnostic sign of dyslexia prior to establishing the biological cause of dyslexia. Historically, the sign has also been described as the cause. For example, the m-theory describes m-cells as a unique characteristic or sign of dyslexia that also causes dyslexia (Stein, 2019). Similarly, the Phonological Deficit Hypothesis describes phonological processing as a unique characteristic or sign of dyslexia, and the NICHD definition of dyslexia states that reading problems are caused by the phonological component of language (Lyon et al., p. 2). In each case, the sign and cause are the same. However, this approach has resulted in many disagreements and contradictory cases.

To move ahead collectively rather than competitively, it may be beneficial to identify a universal diagnostic sign that incorporates the competing theories to provide homogeneity in the target sample before searching deeper for an underlying cause. Timed nonword reading appears a likely candidate if reconceptualized with its biological basis beyond phonology. Timed nonword reading may be better thought of as an orthographic-phonological skill that requires basic visual and auditory input. Thresholds or cut-offs for acceptable timed nonword reading scores in dyslexia would need to be established, but

using a consistent anchor measure (i.e., timed nonword reading) could improve diagnosis from its current state.

Unique Contribution

In this study, I provided the first comparison of m-cell function in children with dyslexia to age-matched and reading-matched peers that uses nearly identical m-cell task specifications as previous studies (Cornilessen et al., 1998b; Talcott et al., 2013). In addition, I controlled for parvocellular pathway contributions to m-cell variability, which has not been previously reported. The matching procedures also provide a nuanced yet critical improvement to previous designs. I matched children with dyslexia to younger peers on literacy maturity, which I defined as automatic consolidation of phoneme-grapheme units and measured with a timed sight word task and timed nonword reading task. Using this raw score composite allowed me to capture reading experience (i.e., timed sight word reading) and facility with smaller orthographic units without the influence of meaning (i.e., timed nonword reading). Previous researchers have used grade-equivalent sight word reading scores or age-equivalent sight word scores, which may not capture the difficulty faced by all children with dyslexia.

Further, I presented timed nonword reading as a potential diagnostic sign of dyslexia and reconceptualized timed nonword reading as more than phonological. In doing so, I hoped to bring cohesion to the larger debate on dyslexia's biological origin as the extant literature typically argues for *either* the phonological, auditory, or visual system and assigns timed nonword reading to the phonological view. I reasoned that using timed nonword reading as a foundation for future research may provide a path for

more consistent dyslexia diagnosis, which could improve research to determine the precise contribution from the competing systems in dyslexia's origin debate.

Limitations

A number of factors limit the results of this study, including limitations with recruitment, the measures, the observational design, and the orthographic focus on English. Several issues with recruitment limit this study's findings. First, participants volunteered to participate, which could represent a select subgroup of children and not a random selection from among the broader population of children ages 6 to 14. Second, participants with dyslexia did not have any co-morbidities, such as Attention Deficit Hyperactivity Disorder, Math Disorder, or Autism. Therefore, the findings from this study should be applied with caution to children with co-morbid dyslexia and other developmental or neurological disorders. Third, this study included children who scored below the 10th percentile or above the 25th percentile on various literacy measures, omitting children who would typically score between the 11th and 24th percentiles. Identifying a true diagnostic sign of dyslexia would surely require comparisons to the readers from the 11th to 24th percentiles on timed nonword and m-cell tasks during future research.

In addition, this study required average performance on two visual measures (e.g., Directionality and Object Discrimination). As a result, the nonverbal measure Block Construction had a restricted range of performance to 98% of participants scoring in the average range. Restricted nonverbal abilities in the dyslexia and control groups may not fully represent the larger population of children with dyslexia and their age- and reading-matched controls. The small sample size also limits the ability to detect differences

between groups, and even more so when comparing subgroups (e.g., all lower literacy performers).

In addition to limitations with recruitment, specifications in the outcome measure limit the results. Although the specifications of the m-cell task in this study mirrored previous studies, the wider scope of literature on m-cells and dyslexia includes a myriad of specifications. Distance from the screen, speed of the dots, dot lifetimes, and response mechanisms vary widely, making direct comparisons across coherent motion studies difficult.

The inclusion requirement for English-speaking children also limits the results of this study, especially when considering the impact of timed nonword reading for dyslexia diagnosis. Timed nonword reading appeared particularly sensitive and specific in English-speaking children. Other orthographies, especially more transparent ones (e.g., Finnish, Spanish), may not have similar findings as the mastery of a transparent orthography does not involve as many smaller consolidated units for statistical and analogical learning. That is, more transparent orthographies have more consistent phoneme-grapheme units than an opaque orthography like English, which may allow children with dyslexia to show improvements in timed nonword reading similar to the trajectory of timed real word reading in this study. It may be that diagnostic signs of dyslexia vary across orthographies, not because of a difference in an underlying biological cause, but because of the phenotypic expression of dyslexia across orthographies.

