Evidence from psychophysical, physiological and neuroimaging studies suggests that functioning of the magnocellular pathway, which is specialized for processing moving stimuli at low-contrast conditions, correlates with reading performance. Nevertheless, the magnocellular-deficit hypothesis remains somewhat controversial. Some researchers continue to publish conflicting evidence, although differences in subject selection criteria and visual stimuli may contribute to discrepant findings. Recent functional neuroimaging studies provide compelling evidence that, in response to moving stimuli at low-contrast conditions, dyslexics evidence a characteristic pattern of physiological under-activation in V5 (MT), an extrastriate area predominated by magnocellular input. Results from this psychophysical study support the hypothesized relationship between magnocellular function and reading ability for adults, but not for children and adolescents. The psychophysical stimuli used in this study might help to advance neuroimaging of early visual correlates with adults. Conversely, these stimuli offer questionable utility to the development of screening instruments that might one day predict developmental dyslexia with young children.

INDEX WORDS: Early Visual Processing, Magnocellular Pathway, Developmental Dyslexia
THE RELATIONSHIP BETWEEN EARLY VISUAL PROCESSING
AND READING ABILITY: INVESTIGATION OF TEMPORAL
PROCESSING IN ADULTS AND CHILDREN

by

AARON E. SKALICKY
B.S., University of Notre Dame, 1996
M.S., Loyola College, 1998

A Dissertation Submitted to the Graduate Faculty of The University of Georgia
in Partial Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA
2002
THE RELATIONSHIP BETWEEN EARLY VISUAL PROCESSING AND READING ABILITY: INVESTIGATION OF TEMPORAL PROCESSING IN ADULTS AND CHILDREN

by

AARON E. SKALICKY

Approved:

Major Professor: George Hynd

Committee: James Brown
            Randy Kamphaus
            L. Steve Miller
            Paul Shutz

Electronic Version Approved:

Gordhan L. Patel
Dean of the Graduate School
The University of Georgia
May 2002
DEDICATION

This work is dedicated to the many who never doubted my God-given abilities and resolve. At the top of the list is Cindy, my best friend and future wife, who provided love, encouragement, and patience during this project. Even from a distance, her support never wavered. I hope to return the same uplifting spirit during our life together.

My father, Emil, and my mother, Dorothy, handed me a limitless world of opportunity when they helped to make my Notre Dame experience a reality. Simply stated, this Ph.D. is one more reflection of their accomplishments as parents.

My sisters, Beth and Sara, have shared visits, phone calls, and occasional statistical advice with me since I initiated this project. I learned, once again, just how easy it is to maintain perspective when you are grounded in family. Beth and Sara continue to provide an example of how to do things the right way in both personal and professional life.

My newest family member, Chris, selflessly put his occupation aside to ensure that I was getting my fix of NCAA basketball during my “working” vacation in Los Angeles. Without him, I would not have realized that it was the wrong time to start writing. And finally, I must recognize a good friend and trusted technical advisor. Without David’s guidance, my home computer may never have taken to SPSS. Turn off your cell phone, Mr. Crooker, you’re officially off retainer.
ACKNOWLEDGEMENTS

I have been fortunate to work with a man at the University of Georgia who is regarded with equally high praise by his students, his staff, and his colleagues around the globe. I am no longer amazed with the kind words I receive at international and national conferences, or on internship in Texas, when people learn the identity of my mentor. From day one, Dr. Hynd has facilitated my doctoral training through meticulous organization, high expectations, and trust. I enjoy our conversations, whether about child neuropsychology, professional issues, travel, football, or life in general. Even in light of this man’s formidable reputation and professional accomplishments, I am most impressed by the respect he shows people who study and work at U.G.A. I will count myself among the many graduates who consider Dr. Hynd a tremendous resource and a valued friend.

I also wish to acknowledge the other distinguished members of my dissertation committee: Dr. James Brown, Dr. Randy Kamphaus, Dr. Steve Miller, and Dr. Paul Schutz. Since I was destined to move away for internship during the latter half of the project, the feedback they provided during the prospectus stage was extremely important. I came to expect clear and focused comments from each of these men. Assembling this group of diverse academic talents enabled me to work confidently in Texas.

Finally, I wish to thank everyone in Dr. Hynd’s office who welcomed me to Georgia in 1999 and graciously met my every request during the past three years. Mrs. Connie Morse and Ms. Julie Patton, in particular, are two wonderful people who do their jobs exceptionally well. Their helpfulness, humor, and kind words were missed as soon
as I left for Texas. Carlin, Lanee, Jason, Julie, Richard, and Mary have all contributed to
the positive and engaging work environment at the Center for Clinical and
Developmental Neuropsychology. Special thanks go to the graduate students who
facilitated this project through data collection and data base management. It has been a
pleasure to work with you all in the clinic.
# TABLE OF CONTENTS

**ACKNOWLEDGEMENTS** ........................................................................................................................................... v

**LIST OF TABLES** ..................................................................................................................................................... x

**LIST OF FIGURES** .................................................................................................................................................... xi

**CHAPTER**

1 **INTRODUCTION** ................................................................................................................................................... 1

   Overview of the Problem ........................................................................................................................................ 2

   Significance of the Study ...................................................................................................................................... 5

   Summary ............................................................................................................................................................. 8

2 **REVIEW OF THE LITERATURE** ........................................................................................................................... 9

   Developmental Dyslexia ..................................................................................................................................... 9

   Parallel Systems ............................................................................................................................................... 12

   Dorsal and Ventral Streams ............................................................................................................................. 18

   V5 as an Indicator of Magnocellular Dysfunction ...................................................................................... 20

   Magnocellular and Parvocellular Mechanisms in Reading ........................................................................... 21

   Psychophysical Investigation of Magnocellular Deficit Hypothesis ............................................................... 24

   Group Inclusion Criteria ................................................................................................................................... 26

   Stimulus Properties .......................................................................................................................................... 30

   Functional Magnetic Resonance Imaging (fMRI) ............................................................................................ 33

   Developmental Considerations .......................................................................................................................... 39
Conclusions..................................................................................................................41

3 METHODOLOGY .......................................................................................................43

Subjects .........................................................................................................................43

Procedure .......................................................................................................................44

Gray Temporal Dots .......................................................................................................45

Red Temporal Dots .........................................................................................................46

Wechsler Abbreviated Scale of Intelligence .................................................................46

Woodcock Reading Mastery Test – Revised (WRMT-R) ...........................................47

Gray Oral Reading Test – 3rd Edition (GORT-3) .........................................................47

Woodcock-Johnson Tests of Cognitive Abilities- Revised ..........................................48

Comprehensive Test of Phonological Processing .......................................................48

Analyses ..........................................................................................................................49

4 RESULTS ....................................................................................................................50

Hypothesis I .....................................................................................................................51

Hypothesis II ....................................................................................................................59

Hypothesis III ...................................................................................................................61

5 DISCUSSION ...............................................................................................................63

Relationship Between Magnocellular Functioning and Reading Ability ....................63

Utility of a Comparison Visual Measure with a Diffuse Red Background .....................64

Global Temporal Processing Deficits ...........................................................................65

Limitations of this Study ...............................................................................................66

Directions for Further Research ..................................................................................69
REFERENCES ................................................................. 73
APPENDICES ............................................................ 88
Appendix A ................................................................. 89
Appendix B ................................................................. 91
LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Summary of Subject Characteristics in Functional Magnetic Resonance Imaging (fMRI) Studies of Visual Processing in Dyslexia</td>
<td>35</td>
</tr>
<tr>
<td>2</td>
<td>Summary of Results of Functional Magnetic Resonance Imaging (fMRI) Studies of Visual Processing in Dyslexia</td>
<td>36</td>
</tr>
<tr>
<td>3</td>
<td>Means and Standard Deviations for Age and Performance Variables for the Adult and Child Groups</td>
<td>52</td>
</tr>
</tbody>
</table>
## LIST OF FIGURES

<table>
<thead>
<tr>
<th>FIGURE</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Cross Section of the Macaque Lateral Geniculate Nucleus</td>
<td>14</td>
</tr>
<tr>
<td>2</td>
<td>Receptive Fields for Typical Color-Opponent Parvocellular Geniculate Neuron and Broadband Magnocellular Neuron</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>Diagrammatic Representation of the Magnocellular and Parvocellular Systems from the LGN to Specialized Areas of the Striate and Extrastriate Cortex</td>
<td>17</td>
</tr>
<tr>
<td>4</td>
<td>Broad Distribution of Ventral and Dorsal Systems of Higher-Level Visual Processing Systems</td>
<td>18</td>
</tr>
<tr>
<td>5</td>
<td>Transient and Sustained Interactions are Hypothesized to Interact During Fixations</td>
<td>23</td>
</tr>
<tr>
<td>6</td>
<td>Hypothesized Interaction between Sustained and Transient Systems</td>
<td>23</td>
</tr>
<tr>
<td>7</td>
<td>Group Differences in Activation between a Dyslexic Group and a Control Group as Revealed by fMRI</td>
<td>34</td>
</tr>
<tr>
<td>8</td>
<td>Slices of Region MT+ in Three Dyslexic Subjects</td>
<td>38</td>
</tr>
<tr>
<td>9</td>
<td>Adult Reading Ability and Performance on the Psychophysical Measure Designed to Assess Magnocellular Functioning</td>
<td>54</td>
</tr>
<tr>
<td>10</td>
<td>Adult Reading Ability and Performance on the Complimentary Psychophysical Measure Designed to Incorporate Magnocellular Suppression</td>
<td>55</td>
</tr>
<tr>
<td>11</td>
<td>Children’s Reading Ability and Performance on the Psychophysical Measure Designed to Assess Magnocellular Functioning</td>
<td>56</td>
</tr>
<tr>
<td>12</td>
<td>Children’s Reading Ability and Performance on the Complimentary Psychophysical Measure Designed to Incorporate Magnocellular Suppression</td>
<td>57</td>
</tr>
</tbody>
</table>
CHAPTER 1
INTRODUCTION