Finally, and likely most importantly, this observational study cannot determine causality, including whether dyslexia can be attributed to disturbances with temporal cells

or difficulties analyzing and/or integrating visual-verbal information in the parieto-temporal area. However, it does provide a description of the current characteristics of dyslexic children, their m-cell function, and a new way to approach future research on dyslexia's biological origin through timed nonword reading.

Implications

Despite the limitations, the results of this study can inform diagnostic and educational practice. The results of this study replicate previous findings that measures of phonological awareness alone do not capture the severity of reading difficulties in all children with dyslexia (Wolf & Bowers, 1999). In fact, almost 10% of the dyslexia group scored in the average range on a phonological deletion task and one child showed no difficulties with phonological processing (i.e., phonological awareness and/or rapid naming task). Though the most-widely adopted model of dyslexia diagnosis relies on a phonological difficulty, those in clinical diagnostic practice must be aware that the interaction between a diagnosis window and phonological intervention is complicated. It is possible that children diagnosed at an early age may not continue to express the same underlying phonological processing deficits because of intensive intervention. Therefore, re-evaluations must consider the history of difficulties, the interaction of those difficulties with intervention, and examine other difficulties retained by older dyslexics.

This study identified timed nonsense word reading as one skill deficit that was present in all children with dyslexia. The Phonemic Decoding Efficiency (PDE) from the Test of Word Reading Efficiency (TOWRE-2; Torgesen et al., 2012) measured speeded nonword reading and appeared particularly sensitive for individuals with dyslexia. This sensitivity persisted in older, middle-school children with remediated phonological

difficulties and a sizeable bank of known words. The ability for nonword reading to retain its sensitivity can be traced back, at least in part, to literacy maturity. Nonword reading measures automatic, consolidated phoneme-grapheme knowledge (i.e., literacy maturity) without the top-down influence of vocabulary. That is, even older children with vast oral vocabularies who have drilled common sight words for years struggled to recognize and analyze the orthographic-phonological units in unfamiliar words. Including a measure of timed nonword reading could be beneficial during diagnosis and future research on dyslexia.

Educators can also benefit from the results of this study. The results of this study suggest that many children with dyslexia have a sizeable bank of known real words. Specifically, 13 out of 30 children with dyslexia scored above the bottom quartile on measures of untimed word reading and on measures of timed sight word reading. However, many of these children had underlying difficulties with phonological processing; those with remediated phonological awareness difficulties still struggled with speeded nonword reading. Therefore, many children who may appear “on the bubble” or even “at level” may still need instruction in novel orthographic pattern analysis and sound pattern analysis, measured with timed and untimed nonword reading tasks. Importantly, poor performance on nonword reading assessment should not limit instruction to nonwords; rather, nonwords can be used as a progress monitoring tool for systematic phonics and spelling instruction.

Overall, all parties involved in dyslexia research or practice may view the results of this study as a call for unification in the ongoing debate over visual and phonological theories of dyslexia. That is, the results of this study do not support a one-sided view of

dyslexia's origin as either "Visual" or "Phonological." Rather, the results offer a way to view dyslexic performance on behavioral and imaging tasks as both visual and phonological. All parties may consider an interdisciplinary approach where biological systems are not excluded prematurely. It will take time to uncover the exact role that the visual system plays in dyslexia, but there is no mistaking that reading requires the occipital lobe. Tempered reactions and responses to the study of the visual system and its relationship with dyslexia may bring more awareness to the study of temporal cells and reading difficulties.

Future Directions

This study demonstrated that higher coherence on V5/MT m-cell tasks may not be unique to dyslexia as younger, typically-developing children scored similarly. This study also supported previous evidence that not every child with dyslexia has a unique or deviant difficulty in phonological processing. However, timed nonword reading proved to be a task worth exploring in future studies, with specific attention to the possibility of timed nonword reading serving as a diagnostic sign of dyslexia.

Though this study cannot provide conclusive results on the role of m-cells in dyslexia, a broader measurement design may improve future studies. Specifically, future researchers may consider examining m-cells along the pathway from the retina to area V5/MT and comparing the function of dyslexics to children of the same age and reading levels. It is possible that isolating one location along the dorsal pathway restricts the true relationship between m-cells and dyslexia or m-cells and literacy maturity. In addition, it may be beneficial for m-cell tasks to collect an overall coherence percentage rather than accuracy at a higher level of coherence.

As Goswami (2015) suggested, longitudinal studies would improve our understanding of dyslexia and m-cell function. From this study, a longitudinal follow-up could determine if younger control children improved m-cells over time as they acquired literacy maturity. A longitudinal follow-up could also determine if dyslexic children remained at their current m-cell levels or if they experienced similar gains as their literacy improved. Important questions could address the rate of m-cell acquisition in relationship with the rate of literacy maturity. Longitudinal designs may be more impactful with the inclusion of pre-readers. Similarly, intervention studies could target early gains in literacy maturity while monitoring m-cell growth over time. Longitudinal and intervention studies could chart m-cell development in dyslexia and measure m-cell responsiveness to literacy interventions, with the goal of ruling out or isolating a specific location of m-cells that could be unique to dyslexia. Longitudinal studies for timed nonword acquisition in typically-developing and dyslexic populations could also provide growth curves to examine the early profiles of children who will go on to be diagnosed with dyslexia.

Finally, future studies on temporal cells and dyslexia may benefit from added measures and subgroups for analysis. Additional subgroups could include co-morbid forms of dyslexia, especially the attention-dyslexia interaction. At minimum, studies may include measures of rapid auditory processing in addition to visual m-cell function. A wider scope of biological systems and their temporal cell function could provide data to examine the role of auditory, visual, and phonological systems in the same sample of children with dyslexia and matched peers.

Conclusion

Research to improve dyslexia diagnosis searches for a unique characteristic among thousands of options. Researchers have access to cortical imaging, behavioral studies, and case studies, yet dyslexia still lacks a definitive diagnostic sign. Without a widely-agreed upon diagnostic sign, researchers may not include and exclude the right participants during studies that search for a biological cause. Further, without a widely-agreed upon diagnostic sign, diagnosticians and educators may not know the potential predictive characteristics of dyslexia that could increase the access to and likelihood of early intervention. The major goal of this study was to determine the usefulness of m-cells as a diagnostic sign of dyslexia. It appears that weakened m-cells do not impact everyone with significant literacy difficulties, including those with dyslexia, thus questioning its use as a diagnostic sign of dyslexia. However, timed nonword reading appeared as a likely diagnostic sign, and given the biological basis of timed nonword reading, the role of temporal cells in dyslexia could not be ruled out. The challenge ahead for the dyslexia research community is to improve diagnostic power to accurately identify who has dyslexia, or to at least agree on a consistent approach, such as timed nonword reading, that could move the field ahead. Then researchers can take on the hefty task of teasing apart the nuanced biological causes, including potential contributions from temporal cells like magnocellular cells.

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Appendix A

TEXT OR CALL 540-312-4645

RESEARCH STUDY ON DYSLEXIA AND THE BRAIN IN CHILDREN

Seeking children with a dyslexia diagnosis between the ages of 8 to 14 to participate in a brief assessment for a study on the brain and reading conducted by the University of Virginia.

- One in-person testing appointment at a **library in your town**
- Approximately one hour of your time

No travel required
One hour appointment

Principal Investigator

- Amanda Rawlins, M.Ed., M.T.
- PhD Candidate, Reading Education
- University of Virginia
- 540-312-4645
- UVA IRB-SBS # 2677

UNIVERSITY of VIRGINIA

Appendix B

TEXT OR CALL 540-312-4645

RESEARCH STUDY ON READING AND THE BRAIN IN CHILDREN

Seeking children between the ages of 6 to 14 to participate in a brief assessment for a study on the brain and reading conducted by the University of Virginia.

- One in-person testing appointment at a **library in your town**
- Approximately one hour of your time

No travel required
One hour appointment

Principal Investigator

- Amanda Rawlins, M.Ed., M.T.
- PhD Candidate, Reading Education
- University of Virginia
- 540-312-4645
- UVA IRB-SBS # 2677

UNIVERSITY of VIRGINIA

Appendix C

Institutional Review Board for Social & Behavioral Sciences
University of Virginia

Office of the Vice President for Research

Human Research Protection Program

Institutional Review Board for the Social and Behavioral Sciences

IRB-SBS Chair: Moon, Tonya

IRB-SBS Director: Blackwood, Bronwyn

Protocol Number (2677) Approval Certificate

The UVA IRB-SBS reviewed "Literacy Maturity and the Magnocellular Theory of Dyslexia: Implications for Clinical Diagnosis" and determined that the protocol met the qualifications for approval as described in 45 CFR 46.

Principal Investigator: Rawlins, Amanda

Faculty Sponsor: Youngs, Peter

Protocol Number: 2677

Protocol Title: Literacy Maturity and the Magnocellular Theory of Dyslexia: Implications for Clinical Diagnosis

Is this research funded? Yes

Funding Source(s): UVA Departmental/Local

All Agency Grant Numbers & Titles currently associated with this protocol:

I received a grant from the Curry School of Education "IDEAs Grant Program" in the amount of $956.26. The Curry School pays for the items requested directly and the money is not given to any member of the research team.