Developmental dyslexia was originally described as a clinical syndrome during the late nineteenth century (Morgan, 1896), and it is currently regarded to be the most prevalent of all learning disabilities (Hynd, Hooper & Takahashi, 1998). Contributions from the fields of behavioral neurology and neuropsychology have advanced the notion that reading problems may be one manifestation of a more global neurodevelopmental symptom complex. Hughes and Denkla (1978) supported this position when they comprehensively assessed poor readers and reported common profiles that included hyperactivity, dyscalculia and motor incoordination. Since that time, the list of suspected behavioral manifestations associated with dyslexia has expanded to include temporal processing deficits related to both vision (Eden et al., 1996; Lehmkuhle et al., 1992; Livingstone et al., 1991) and audition (Tallal et al., 1993).

As the development of reading skills assumes greater emphasis in early education during this “information age” (Eden & Zeffirino, 2000), the educational impact of reading disabilities warrants greater attention than was afforded in past generations. Most directly, failure to achieve reading competency limits a child’s ability to acquire information in most academic subjects. The less obvious effects upon social, emotional and behavioral development are only beginning to emerge. While few researchers have examined the affects of developmental dyslexia across the life-span, there is initial
evidence that learning disabilities are significantly related to psychosocial deficits in coping, relationship formation, and pragmatic language (Gresham & Reschly, 1986).

Given the primacy of these psychosocial abilities in overall adaptive functioning, it follows that reading disabilities may negatively impact personal and professional development into adulthood. In order to minimize these negative outcomes, research has progressed significantly in understanding some of the underlying neural substrates of developmental dyslexia. At this time; however, most findings relate to adult presentations of dyslexia and offer little to advance identification and intervention at the “critical” periods of child development, when the component skills required for proficient reading are believed to be most easily acquired (Lyons, et al., 1995). Thus, there is a need for early and reliable identification of children who are likely to exhibit developmental dyslexia, as well as a need for the development of corresponding interventions.

Novel identification and intervention techniques should be based upon the continuing empirical evidence for characteristic temporal processing deficits in developmental dyslexia. This study aims to build upon the temporal processing literature by examining the relationship between early visual processing, reading skills, and related neuropsychological variables within a sample of children and a sample of adults.

Overview of the Problem

The body of literature related to temporal processing ability and reading disability, as measured with psychophysical means, is relatively new and has only benefited from the guidance of complimentary neuroimaging studies for a few years. The literature, with regard to children specifically, is even less evolved. When reviewed within the context of empirical neuroscience and neuropsychology, there are several
examples of research designs and methodologies that do not optimally examine the constructs of interest. These problems are predominantly rooted in stimulus inconsistency, questionable group selection criteria, and failure to use a control measure.

Psychophysical investigation of motion sensitivity has yielded initial support for the existence of early visual deficiencies for adults (Eden et al., 1995; Demb et al., 1998) and children (Cornelissen, 1997) who experience significant reading difficulties. While the preponderance of studies in this field provide reasonable, neuroscientific justification for their choice of stimuli, and claim to measure a unitary construct referred to as “early visual processing,” this body of literature lacks overall consistency in methodology. If the body of literature was more extensive, and conclusive, this would not be targeted as a primary area for improvement. However, the effects of subtle differences in stimulus presentation at this stage, whether truly significant or insignificant, are simply unknown. To solve this problem, and facilitate a more meaningful synthesis of findings related to temporal processing and reading ability, more replication of stimuli is needed.

The second problem in the literature involves the continued use of questionable group selection criteria. There is a natural tendency, it seems, to rely upon a cut-off score to define “affected” and “unaffected” populations, even when a clinical syndrome under investigation is characterized by continuous, rather than discrete, behavioral variables. Analysis of group differences can be very useful, but only when inclusion criteria is consistent with theory and creates meaningfully divergent groups. To date, group comparisons predominate the investigation of temporal processing and reading ability. Unfortunately, most of these comparisons have been based upon an unsubstantiated belief that the discrepancy between overall I.Q. and reading achievement can define a dyslexic
versus a non-dyslexic population. While this schema has served a pragmatic function to the gatekeepers of public special education services in the United States, its validity in defining meaningfully divergent populations and determining who is likely to benefit from intervention is strongly criticized (Fletcher, 1998; Velluntino et al., 1996). An alternative schema that de-emphasizes overall intellectual ability as a group criterion (Shaywitz & Shaywitz, 1992) initiated useful debate but also failed to attain consensus.

Lost in most group comparisons using only reading achievement and I.Q. are other neuropsychological variables that have demonstrated a relationship with reading disabilities. One example is the finding that reading difficulty, early visual deficits, and phonological processing difficulties are significantly related (Borsting, 1996). Part of the solution to the problem of questionable group criteria can be achieved if studies collect a more diverse range of temporal processing variables, such as early visual processing and phonological processing, to complement reading performance and an estimate of I.Q. Analysis of the relationships between these variables must be explored more extensively to abate the current over-reliance upon group comparisons. Given the range of continuous scores for these performance variables, regression analysis is the most appropriate manner to investigate potential neurocognitive “markers” of reading disability.

Finally, comparison of perceptual ability between dyslexic and normal reading groups has utilized stimuli designed only to enhance magnocellular activation. While the results from these studies generally suggest that individuals with reading disability experience a relative difficulty detecting visual motion, they have not convincingly identified the magnocellular pathway as the compromised mechanism. Most notably, there has been insufficient effort to rule out cognitive and developmental factors that may
significantly affect performance on psychophysical measures. While parvocellular visual neurons are known to be selective for specific wavelengths, and possess the capacity to inhibit magnocellular visual neurons when activated (Livingstone & Hubel, 1988), these wavelengths have not been integrated into complimentary psychophysical measures.

**Significance of the Study**

This study contributes to the body of literature pertaining to temporal processing ability and reading performance in several important ways. The areas of significance were formulated in direct response to the weaknesses described in the preceding section. They include meaningful comparison with similar investigations, data analysis that de-emphasizes group comparisons that are not empirically supported, and inclusion of a psychophysical control measure that is empirically supported and under-utilized in this field of study.

As described above, seemingly inconsistent findings in the literature are currently difficult to reconcile because several similar psychophysical measures have been utilized to assess early visual processing (Eden et al., 1995; Cornelissen, 1997; Demb et al., 1998). After reviewing these and other experiments aimed at activation of the magnocellular pathway, all variations of stimuli appeared, by description, to be highly consistent with visual theory. Since there was no clear superiority in design, the stimuli and subject positioning described by Eden and colleagues (1995) was chosen for replication in this study because it is described in a manner judged to best facilitate accurate replication. This investigation will therefore enable a more direct comparison with earlier findings than is presently available between laboratories.
Currently, there is no single published study of early visual processing and neurolinguistic ability that includes children and adults and permits analysis of the relationships between reading component skills, intellectual ability, and temporally-mediated early visual processing. By utilizing a sample that includes children and adults of diverse reading ability, this study can provide unique insight into the role of development upon the relationships between neuropsychological functioning and early visual processing.

Finally, this study is significant in design because a complimentary psychophysical measure is included to help determine the role of magnocellular dysfunction on reading performance with more certainty than has been possible in previous designs. Adhering to all other psychophysical properties (e.g., stimulus presentation at low mean luminance) and nuances of subject positioning (e.g., standard viewing distance) included in the published description of Eden and colleagues’ (1995) “Temporal Dots” task, the complementary measure in this study was created with a diffuse red background rather than the original gray background.

In earlier studies, deficient magnocellular functioning was posited when an individual experienced difficulty on the experimental measure. In this design, it is possible to compare an individual’s performance on the experimental task with his or her performance on another task that presents similar cognitive challenges (e.g., decision making) but requires sensory processing that is not wholly predominated by the magnocellular pathway. This design limits presumptive statements of causality should the experimental and control measures produce consistent results. Conversely, when a subject performs poorly on the experimental measure and significantly better on the
control measure, magnocellular dysfunction can be inferred as the reason for perceptual difficulty with greater confidence than has been permitted by previous psychophysical designs. The following predictions are made:

1. Analysis of early visual processing and passage reading will support the hypothesized relationship between magnocellular dysfunction and reading difficulty. In the child and adult samples, performance on the magnocellular-dependent task “Gray Temporal Dots” will correlate positively with, and account for significant variance in performance on a measure of passage reading. This analysis will also consider relevant indices of intellectual and neurolinguistic ability.