Review category: Expedited Review

6. Collection of data from voice, video, digital, or image recordings made for research purposes
7. Research on individual or group characteristics or behavior or research employing survey, interview, oral history, focus group, program evaluation, human factors evaluation, or quality assurance methodologies

Review Type:

Modifications: Yes
Continuation: No
Unexpected Adverse Events: No

Approval Date: 2019-08-21

As indicated in the Principal Investigator, Faculty Sponsor, and Department Chair Assurances as part of the IRB requirements for approval, the PI has ultimate responsibility for the conduct of the study, the ethical performance of the project, the protection of the rights and welfare of human subjects, and strict adherence to any stipulations imposed by the IRB-SBS.

The PI and research team will comply with all UVA policies and procedures, as well as with all applicable Federal, State, and local laws regarding the protection of human subjects in research, including, but not limited to, the following:

1. That no participants will be recruited or data accessed under the protocol until the Investigator has received this approval certificate.
2. That no participants will be recruited or entered under the protocol until all researchers for the project including the Faculty Sponsor have completed their human investigation research ethics educational requirement (CITI training is required every 4 years for UVA researchers). The PI ensures that all personnel performing the project are qualified, appropriately trained, and will adhere to the provisions of the approved protocol.
3. That any modifications of the protocol or consent form will not be implemented without prior written approval from the IRB-SBS Chair or designee except when necessary to eliminate immediate hazards to the participants.
4. That any deviation from the protocol and/or consent form that is serious, unexpected and related to the study or a death occurring during the study will be reported promptly to the SBS Review Board in writing.
5. That all protocol forms for continuations of this protocol will be completed and returned within the time limit stated on the renewal notification letter.
6. That all participants will be recruited and consented as stated in the protocol approved or exempted by the IRB-SBS board. If written consent is required, all participants will be consented by signing a copy of the consent form unless this requirement is waived by the board.
7. That the IRB-SBS office will be notified within 30 days of a change in the Principal Investigator for the study.
8. That the IRB-SBS office will be notified when the active study is complete.
9. The SBS Review Board reserves the right to suspend and/or terminate this study at any time if, in its opinion, (1) the risks of further research are prohibitive, or (2) the above agreement is breached.

Date this Protocol Approval Certificate was generated: 2019-08-26

Appendix D

Additional results tables.

Table D1

Unusual Data Points: M-cell Function

	Model 1			Model 2 No Outliers			Model 3 Robust Reg	
Variable	*b*	*SE b*	*b**	*b*	*SE b*	*b**	*b*	*SE b*
Age-Matched	-6.13*	2.43	-.28	-4.51	2.27	-.22	-5.31	2.72
Reading-Matched	1.82	2.88	.08	4.04	2.70	.19	2.42	2.72
Nonverbal Ability	-0.70	0.42	-.16	-0.90*	0.40	-.22	-0.75	0.46
Dyslexia [Constant]	23.68***	2.11		22.09***	1.92		22.82***	1.93
adjusted R^2		.11			.15			.09
RMSE		9.83			9.19			10.50
F		6.56			7.15			3.90
N		90			87			90

*$p < .05$. **$p < .01$

Table D2

M-cell Function by Phonological Ability in Dyslexia (n=30)

		Phonological Ability		
		Average	Below	Poor
M-cell ability	Average or better	4	4	4
	Below average	5	7	6

Note. Average m-cell ability = 17.46% coherence

Table D3

Exploratory Analysis of Timed Nonword Reading (z PDE)

	Model 1 Groups			Model 2 RAN			Model 3 RAN + zLWI			Model 4 zSWE			Model 5 zSWE + zElision		
Variable	*b*	*SE b*	*b**	*b*	*SE b*	*b**	*b*	*SE b*	*b**	*b*	*SE b*	*b**	*b*	*SE b*	*b**
Age-Matched	1.88***	0.15	.89	1.64***	0.18	.78	0.91***	0.13	.43	1.16***	0.11	.55	1.08***	0.11	.51
Reading-Matched	0.40**	0.15	.19	0.16	0.18	.07	0.41***	0.12	.19	0.60***	0.10	.29	0.53***	0.10	.25
RAN				0.01*	0.01	.18	0.01*	0.00	.61						
z LWI							0.62***	0.05	.12						
z SWE										0.60***	0.05	.60	0.51***	0.06	.51
z Elision													0.16***	0.05	.16
Dyslexia [Constant]	-0.76***	0.11		-1.76***	0.46		-1.22***	0.29		-0.59***	0.07		-0.05***	0.07	
adjusted R^2		.65			.67			.86			.86			.87	
RMSE		0.59			0.56			0.37			0.37			0.36	
F		83.54			83.04			144.43			186.61			154.09	
N		90			90			90			90			90	

Note. z PDE = Phonemic Decoding Efficiency z-score; RAN = Rapid Symbolic Naming; z LWI = Letter Word Identification z-score; z SWE = Sight Word Efficiency z-score.
*$p < .05$. **$p < .01$ ***$p < .001$

Table D4

Unusual Data Points: Timed Nonword Reading

	Model 1			Model 2 No outliers			Model 3 Robust regression		
Variable	*b*	*SE b*	*b**	*b*	*SE b*	*b**	*b*	*SE b*	*b**
Age-Matched	1.08***	0.11	.51	0.62***	0.12	.30	0.65***	0.14	
Reading-Matched	0.53***	0.10	.25	0.53***	0.10	.25	0.56***	0.11	
z SWE	0.51***	0.06	.51	0.33***	0.06	.34	0.34***	0.07	
zSWE*AgeMatch				0.70***	0.12	.37	0.70***	0.14	
zSWE*ReadingMatch				0.15	0.09	.08	0.16	0.10	
z Elision	0.16***	0.05	.16	0.15***	0.04	.16	0.15	0.05	
Dyslexia [Constant]	-0.05***	0.07		-0.56***	0.06		-0.58***	0.07	
adjusted R^2	.87			.92			.89		
RMSE	0.36			0.28			0.33		
F	154.09			163.24			123.80		
N	90			87			90		

Appendix E

Trajectories for relevant variables are presented by group.

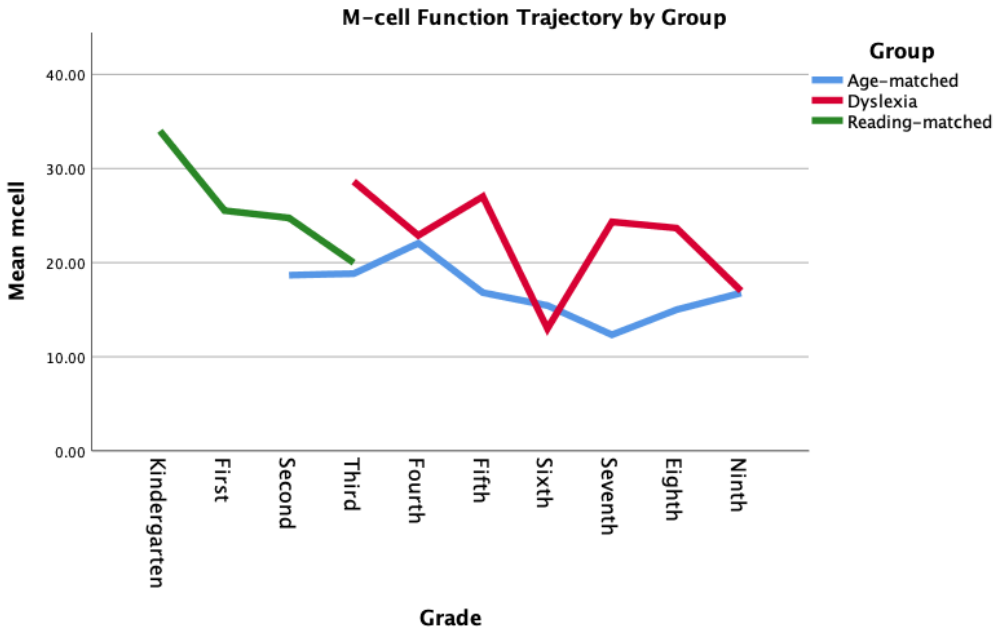

Figure E1. M-cell Trajectory by Group (N = 90)

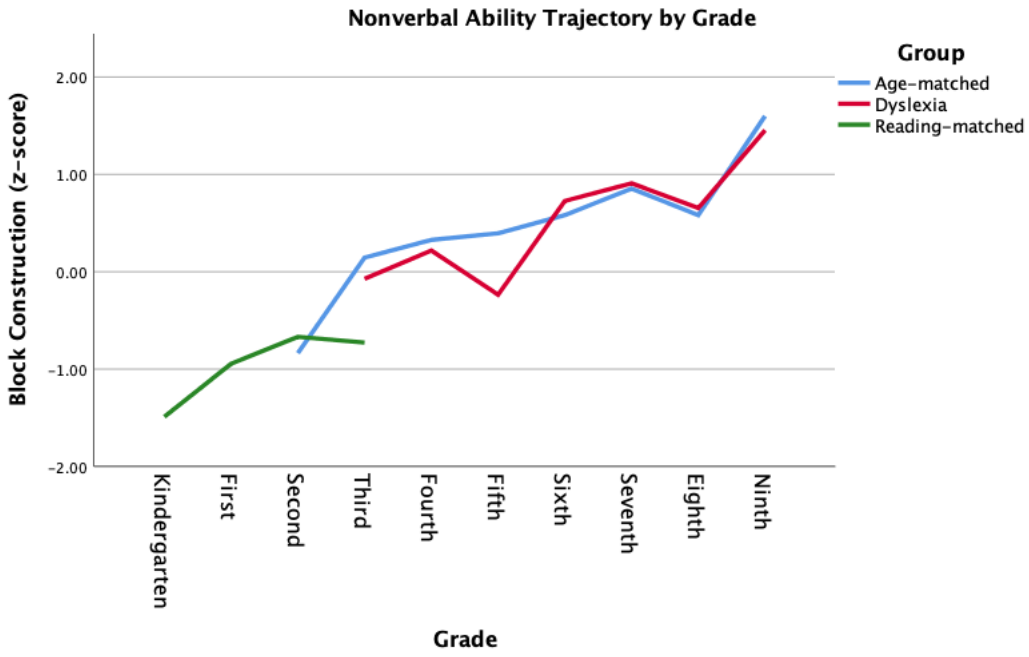

Figure E2. Nonverbal Ability Trajectory by Group (N = 90)