2. Performance on the “Gray Temporal Dots” measure and performance on the corresponding control measure, “Red Temporal Dots,” will be analyzed in relation to reading ability to investigate the clinical utility of the novel “Red Temporal Dots” measure. A diffuse red background has been shown to inhibit the selective perceptual properties of M cells. Thus, positing that magnocellular functioning is significantly related to reading ability, performance on the “Gray Temporal Dots” task is expected to surpass performance on the “Red Temporal Dots” task in accounting for variance in passage reading.

3. There is a hypothesized convergence between the neuroanatomical pathways that enable visual motion processing and auditory processing of language. The former innervates the inferior parietal cortex and the latter traverses regions proximal to the inferior parietal cortex, such as the posterior superior temporal gyrus, the inferior parietal lobule, and the angular gyrus. Consistent with the theory that a common developmental perturbation may account for deficits in the temporal processing of visual and auditory
stimuli, performance on measures of phonological processing and sequential auditory processing are expected to account for significant variance in subject performance on the “Gray Temporal Dots” task.

Summary

Chapter I has presented an introduction to the problems, which include the uncertain relationship between temporal processing and reading impairment, and the unknown utility of an experimental measure of early visual processing. Following a description of this study’s significance, specific predictions were stated regarding the outcome of this investigation. Chapter II will review the relevant literature and Chapter III will provide an overview of the methodology that was used. Chapter IV will describe the results of statistical analyses and Chapter V will discuss implications for future investigation.
CHAPTER 2
REVIEW OF THE LITERATURE

This chapter addresses the relationship between developmental dyslexia and early visual processing. Evidence from psychophysical, physiological and neuroimaging studies suggests that functioning of the magnocellular pathway, which is specialized for processing moving stimuli at low-contrast conditions, correlates with reading performance. Nevertheless, the magnocellular-deficit hypothesis remains somewhat controversial. Some researchers continue to publish conflicting evidence, although differences in subject selection criteria and visual stimuli may contribute to discrepant findings. Results of recent functional neuroimaging studies provide compelling evidence that, in response to moving stimuli at low-contrast conditions, dyslexics evidence a characteristic pattern of physiological under-activation in V5 (MT), an extrastriate area predominated by magnocellular input. In the future, functional imaging may help to establish magnocellular function as a reliable marker of dyslexia or dyslexia subtype.

Developmental Dyslexia

Developmental dyslexia has been conceptualized as a “specific language based disorder of constitutional origin, characterized by difficulties in single-word decoding, usually reflecting insufficient phonological processing abilities” (Operational definition of the Orton Dyslexia Society Research Committee, April 18, 1994, as reported in B. A. Shaywitz et al., 1995). Some researchers report that 3-9% of school-age children are affected (Rutter & Yule, 1975), although more recent estimates suggest that no more than 6% of children adequately meet diagnostic criteria (Hynd et al., 1998). Analysis of the
linguistic abilities of poor readers reveals characteristic deficits related to phonological awareness (Bruce, 1964; Scarborough, 1991; Snowling, 1980) and verbal fluency (Denkla & Rudel, 1976). Due to the relative consistency of these findings, linguistic processes have served as the foci for neuropsychological investigation of developmental dyslexia. While this emphasis is empirically supported, competing, and potentially complimentary, theories have not been afforded equal scrutiny. As the investigation of developmental dyslexia has been drawn predominantly towards the neurolinguistic domain, additional avenues of inquiry that may shed light upon the origins of reading deficits, or lead to more accurate nosology, have been prematurely de-emphasized.

One such alternative is the hypothesis that a global temporal-processing deficit, encompassing visual and auditory processing of rapidly presented information, underlies a significant proportion of reading impairments. Even though evidence for characteristic sensory deficits is far from conclusive, there is sufficient reason to remove “sensory deficits” from the widely accepted list of exclusionary criteria for a reading disability (American Psychiatric Association, 1994; Rutter & Yule, 1975) where it currently accompanies “poor motivation, inadequate educational environment, and acquired brain lesion.”

The belief that reading disabilities occur within a relatively wide array of neurolinguistic profiles is widely accepted. An occasional peer-reviewed study will even go so far as to use the plural “developmental dyslexias” (Wolf & Bowers, 1999). This assumed heterogeneity is one of the main reasons why a single, reliable behavioral “marker” of developmental dyslexia has not emerged. While there is evidence to suggest that significant difficulty in phonological processing (e.g., rhyming, Pig Latin) at a pre-
reading age is predictive of future reading disability in some children (Stanovich, 1988),
these findings do not sufficiently generalize to support one reliable predictor in the
overall population.

Since the advent of interest in phonological processing as the “core” deficit, other
linguistic frameworks for defining and helping to explain reading deficiency have also
emerged in the literature. Most notably, the “double-deficit” hypothesis of reading
disability (Bowers & Wolf, 1993) asserted that a combination of phonological and rapid-
naming dysfunction accounts for a substantial portion of developmental dyslexia, and
produces poorer reading profiles than would be expected given either deficit
independently. While underdeveloped phonological processing, and perhaps rapid-
naming abilities, may constitute the “core” of many reading disabilities, research suggests
that in order to understand the full range of reading disabled profiles, investigation should
be geared towards a more general conceptualization of temporal processing (Tallal et al.,
1993) including low-level visual processing (Lovegrove et al., 1992).

Consistent with the dominant linguistic conceptualizations of developmental
dyslexia, inquiry aimed at uncovering the neurobiological bases of reading disability has
focused upon regions of cerebral cortex associated with language. Researchers have
typically examined the posterior temporal region, documenting cellular abnormalities
(Galaburda, 1985) and morphological variation of the planum temporale (Hiemenz &
Hynd, 2000; Morgan & Hynd, 1998; Plante et al., 1991; Hynd et al., 1990; Semrud-
Clikeman, et al., 1991). While these studies help to explain the disruption of linguistic
processing in the cortex, there is an underlying assumption that visual information, in the
dyslexic brain just as in normals, maintains functional integrity en route from the retina,
through several lower and higher visual pathways, to the posterior temporal region of the language cortex.

Alternatively, documented cases of dysplasias (Galaburda, 1985) and reversed asymmetry (Hiemenz & Hynd, 2000) in the posterior-temporal region and perturbations in the sensory processing system need not be considered mutually exclusive of each other. As will later be discussed in more detail, the anatomical proximity of linguistic processing centers and temporal processing centers within the temporo-parietal region permits speculation regarding common sources of pathology. To determine whether this speculation will yield a valid model for understanding dyslexic brains, there is a need to reconcile neurological findings implicating disruption of the language cortex with behavioral (Lovegrove, 1993; Cornelissen et al., 1994, Eden et al., 1995; Demb., 1998), anatomical (Livingstone et al., 1991), and physiological (Eden et al., 1996; Demb., 1998) evidence for disruption of the magnocellular visual pathway.

Parallel Systems

Discrete lesions within the primate visual system have been documented to produce several distinct deficits (Damasio, 1980). This is consistent with histological research that describes early visual processing as comprised of coordinated networks of specialized cells and pathways from the retina (DeValois & DeValois, 1980) to the occipital cortex (Zeki et al., 1991). Within this model, investigation of visual processing in primates and humans has yielded neurobiological evidence for a sensory processing system that generally functions in a “parallel” manner (Hubel, 1988; Merigan et al., 1990). With advances in technology, cellular and physiological differences between the
pathways were established (Livingstone & Hubel, 1988). These systems are referred to as magnocellular (consisting of M cells), and parvocellular (consisting of P cells).

Though differences in M and P cells are most evident at the lateral geniculate nucleus (LGN), there are distinctions between the magnocellular and parvocellular pathways at lower-levels of visual processing (Livingstone & Hubel, 1988). Selective perception is initially evident at the retina, where photons are transduced into graded local potentials. Distinct retinal ganglion cells then innervate the two geniculate subdivisions: The magnocellular system is innervated by relatively large type A retinal ganglion cells, while the parvocellular layer is innervated by relatively small, type B retinal ganglion cells. Further investigation is needed to determine if these parallel pathways originate at an even lower level, such as the bipolar horizontal cells of the retina, although it is fairly certain that both systems receive basic sensory information from the same rods and cones (Livingstone & Hubel, 1988).

The segregated arrangement of cellular layers at the level of the LGN is tenable to microscopic investigation (see Figure 1). Thus, the LGN has become the preferred site for anatomical study of the magnocellular and parvocellular pathways. Research has been conducted primarily with macaque (rhesus monkeys) due to the striking similarity between the macaque brain and the human brain, evident when comparing thalamic nuclei (Maunsell, 1995; Sereno et al., 1995). Humans and some other primates, such as macaque, possess six cellular layers at the LGN, whereas the number of layers found in other mammalian brains varies.
Figure 1. This image from Rosenzweig et. al. (1998) is a cross section of the macaque lateral geniculate nucleus. It demonstrates the organization of four parvocellular layers dorsal to two magnocellular layers. Layers 1, 4, and 6 receive inputs from the contralateral eye.

Within this gross subdivision of small dorsal (P) cells and large ventral (M) cells at the level of the LGN are three topographic maps from each hemi-retina. Each hemi-retina is mapped twice to the parvocellular layer and once to the magnocellular layer. The six topographic maps are believed to be highly precise.