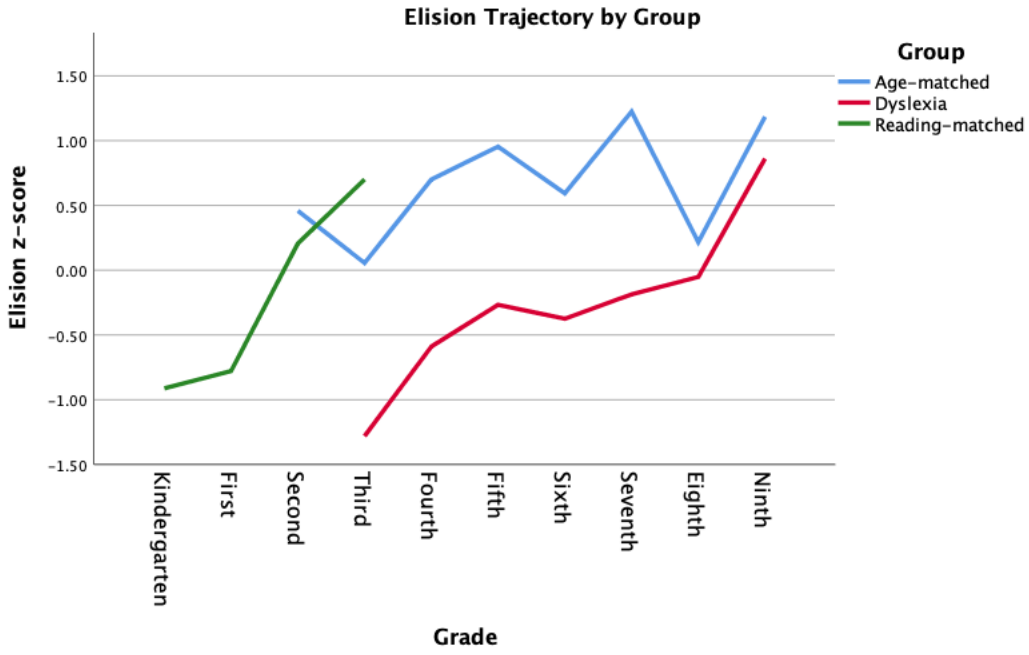

Figure E3. Phonological deletion (i.e., Elision) Trajectory by Group (N = 90)

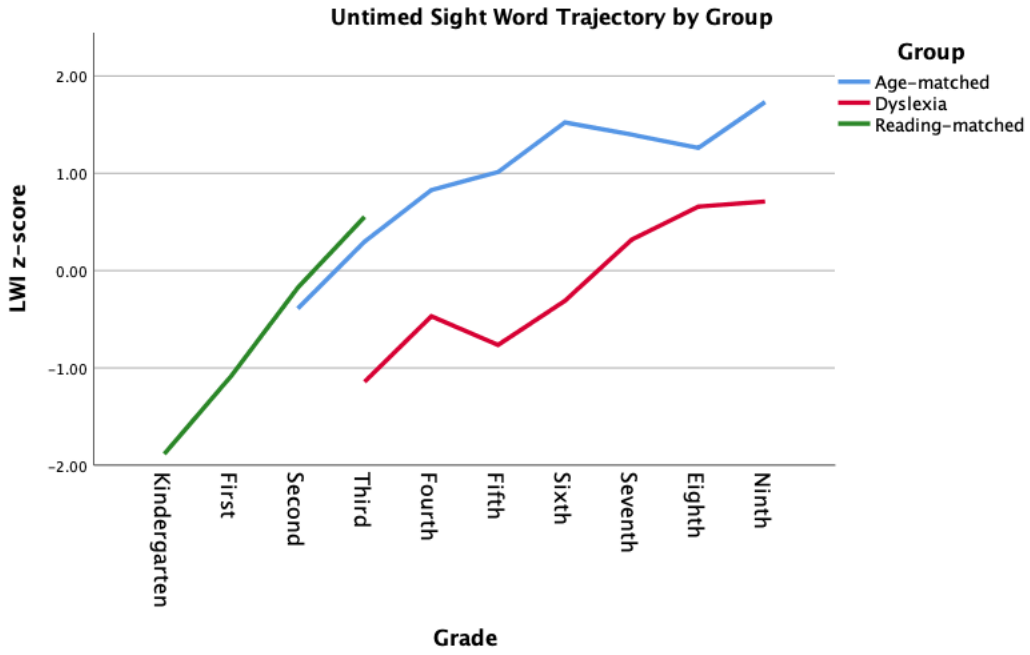

Figure E4. Untimed Sight Word Trajectory by Group (N = 90)