The major anatomical similarity between M and P cells are receptive fields that function through center-surround opponency (as illustrated in Figure 2). Both M and P cells are excited by illumination of a small retinal region and inhibited by illumination of
a larger surrounding region. The reverse of this has also been noted (Livingstone & Hubel, 1988). This antagonistic mechanism, shared by the M and P cells, underlies basic processing of subtle discontinuities in light patterns. Beyond this most basic receptive mechanism, physiological characteristics of the M and P systems are relatively distinct.

Comparison of M and P cells at the LGN reveal differences in the size and type of receptive fields (Livingstone & Hubel, 1988; Rosenzwieg, et al., 1999). Consistent with these anatomical findings, M cells differ from P cells in several primary perceptual domains. M cells are comparatively larger and respond at stimulus onset and offset. They are specialized for detection of motion, direction, speed, course stereopsis (depth perception) and pursuit. The magnocellular system is basically color-blind, since the inputs of all three cone types (blues, greens, and reds) are summed. As a result the magnocellular system possesses broad spectral sensitivity, and response to changes in illumination is either on or off, without regard to wavelength (Livingstone & Hubel, 1988). Based upon selectivity to movement and low spatial frequencies, researchers sometimes refer to the magnocellular system as the “where” or “transient” system.

**Figure 2.** Receptive fields for (left) typical color-opponent parvocellular geniculate neuron, excited over a small region by red light and inhibited over a larger region by green light and (right) a typical broadband magnocellular neuron, excited by all wavelengths in the center and inhibited by all wavelengths in its surround (Livingston & Hubel, 1988).
Conversely, P cells are smaller and respond throughout stimulus presentation. They are receptive to changes in color, shape, fine acuity, and fine stereopsis (Tychsen, 1994). High contrast is necessary to stimulate a P cell, resulting in relatively poor motion detection. In addition, most P ganglion cells demonstrate differential responses to varied wavelengths, providing the basis for color vision. Based upon selectivity to stationary form and high spatial frequencies, the parvocellular system is sometimes referred to as the “what” or “sustained” system.

Schiller and colleagues (1990) demonstrated the specificity of M and P cells by selectively lesioning the brains of macaque at the LGN. When M cell layers were ablated without damage to the P layer, animals exhibited deficits in flicker and motion perception. P cell layers were also lesioned, without comprising the M cell layers, resulting in dramatic loss of color, texture, and pattern discrimination. While characteristics of M and P cells are generally distinct, there are minor exceptions (e.g., approximately 10% of P cells that possess broad-band spectral sensitivity) (Livingstone & Hubel, 1988).

Although the magnocellular and parvocellular pathways function in a roughly “parallel” manner, anatomical investigation has demonstrated that these pathways do not remain dissociated to the extent observed at the level of the LGN (Livingstone & Hubel, 1988). Rather, the magnocellular and parvocellular systems progressively intermingle en route from the LGN to the calcarine sulcus and surrounding occipital cortex (Maunsell et al., 1990; Merigan & Maunsell, 1993).
Figure 3. A diagrammatic representation of the magnocellular and parvocellular systems from the LGN to the specialized areas of the striate and extrastriate visual cortex. (Zeki & Shipp, 1993). Note both magnocellular and parvocellular innervation of area V1, while projections to area V5 are predominated by the magnocellular pathway.

Through inter-mixing of the magnocellular and parvocellular pathways, these “parallel” systems achieve a degree of functional inter-dependence (Lovegrove, 1993). Accordingly, the transient (magnocellular) and sustained (parvocellular) systems are both capable of inhibiting each other during complex perceptual processes, depending on the physical and temporal properties of visual information, and the integrity of the visual system. This concept of pathway inter-dependence and inhibition takes on a meaningful role in the discussion of developmental dyslexia. As will be elaborated when the visual mechanisms underpinning reading are discussed, dysregulation of these inter-dependent systems has been associated with specific perceptual difficulties and developmental dyslexia (Breitmeyer, 1980).
Dorsal and Ventral Streams

Overall, the magnocellular and parvocellular pathways provide visual information to over 30 specialized areas of striate and extrastriate cortex, either by direct means or by way of interconnections (Rosenzweig, et. al., 1999). The large number of specialized visual areas are clearly inter-related, as indicated by inter-connecting fibers; however, the organization of higher-level visual processing was largely unknown until Mishkin and Ungerleider (1982) proposed two primary cortical streams.

Based upon work with primates, it was posited that all visual information initially innervates the primary visual cortex (V1). From this common area emerges a ventral processing stream, responsible for visual identification of objects, and a dorsal stream, specialized for processing spatial properties and guiding movement towards objects. The results of PET studies and lesion analysis support the division of an analogous dorsal and ventral stream in humans (Ungerleider, 1998).

Figure 4: Broad distribution of the ventral and dorsal streams of higher-level visual processing (Ungerleider, 1998).
Livingstone and Hubel (1988) initially suggested that the ventral processing stream and the dorsal processing stream are extensions of the parvocellular and magnocellular pathways, respectively, based upon the similarities in perceptual specialization. They reported that magnocellular and parvocellular fibers were partly segregated in areas V1, and believed the pathways became completely segregated beyond area V2. From V2, it was thought that the original magnocellular system innervates the parietal regions, and the parvocellular system innervates the inferotemporal regions. For a time, the magnocellular and parvocellular systems were described as early components of a more global parallel processing system, appearing to project almost seamlessly from the dorsal and ventral stream pathways described by Mishkin & Ungerleider (1982).

More recently, however, several researchers have reported findings contrary to the notion of parallel visual processing at the cortical level. As a result, Mishkin and Ungerleider’s (1982) theory, at one time supported by Livingstone & Hubel (1988), is now generally considered over-simplistic. Researchers critical of the original position, typically note the considerable overlap of magnocellular and parvocellular fibers beginning in area 4b of V1 (Maunsell, et al., 1990, Ferrara, et al., 1992). A comprehensive review by Merigan & Maunsell (1993) concluded that the theory of magnocellular and parvocellular extension into the higher regions, although appealing in its simplicity, should be abandoned, based upon subsequent investigations.

While there is ample evidence to undermine the theory of separate cortical magnocellular and parvocellular pathways (Rosenzweig et al., 1999), researchers who argue against a direct extension of magnocellular and parvocellular systems emphasize a component of the theory that remains valid. Despite the intermixing of magnocellular and
parvocellular inputs to the cortical streams, it remains true that the magnocellular system supplies the *predominant* anatomical contribution to the dorsal stream (Maunsell, et al., 1990, Ferrara, et al., 1992; Merigan & Maunsell, 1993).

This is a critical point in light of current fMRI technology. Armed with the capability to monitor event-related changes in cellular metabolism within cortical regions known to be predominated by magnocellular fibers, investigators can reasonably infer the integrity of magnocellular function. This has been demonstrated by imaging the activation of extrastriate area V5 when subjects are exposed to visual stimuli designed to elicit selective activation of the magnocellular pathway (Eden, et al., 1996; Demb et al., 1998).

**V5 as an Indicator of Magnocellular Dysfunction**

Area V5 has been defined anatomically as an extrastriate area that predominantly receives magnocellular fibers and projects to the dorsal stream of the macaque brain (Newsome et al., 1985). Later, positron-emission tomography (PET) helped to establish a homologous area in the human brain that was given the same name (although it is also commonly referred to as the “medial temporal” region or “MT”). Duffy and Wurtz (1991) later confirmed results of the PET study by selectively lesioning area V5 in macaque to demonstrate specialized function in that region. It was reported that correct detection of object speed was consistently compromised by lesions to V5, while eye movements to stationary targets were unaffected. This lesion study was consistent with other reports claiming that V5 is selective for motion of visual stimulus and necessary for the analysis of motion (Kandel, 1991; Maunsell & Newsome, 1977).
Maunsell and colleagues (1990) documented similar findings as well. They reported that visual responses in the middle temporal region (V5) and the medial superior temporal (MST) region were always diminished, and typically eliminated, when the magnocellular pathway was disrupted at the LGN, but rarely affected when parvocellular activity was terminated. Collectively, results of these studies confirm that V5 is a motion selective extrastriate area, innervated predominantly by magnocellular projections. As will be discussed in greater detail, initial fMRI investigations suggest that dyslexics display a characteristic under-activation of area V5 when compared to normal readers (Eden, et al., 1996; Demb, et al., 1998). There are several theories suggesting how diminished magnocellular function, as observed in corollary area V5, may disrupt the reading process.

**Magnocellular and Parvocellular Mechanisms in Reading**

Before discussing the possible effects of magnocellular dysfunction on reading mechanisms, a brief review of basic eye movements may be helpful. Specifically, there are two types of eye movements that appear seminal to the reading process; saccades and fixation (Eden, et al., 1994). Saccades are rapid eye movements that permit version of the eyes from one fixation point to the next. It is believed that saccade movement may reach a velocity of 600° per second, and as a result, approximately 170 saccades may occur during one minute of reading (Hallett, 1986; Stark, Giveen, & Terdiman, 1990). Typically, saccades are abrupt, jerky movements, generally in the direction of reading across the page. The duration of a saccade can be as brief as 0.05 second (Hallett, 1986), during which 6-8 characters or approximately 2° of visual angle are transversed (Rayner
& McConkie, 1976). Essentially, each saccade determines where the reader will next fixate in the line of text.

Fixation movements are executed when the eyes are directed to the intended target. Optimal fixation occurs when the target projects directly to the fovea, the retinal area possessing the greatest density of photoreceptors (Matlin, 1983). Fixation occurs in three forms, referred to as microsaccades, microtremors, and drift. Each of these fixation types functions as a corrective mechanism that prevents images from becoming diffuse and undifferentiated. Visual information is extracted from the text during the fixation period, which is noted to last for approximately 200 to 250 msec. (Lovegrove, 1993). When these very brief periods are summed, fixation may account for over 90% of total reading time (Eden et al., 1994). A reader’s “perceptual span” is the area from which visual information can be extracted during fixation. It is reported to extend 5 (Lovegrove, 1993) to 20 (Morris & Rayner, 1990) letters to the right of central fixation.

In order to read effectively, Breitmeyer (1980) posited that the image formed during fixation must be mediated by the sustained (parvocellular) visual system. (see Figure 5). This model suggests that the transient (magnocellular) system regularly inhibits the sustained system, as has been shown in physiological investigation (Singer & Bedworth, 1973), to prevent superimposition of consecutive images. Lovegrove (1993) suggested that the ability of the magnocellular system to inhibit sustained visual processing in normal reading is analogous to the general ability of peripheral vision to over-ride centralized, sustained vision.
Figure 5. Transient and sustained interactions are hypothesized to interact during fixations. These traces schematically represent three successive eye movements, characterized by saccades and fixation pauses. It is posited that the sustained response (line 2) is inhibited by the transient response (third line) during normal reading, resulting in the ability to scan a line of words without the occurrence of visual persistence (Breitmeyer, 1980; & Evans, 1994).

![Figure 5](image)

Figure 6. If sustained activity is not inhibited, and persistence occurs during reading, several fixations may be needed to process what could otherwise be extracted from a single perceptual scan (Lovegrove, 1993, adapted from Hochberg, 1978).

There is little direct empirical evidence for, or against, Breitmeyer’s (1980) explanation of coordinated visual processes during reading (Lovegrove, 1993). However, measurement of eye movements between dyslexic and normal groups suggests that dyslexics may demonstrate persistence and masking effects related to magnocellular system deficit.
Eden and colleagues (1994) reported that their sample of children with reading disabilities displayed significantly worse eye movement stability during fixation towards small targets. In addition, a qualitative assessment of saccadic eye movements revealed that this group exhibited significant fixation instability at the end of saccades. In spite of these findings, Eden and colleagues (1994) acknowledged that differences in eye movement between dyslexic groups and controls were probably better accounted for by linguistic difficulties. This sentiment is consistent with an earlier finding by Pirozollo and Rayner (1978), who demonstrated that group differences in eye movement were minimized when dyslexics were provided with text at their appropriate reading level.

As an alternative to directly assessing eye movements, psychophysical research has provided evidence that visual persistence is related to reading ability. Riding and Pugh (1977) examined 47 randomly selected 9 year-old children without diagnosable reading delay, using the Neale Analysis of Reading Ability. In general, children who exhibited a duration of visual persistence within the average range demonstrated reading ability in advance of those with visual persistence measured outside the normal range (Riding & Pugh, 1977). Similarly, Macknik and colleagues (1991) reported that irregular inhibition of saccades was associated with perceptual errors characteristic of magnocellular deficit. While psychophysical evidence suggests that the magnocellular system mediates reading fluency, more eye-movement studies are needed to define the specific mechanisms.

Psychophysical Investigation of the Magnocellular Deficit Hypothesis

To date, there have been several psychophysical comparisons of low-level visual performance between dyslexic and normal readers. Based upon the receptive properties
of the magnocellular and parvocellular systems, experimental tasks have generally focused on visual thresholds for stimuli presented at low spatial frequency with low-contrast and high temporal frequency. Collectively, these studies provide support for the magnocellular deficit hypothesis, although some inconsistent results necessitate further investigation. Disparate findings are likely related to variance in subject selection and stimulus design.

Felmingham and Jakobson (1995) compared the performance of nine reading disabled children and nine normal reading children, matched for age and overall intellectual ability, on a measure of flicker sensitivity. When thresholds were compared, the dyslexic group was found to demonstrate significantly less sensitivity. Further, the group differences found by Felmingham and Jakobson (1995) were noted to increase when temporal frequencies were raised. This was interpreted as evidence for a magnocellular deficit related to dyslexia, given that M cells are specialized for speed detection under low-contrast conditions (Livingstone & Hubel, 1988). In addition, this result supported the findings of several other psychophysical studies (Cornelissen et al., 1993; Slaghuis, et al., 1993; Martin & Lovegrove, 1987, 1988) that reported deficient flicker/motion detection abilities in dyslexic groups.

An investigation that highlights the inconclusive nature of the literature in this area was conducted by Brannon and Williams (1988). They presented a uniform flicker field surrounded by an area of equivalent luminance to “poor” readers and normal readers. Results indicated that the poor readers demonstrated reduced sensitivity to stimuli across several grades of temporal frequency (e.g., 4, 8, 12, 16, 20 and 24 Hz).
More specifically, the poor readers displayed the most atypically elevated thresholds for low to medium temporal frequencies (e.g., 4, 8, and 12 Hz).

While this finding supports the association between visual processing deficits and reading disability, the results are more consistent with a compromised parvocellular pathway than with a compromised magnocellular pathway. Skottum (2000) reviewed this study and provided a caution that is relevant to all psychophysical studies in this field. He asserted that the visual stimuli used by Brannon and Williams (1988) may have unintentionally produced a “sharp edge” between the flickering field and the surround during each flicker cycle. This may have elicited more parvocellular activation than intended across the range of temporal frequencies.

Livingstone and colleagues (1991) used visual evoked potentials (VEP’s) to determine whether anatomical abnormalities reported in the magnocellular layer of the lateral geniculate nucleus might be related to functional deficits in visual processing. It was reported that dyslexics produced diminished VEP’s for low-contrast, pattern-reversal stimuli presented at high rates of reversal, consistent with the magnocellular deficit hypothesis. This study was soon replicated by Johannes and colleagues (1996), who found similar results using transient and steady-state VEP’s to assess physiological activation of the magnocellular system. Both of these investigations assessed groups of adult dyslexics.

**Group Inclusion Criteria**

Unfortunately, one of the most consistent characteristics of group selection in the literature is the use of an I.Q. – reading achievement discrepancy to differentiate dyslexic
and normal reading groups. The lack of diagnostic validity for this schema was outlined in the introductory section.

Inconsistencies in group selection criteria have also been identified as a problem (Borsting et al., 1996). With regard to specific instruments, most researchers have used a measure from the Wechsler series (e.g., WISC) to establish an estimate of intelligence. While this instrument facilitates valid measurement of I.Q., and permits comparison of results between studies, the subjects labeled “dyslexic” almost invariably produce I.Q. scores significantly below those of the controls. Most often this gap between group I.Q.’s occurs within the two standard deviation range considered average and is rarely discussed as a confound.

Given the expected covariation of reading ability and overall intellectual ability (Woodcock, R. W., 1989), it is difficult to match reading disabled and normal subjects for intellectual ability if the author wishes to include individuals with severe reading impairment. The problem of comparing groups with discrepant levels of intellectual ability is very significant from a neurobiological perspective, as there is no systematic means to account for the myriad differences in anatomy and/or physiology that may be related to overall intellectual ability. As a result, assertions regarding the neurobiology of the reading disability itself can not be stated with confidence.

As discussed in the preceding section, achievement instruments are rarely consistent across studies. Group comparisons of reading disabled subjects and normal reading controls in this field have employed many instruments to attain a normed estimate of reading ability including the Wide Range Achievement Test – 3rd Edition (WRAT-3) (Demb, et al., 1998), Gray Oral Reading Test – 3rd Edition (GORT-3) (Eden
et al., 1996), Neale Analysis of Reading Ability (Martin & Lovegrove, 1984) and British Ability Scales (Cornelissen, et al., 1998). The results of these studies are often compared although there is no consensus regarding which instrument, or which combination of instruments, should be used as best practice to discern when an individual exhibits reading disability that is meaningfully disparate from normal reading.

With the issue of test selection unresolved, Borsting (1996) sought to address the heterogeneity of dyslexic groups that was evident between studies. He noted that groups labeled “dyslexic” often possess disparate neurolinguistic profiles, which is a predictable result of the vague working definitions of developmental dyslexia that were discussed at the outset of this article. Despite the common labeling of dyslexic groups in the literature, the heterogeneity issue presents a problem to any person who would wish to meaningfully synthesize, or meta-analyze, the collective body of published results.

Borsting (1996) noted that approximately 75% of subjects labeled solely as “dyslexic” in the visual processing literature had historically demonstrated magnocellular processing deficits if administered relevant psychophysical measures. While this percentage is compelling in itself, he sought to investigate the reasons why 25% of reading disabled subjects produced non-significant results. Intending to describe a more precise relationship between reading disability and early visual processing than was possible using a generic “dyslexic” group, Borsting (1996) classified his reading disabled subjects as demonstrating the characteristic deficits of either of two sub-types. He compared 8 “dysphoneidetic” dyslexics (primary deficit believed to involve phonological processing) with 9 “dyseidetic” dyslexics (primary deficit believed to involve orthographic processing) and nine normal readers. Dysphoneidetic dyslexics demonstrated reduced
sensitivity to low spatial frequencies at 10 Hz, while dyseidetic dyslexics demonstrated reduced sensitivity below 10 Hz, and the control group displayed normal sensitivity at both luminance conditions. This was interpreted to suggest that phonological deficits are most closely associated with an inefficient magnocellular pathway.