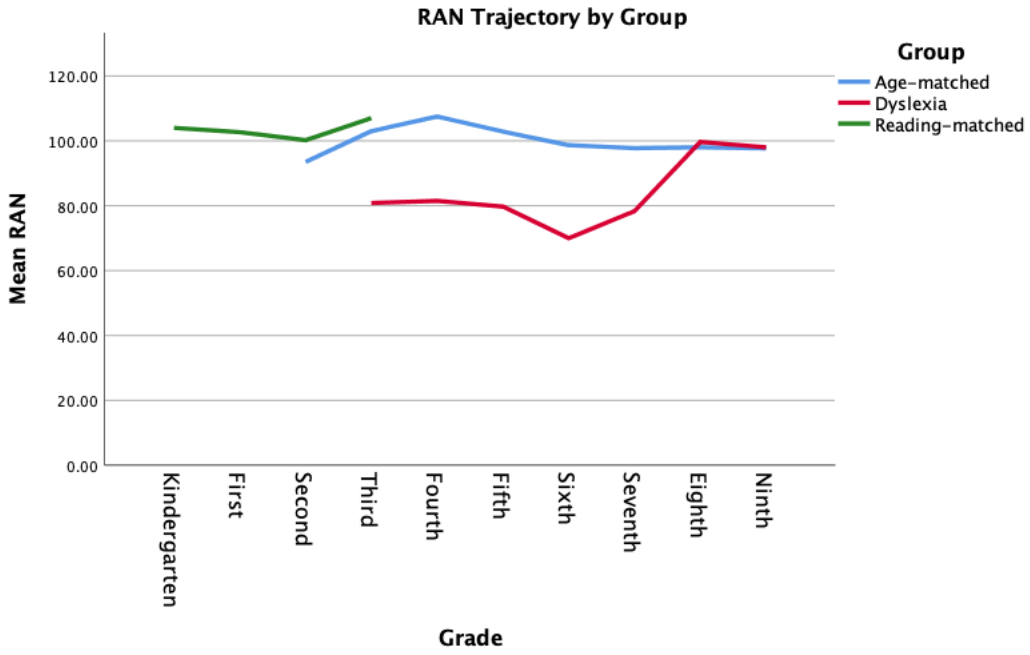

Figure E5. Rapid Symbolic Naming (RAN) Trajectory by Group (N = 90)

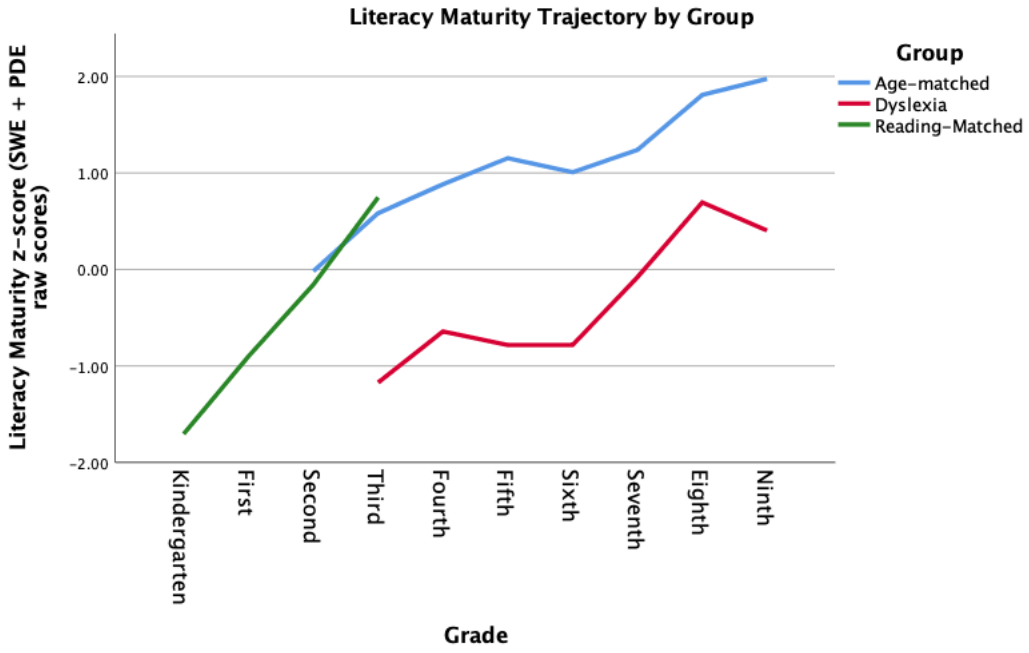

Figure E6. Literacy Maturity Trajectory by Group (N = 90)