While the relatively small sample sizes described above limit confidence in Borsting’s (1996) positive finding, there has been little effort to confirm or challenge the utility of sub-typing. Results suggesting that there is no significant difference in flicker sensitivity between adult dyslexics and normal readers (Gross-Glenn et al., 1995) have not compared dyseidetic versus dysphoneidetic subtypes prior to publication. Further, reading skills are generally not assessed comprehensively enough to permit retrospective classification of sub-types.

Other factors that have hindered meaningful comparisons between studies are the disparity in ages found between dyslexic groups and the use of relatively small samples. While the study of magnocellular pathway involvement in dyslexia across the life-span may eventually bring relevant developmental factors to light, research designs to date have generally assessed either child or adult subjects exclusively. Examples of this are evident when some of the most methodologically sound, and important, studies in this field are examined. Consider that two of the seminal psychophysical studies previously mentioned include a mean subject age of approximately 10 years (Cornelissen et al., 1993; Slaghuis, et al., 1993). In comparison, only adults have participated in the functional neuroimaging studies of dyslexia and early visual processing that will be discussed (Eden et al., 1996; Demb et al., 1998). If the former is truly providing the theoretical foundation for the latter, this inconsistency must be addressed.
Sample sizes must also increase significantly if the validity of the magnocellular-deficit hypotheses is to be tested more conclusively. Reported findings from groups containing as few as 6 dyslexic subjects (Eden et al., 1996) are currently afforded significant importance within the current body of knowledge, particularly when neuroimaging is involved. It is hoped that greater access to advanced technology, such as fMRI, will facilitate larger experimental groups in the future. Should practical limitations continue to limit the number of participants in most studies, investigators must strive for greater uniformity in stimulus design in order to produce a theoretical consensus based upon findings from several research laboratories. Finally, it is apparent that investigators have not taken advantage of a specific advantage presented by relatively small groups. There has been no published account of follow-up evaluation subsequent to initial psychophysical or neuroimaging studies. Without longitudinal investigation, the stability of behavioral and physiological findings for dyslexic groups and controls will remain speculative.

**Stimulus Properties**

Since the stimulus properties used to activate the magnocellular system in the literature are typically quite similar (e.g., low contrast, low mean luminance, low spatial frequency, high temporal frequency), but presented within a visual format unique to each particular experiment, nuances in stimulus design may impact whether comparisons between findings are meaningful. Due to the highly specific receptive fields of M and P cells, a single unintended stimulus property may influence activation of the cell type that is believed to be suppressed. If an experiment intends to elicit predominant activation
from just one of the early visual pathways, this occurrence presents a significant confound.

The following study exemplifies the extent to which modification of one stimulus property can effect psychophysical results. Cornelissen and colleagues (1995) reported no group differences between dyslexic children and age-matched controls when assessed for luminance contrast thresholds at photopic levels. However, when comparisons of motion detection were made between the same groups at scotopic levels, the dyslexic group was found to be significantly less sensitive to motion. This finding is consistent with other studies (Mason et al., 1993; Martin & Lovegrove, 1987) that have suggested dyslexics can be reliably differentiated from normals by measures of contrast sensitivity at low luminance levels and not at high luminance levels (Martin & Lovegrove, 1994).

To date, a consensus has not been reached in the literature concerning which type of psychophysical stimuli is optimal to elicit magnocellular activation with less than baseline parvocellular contribution. Unfortunately, this problem has already surfaced in the fMRI literature as well. The initial fMRI study in the field employed coherent patterns of dots (Eden et al., 1996), whereas the second fMRI study collected imaging data using sinusodial gratings (Demb et. al., 1998). While lack of stimulus uniformity has significantly hindered meaningful synthesis of the psychophysical literature, there is still time for the emerging fMRI literature to support a consistent visual stimulus design. If future researchers assume a more collaborative role, fMRI investigations can produce a more cohesive body of literature related to early visual processing and developmental dyslexia than presently exists.
In addition to stimulus inconsistency in the psychophysical literature, there have not been published attempts to introduce a control measure. Normal readers are often matched across general demographic and cognitive variables, such as age and I. Q., and typically serve as control groups to reading disabled subjects. This only serves to minimize error variance contributed by subject characteristics.

The predominance of psychophysical measures designed to elicit magnocellular functioning and suppress parvocellular functioning in linguistic studies have been designed with low mean luminance, minimal contrast, and motion. These properties are consistent with findings from the neuroscience literature. A control measure in which one of these magnocellular-eliciting properties is meaningfully altered is needed, as this comparison could help to determine which visual stimuli designed to elicit predominant magnocellular activation is most effective.

The psychophysical measure in this experiment that presents a diffuse, red background may advance current theory, as there have been few attempts to suppress magnocellular suppression in the dyslexia literature. The idea follows Brown and Koch’s (2000) recent use of a diffuse, red background in the basic visual science literature. They cited physiological (Wiesel & Hubel 1966; Dreher, Fukuda & Rodieck, 1976) and psychophysical (Breitmeyer & Williams, 1990; Edwards, Hogben, Clark & Pratt, 1996) evidence that a diffuse red background suppresses magnocellular pathway activity and explained this is due to the M pathway’s broad response to wavelength. They hypothesized that M cells would experience tonic suppression, since the inhibitory surrounds are responsive to long-wavelength red light, and found that diffuse red light was “surprisingly capable” of influencing magnocellular functioning (Brown & Koch,
This being the case, reading disabled subjects with a magnocellular deficit who are administered a magnocellular activating task, and the same task with a diffuse red background, should perform more like controls on the latter.

**Functional Magnetic Resonance Imaging (fMRI)**

In addition to the psychophysical studies previously noted, two of the more recent investigations of early visual processing and developmental dyslexia also monitored hemodynamic response to visual stimuli in the striate and extrastriate cortex. Ideally, functional neuroimaging would provide definitive insight into the question of magnocellular deficit, by directly detecting hemodynamic changes in the early visual pathways. Unfortunately, this direct measurement would require an advance in imaging sensitivity in order to isolate the physiological activation of the pathways as they interweave through the thalamus and continue their ventral projection. Since current neuroimaging technology does not permit analysis of rapidly changing blood oxygenation at the level of the magnocellular pathway, investigators have focused on corollary areas of cerebral cortex known to receive the predominance of magnocellular projections. These regions of interest are located in the primary visual cortex and extrastriate cortex, areas conducive to study with fMRI. Tables 1 and 2 summarize fMRI findings.

Eden and colleagues (1996) used fMRI and a related psychophysical measure to investigate the pathophysiology of dyslexia at the level of early visual processing. They assessed 6 adult male dyslexics and 8 controls matched for gender, age, socio-economic status, educational background and overall intelligence. In order to ensure that all reading deficits were of primary developmental origin, neurological abnormalities, including subtle neurological signs and attention-related diagnoses, served as bases for exclusion.
The groups in this study were differentiated based upon four criteria: The dyslexic subjects demonstrated a documented history of reading disability, an absolute reading deficit (GORT-3 Passage standard score <90), a discrepancy of at least 2 standard deviations between reading ability (GORT-3 Passage) and verbal I.Q. (WISC-R) and significantly poorer phonological awareness than controls when assessed with a pseudo-word reading task. This study found comparable neurophysiological activation for both groups in the area of the primary visual cortex (VI/V2) and several extrastriate visual areas. However, all 6 dyslexic subjects failed to show normal activation in area V5. Figure 7 illustrates the activation difference between groups.

Figure 7. This graphic illustrates group differences in activation between the dyslexic group and the control group, as revealed by fMRI (Eden et al., 1996). Note the arrows indicating that activation of area V5 is limited to the control group in response to moving stimuli.
Table 1. Summary of subject characteristics in functional magnetic resonance imaging (fMRI) studies of visual processing in dyslexia.

<table>
<thead>
<tr>
<th>Study</th>
<th>Diagnostic criteria</th>
<th>Exclusion of subjects</th>
<th>Selection of control</th>
<th>N (M/F)</th>
<th>Age (years)</th>
<th>IQ</th>
<th>Reading</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eden et al</td>
<td>Documented childhood history of reading disability</td>
<td>No ADHD or neurological diagnoses</td>
<td>Matched for gender, age, education, SES, &amp; IQ</td>
<td>(a) 4.7</td>
<td>(b) 14.3</td>
<td>(c) 40.8</td>
<td>(d) 17.2</td>
</tr>
<tr>
<td>1996</td>
<td>≥ average FSIQ on the WISC-III</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≤ 8th %tile on the GORT-3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥ 20 point discrepancy between GORT-3 Passage and</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diagnosis of dyslexia as an Adult from a university disabilities resource center.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Demb et al.</td>
<td>Documented childhood history of reading disability</td>
<td>No neurological or psychiatric illnesses, 3 dyslexics also</td>
<td>Matched for gender, age &amp; education</td>
<td>(a) 57.4</td>
<td>(b) 50.0</td>
<td>(c) 40.6</td>
<td>(d) 17.2</td>
</tr>
<tr>
<td>1998</td>
<td>Diagnosis of dyslexia as an Adult from a university disabilities resource center.</td>
<td>were diagnosed with ADHD but were free of medication at the</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>time of the study</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- the author reasons that all subjects were Stanford University students and thus assumed to be of above average intelligence.
Table 2. Summary of results of functional magnetic resonance imaging (fMRI) studies of visual processing in dyslexia.

<table>
<thead>
<tr>
<th>Study</th>
<th>Neuroimaging Technique</th>
<th>Visual Conditions</th>
<th>Results</th>
<th>Conclusions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eden et al., 1996</td>
<td>Multislice EPI data of local blood oxygenation level dependent (BOLD) contrast signals using a 1.5 Tesla system. Contiguous coronal (5 mm) slices (30) were collected, of 5 mm cubic voxels that spanned occipital and posterior parietal cortex. Ninety scans were obtained for each subject.</td>
<td>During the scans, subjects maintained fixation while viewing: 1) a fixation cross with uniform illumination 2) a low contrast (Michelson 5%) array of black dots on a grey background moving at 100% coherence at 10 degs/sec. 3) a high contrast (40%) stationary, patterned dot field.</td>
<td>For all dyslexic subjects, presentation of moving stimuli at low contrast failed to produce the same task-related activation of areas V5/MT, regions believed to be predominated by the magnocellular pathway. Presentation of stationary, high contrast stimuli produced equivalent activations in V1/V2 and extrastriate cortex in both groups.</td>
<td>The absence of motion sensitivity in areas V5/MT in dyslexics may result in a disruption of normal coordinated temporal interaction with other motion processing areas. Results may also be related to input from non-geniculate fibers. Motion detection deficits for the dyslexic group were subtle and not comparable to lesion related deficits.</td>
</tr>
<tr>
<td>Demb et al., 1998</td>
<td>Local blood oxygenation Dependent (BOLD) contrast signals using a 1.5 Tesla system. Configural (4 mm) slices were collected. Data was collected in planes wither perpendicular to the calcarine sulcus or parallel to the calcarine sulcus with the lowest slice near the ventral surface of the occipital pole.</td>
<td>During the scans, subjects maintained fixation while viewing: 1) 0.4 cycle/deg/sec sinusoidal gratings that moved 20.8 deg/sec with low mean luminance (2cd/m²) Contrast was varied (3, 6, 25, 50 &amp; 100%). Orientation and direction of motion changed every 500 ms to minimize between adaptation.</td>
<td>Dyslexics showed less brain activity both in the primary visual cortex (V1) and in several extrastriate areas including areas MT, MT+, V3, V3A, &amp; V4v. Individual performance revealed a significant correlation between lower brain activity in areas V1 and MT+ and poorer motion perception ability. Differences in brain activity groups accounted for 64% of the variance on the measure of reading speed.</td>
<td>This is the first study to present evidence for activation differences in area V1. The results support reading speed as the most sensitive marker of dyslexia. A strong 3-way correlation was found between V1 and MT+ brain activity, sped discrimination thresholds, and reading speed.</td>
</tr>
</tbody>
</table>
2) Control stimuli were 0.4 cycle°
flickering sinusoidal gratings
(contrast reversing at 8.3 Hz) at
higher mean luminance (36 cd/m²)
Contrast was also varied at the same
Increments at the experimental
condition. Orientation remained
constant throughout the scan.
Demb and colleagues (1998) followed this initial study (as described in Tables 1 & 2). Their results supported the assertion that differences in V5 activation are evident between dyslexic and control groups. In addition, they reported that the dyslexic group demonstrated relative under-activation in striate area V1 and extrastriate areas V2, V3, V3A, and V4v. Since these initial fMRI investigations both produced evidence of qualitative physiological differences during early visual processing between developmental dyslexics and normal readers (inferred through hemodynamic activation at higher levels), and all subjects were deemed free of postnatal neurological insult, there is reason to speculate on the developmental nature of early visual processing deficiencies.

Figure 8. Location of area MT+. A) Parasagittal anatomical image from one subject indicating the slice selection (parallel to the calcarine sulcus) for the moving dots and the test conditions. The blue lines were slices that contained the MT+ region of interest in this subject. B-D) Brain activity in one slice containing the MT+ region of interest from three control subjects. Reddish voxels show regions with greater response to moving versus stationary dot patterns. Images were chosen to optimally show MT+ in the right hemisphere (arrows), although activity from the left hemisphere MT+ and V1 are also present in some cases. The image in B is from the same brain as the sagittal image in A and corresponds to the most inferior of the three blue slices. The MT+ region of interest as defined by outlining (dotted line in B) the strongest area of activity that was approximately lateral to the junction between the calcarine sulcus and the parieto-occipital sulcus, and beyond the retinotopically organized visual areas. E-G) Slices with MT+ region of interest in three dyslexic subjects (Demb, et al., 1998).
Developmental Considerations

Until further scrutiny is undertaken to confirm the existence of the magnocellular-deficit hypothesis, developmental considerations are driven by relevant findings in basic neuroscience and neuropsychology. Accordingly, three general theories of etiology appear most likely. The first asserts that the magnocellular pathway may be compromised by a hereditary or congenital factor. Conversely, the second theory focuses upon sensory deprivation (e.g., lack of exposure to print) as the primary factor of magnocellular disruption. The third theory proposes an interaction effect between organic and environmental factors as the best explanation for the deficit.

There is sound empirical evidence suggesting that primarily heritable or congenital factors underly magnocellular deficits. This notion is afforded credence through its consistency with the more expansive theory of minimal brain damage. Livingstone and colleagues (1991) reported the occurrence of abnormal M cells during autopsy of developmental dyslexics. Aberrant cellular induction (e.g., trophic influences) during ontogenesis and dysregulation of auto-immunological response have been posited to explain both the presence of abnormally large somas within layer 1 of the language cortex (Galaburda et al., 1985) and unusually small somas within the magnocellular layer of the LGN (Livingstone, et al., 1991) in some dyslexic brains.

With respect to behavior, it has been suggested that the co-occurrence of reading problems and “soft” neurological signs may indicate a common neuro-developmental perturbation (Denkla, 1998; Eden, et al., 1994). Considering that dysplasias found in dyslexic brains were noted in diffuse cortical locations, while in greatest density in
linguistic centers (Galaburda, 1985), there is reason to suspect that a common heritable or congenital factor may compromise proximal linguistic and visual processing centers.

Basic neuroscience also supports the possible role of environmental factors on the development of magnocellular functioning. Wiesel & Hubel (1965) provided the most direct evidence for neural plasticity within the lower-level visual pathways by manipulating the visual experience of kittens shortly after birth. The result was a significant impact upon the maturation of magnocellular and parvocellular projections to the striate and extrastriate cortex. With regard to human perceptual processing, experiences through adulthood have been shown to alter the somesthetic cortex (Kaas, 1995) and auditory receptive fields (Weinberger, 1998). Specific to the visual system in humans, Bennett and colleagues (1964) demonstrated neural plasticity, but only during a sensitive or “critical” period of development. A period of significant physiological and anatomical adaptation to the environment typically endures from birth until approximately 9 years of age (Vaegan & Taylor, 1980). This being the case, early childhood experiences could impart a significant and lasting effect upon development of the early visual system.

Finally, it is interesting to speculate that an interaction between heritable or congenital perturbation and subsequent cerebral adaptation may account for a broad temporal-processing deficit. Initial evidence suggests that plasticity can develop across sensory modalities in certain cases. Neville (1998) used neuroimaging to assess deaf subjects and reported that their cortical language areas and temporal areas associated with visual-motion processing both exhibited functional reorganization following loss of hearing. Further, this functional reorganization between language processing and visual
motion areas of the temporal cortex translated into superior performance on psychophysical tasks requiring magnocellular functioning when compared to subjects with normal hearing. Eden and Zefferino (in press) noted that the mechanism for reorganization between language and visual processing areas is unknown, but may someday provide an explanation for the association of temporal processing deficits (e.g., early visual processing, phonological processing) with reading disability.

**Conclusions**

It is encouraging that most proponents of the magnocellular-deficit hypothesis do not portray early visual dysfunction as the cause of dyslexia. Instead, the co-occurrence of magnocellular pathway deficit and linguistic difficulties is generally conceptualized as manifestation of a more global neurodevelopmental symptom-complex. This is consistent with the notion of minimal brain dysfunction (Demb, et al., 1998).

Psychophysical investigation has been useful in probing the relationship between early visual processing and reading disability, and has established a theoretical foundation for further investigations. Of the emerging directions for study in this field, functional imaging appears to hold the key for future discovery. Initial fMRI studies have utilized stimuli designed to elicit predominant magnocellular activation and reported that small samples of dyslexics displayed characteristic under-activation within an extrastriate visual region (V5) predominated by magnocellular inputs (Eden et al., 1996; Demb, et al., 1998). These findings are consistent with earlier psychophysical (May, et al., 1991; Lehmkuhle et al., 1993; Kubova et al., 1996) and physiological (Livingstone et al., 1991) evidence for a magnocellular deficit in dyslexia. In spite of these results, the literature has
not reached a theoretical consensus. Discrepant results may be the result of questionable group criteria and inconsistencies in stimulus design.