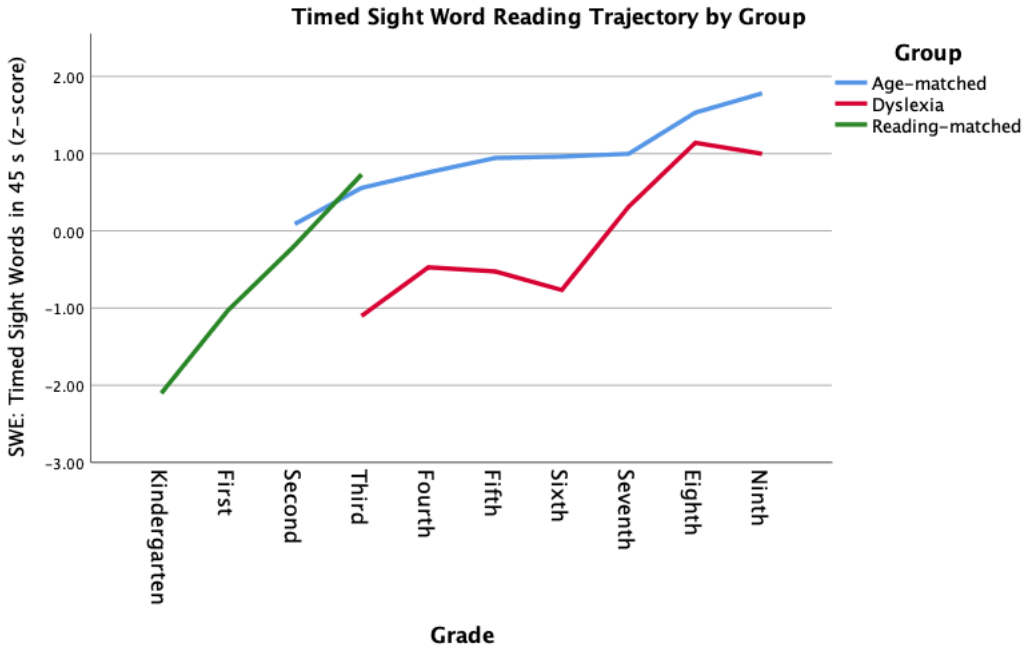

Figure E7. Timed Sight Word Reading Trajectory by Group (N = 90)

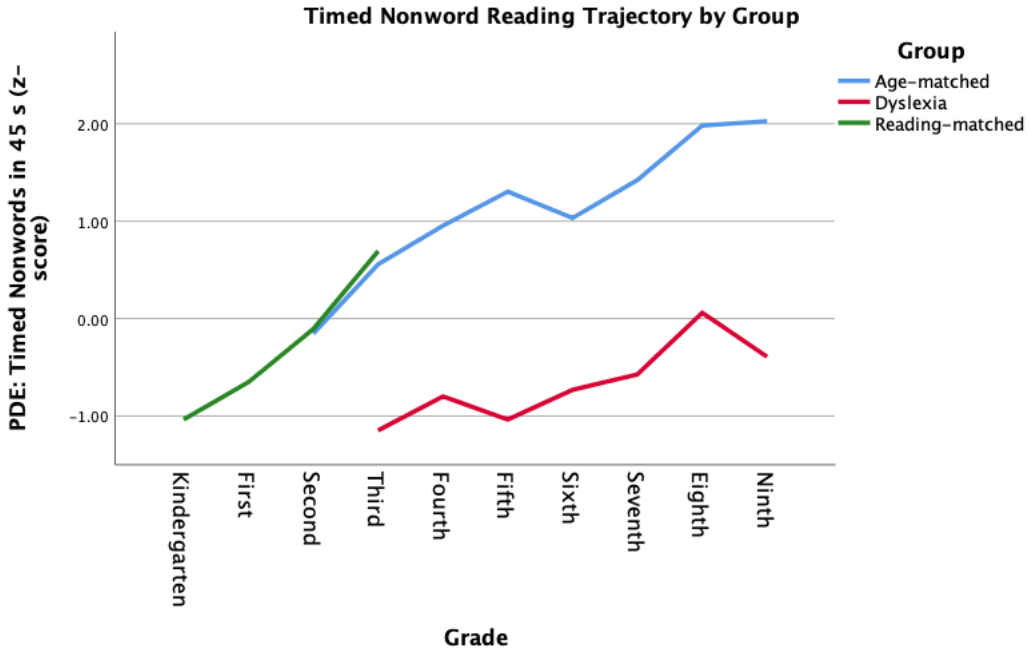

Figure E8. Timed Nonword Word Reading Trajectory by Group (N = 90)