While the problems noted above have led to some uncertainty, these issues can largely be remedied through improvements in design and methodology. The following chapter describes how this study aims to clarify the relationship between reading ability and magnocellular pathway function by de-emphasizing group comparisons, accounting for a variety of cognitive and linguistic variables, and utilizing a complimentary psychophysical measure with a diffuse red background.
CHAPTER 3

METHODOLOGY

This chapter presents a discussion of the methods employed in this study. This includes description of subjects, procedures, and neuropsychological and psychophysical instruments. This chapter concludes with an overview of the methods of data analysis.

Subjects

The subjects for this study were recruited through school psychologists in north Georgia as part of the protocol for a grant sponsored by the National Institutes of Health. The ongoing research project is entitled “Brain Morphology and Neurolinguistic Ability in Dyslexia.” Flyers were mailed to school psychologists stating that the University of Georgia Center for Clinical and Developmental Neuropsychology (CCDN) was accepting referrals for a research program. The flyers offered a neuropsychological evaluation for children ages 8-12 with a history of reading problems. In addition to the referred child, reading disabled and normal reading siblings were also included in the study when possible.

Biological parents of the referred child, who were interested in obtaining a neuropsychological evaluation for their child, were interviewed over the telephone by a member of the CCDN staff. In order for a family to be accepted for the program, it was required that one or both biological parents agree to participate in the study. In order to
assess early visual processing within this grant paradigm, psychophysical measures were added to the full-day neuropsychological assessment protocol.

Since the psychophysical measures were additions to the original grant protocol, separate institutional review board (IRB) approval was applied for and received (see Appendix A; dated 12-18-2000). Informed consent was obtained from all subjects prior to the evaluation. In addition, an IRB-approved assent script was read to minors, which clearly conveyed the opportunity to decline participation (see Appendix B).

Children who participated in this study ranged in age from 8 years, 4 months of age to 16 years, 2 months of age (mean age = 10 years, 7 months). The child sample included 20 subjects, comprised of 14 males and 6 females. Adult participants in this study ranged from 29 to 49 years of age (mean age = 38 years). The adult sample included 28 subjects, including 15 females and 13 males. With respect to both children and adults, potential subjects with a history of neurological disorder (e.g., epilepsy, traumatic brain injury) were excluded during the screening process. All participants without normal visual acuity wore corrective lenses during testing.

Procedure

The two psychophysical measures administered to assess early visual processing are referred to as “Gray Temporal Dots” and “Red Temporal Dots.” In order to assess overall cognitive functioning, neurolinguistic abilities, and reading competency neuropsychological tests were individually administered in the university clinic. The test battery included the Wechsler Abbreviated Scale of Intelligence (WASI; Psychological Corporation, 1999); the Woodcock Reading Mastery Test- Revised (WRMT-R; Riverside, 1987) Word Identification and Word Attack subtests; the Gray Oral Reading
Test- 3rd Edition (GORT-3; Pro-Ed, 1992), the Woodcock-Johnson Tests of Cognitive Abilities- Revised (WJC-R) Memory for Words and Memory for Sentences subtests, and the Comprehensive Test of Phonological Processing (CTOPP; Pro-Ed, 1999) Phonological Awareness and Rapid Naming Composites.

**Gray Temporal Dots**

This measure was designed to replicate the psychophysical properties of the “Temporal Dots” measure created by Eden and colleagues (1995) to assess magnocellular processing. Discrete stimuli were presented at intermittent intervals, at high temporal frequency, and with mesopic illumination. Performance on this instrument was measured as the percent of responses that were correct divided by the total number of trials (e.g., correct responses/72 trials). This measure was administered using E-Prime software on a Gateway computer powered by a Pentium III microprocessor.

Since reading disabled subjects are believed to experience deficits in contrast sensitivity within the mesopic range, figure and ground stimuli for each of the four measures were calibrated within a range of 10.5 to 15.5 candelas per meter square (cd/m²). Similar to the original task (Eden et al., 1995), small squares appeared intermittently at very brief intervals. The squares measured approximately .16 cm² and appeared as “dots” when viewed from a chin-rest (positioned at a standardized distance of 30.0 centimeters). At that distance the “dots” subsumed .30 degrees of visual angle and flashed in succession within a centrally located, white, 2.0 cm² fixation box. The fixation box remained on the screen continually against the gray background.

Each flashing dot appeared for a random interval between 200 and 400 msec. Eden and colleagues (1995) reasoned that randomized intervals reduce the subject’s
opportunity to apply rhythmic counting strategies to facilitate an accurate estimate. The time interval was also considered to be of adequate duration to prevent backward masking (Eden et al., 1995).

Between 3 and 8 dots were randomly presented in each trial and there were 12 trials at each dot number, creating 72 trials in all. Unlike the original study, which required the subject to respond by pressing a number on the monitor, this adaptation required the subject to state their estimate verbally. The investigator then entered the response on the keypad. This modification was made to minimize subject movement from the chin-rest and to prevent inadvertent responses due to poor manual coordination. Further, the possible effect of sustained attention upon performance was minimized by requiring subjects to initiate each dot presentation by pressing the space bar.

**Red Temporal Dots**

Based upon findings that diffuse red stimuli suppresses magnocellular pathway activity at low mean luminance (Williams, Breitmeyer, Lovegrove & Gutierrez, 1991), this measure was designed as a compliment to the “Gray Temporal Dots” measure. Specifically, this control measure assessed subjects’ ability to process the visual information described above when presented against a diffuse red background instead of a gray background. All stimulus properties were consistent with the “Gray Temporal Dots” measure described above except that the background was red. Again, a photometer was utilized to calibrate the figure (dots) and background to ensure low mean luminance.

**Wechsler Abbreviated Scale of Intelligence**

The WASI is normed for ages 6-0 to 89-11 years and consists of four subtests (Vocabulary, Similarities, Block Design & Matrix Reasoning), all of which were
administered during this study. At the subtest level, the reliability coefficients for children within the age range for this study are from .86 to .93 for Vocabulary, from .81 to .91 for Similarities, from .84 to .93 for Block Design, and from .86 to .96 for Matrix Reasoning. The reliability coefficients for the adult sample are .90 to .98 for Vocabulary, from .84 to .96 for Similarities, from .90 to .94 for Block Design, and from .88 to .96 for Matrix Reasoning. The correlation coefficient between the WASI FSIQ and the WAIS-III FSIQ is .92.

**Woodcock Reading Mastery Test- Revised (WRMT-R)**

The WRMT-R consists of several tasks that assess individual reading skills and is intended for use with subjects age 5-0 to 75+. The WRMT-R subtests utilized in analysis included Word Attack (visual decoding) and Letter and Word Recognition. Raw scores on these subtests have standard score equivalents. Split-half reliability coefficients of the Word Attack and Letter and Word Identification subtests are .87 and .97 respectively.

**Gray Oral Reading Test- 3rd Edition (GORT-3)**

The GORT-3 is an individually administered, standardized measure of reading in which subjects are timed as they read aloud. The test assesses accuracy, speed, comprehension and overall reading proficiency. The GORT-3 is normed for use with subjects ages 7-0 to 18-11. Passages are initially two to three sentences in length and comprised primarily of monosyllabic words. Progressively, passage length increases and irregular spellings become more frequent. In relation to the age criteria for this study, the test-retest reliability coefficients range from .86 to .95 for the Reading Rate subtest, from .79 to .95 for the Reading Accuracy subtest, and from .84 to .96 for the Passage Comprehension subtest. Concurrent validity scores for the GORT-3 with other reading
tests, such as the California Achievement Test (CAT) and Diagnostic Achievement Battery-2\textsuperscript{nd} Edition (DAB-2), are reported in the manual to yield a median .57 correlation.

**Woodcock Johnson Tests of Cognitive Abilities-Revised (WJC-R)**

The full WJC-R consists of 14 tests that measure various aspects of intellectual ability and is normed for use with subjects from 24 months of age to over 90 years of age. With regard to subtests used in this study, Memory for Words measures the ability to repeat lists of unrelated words presented from an audio tape and Memory for Sentences measures the ability to repeat progressively longer sentences presented from an audio tape. Both the Memory for Words and Memory for Sentences subtests load highest on the short-term memory factor of the WJC-R with coefficients of .66 and .85 respectively. The latter also loads on the auditory processing factor at .30. Test-retest reliability for the Memory for Words and Memory for Sentences subtests are presented through test-stability coefficients of .73 and .79 respectively.

**Comprehensive Test of Phonological Processing (CTOPP)**

The CTOPP is a test of phonological awareness, phonological memory, and rapid naming that is normed for use with individuals ranging from 7 through 24 years of age. Specifically, it is designed to identify a profile of phonological strengths and weaknesses that facilitates skill enhancement strategies (Wagner, Torgeson & Rashotte, 1999). Subtest performance on the CTOPP can be converted into standard scores, providing for comparison with other measures. Composite scores were utilized that provide an index of Rapid Naming (Rapid Digit Naming and Rapid Letter Naming) and Phonological Awareness (Blending Nonwords and Segmenting Nonwords). Test-retest reliability
coefficients, as assessed with an 8-17 year old sample are .79 in the Rapid Naming Composite, and .88 in the Phonological Awareness Composite.

Analyses

For the child and adult samples, simple and multiple regression analyses were used to determine the extent to which performance on the psychophysical measure designed to elicit magnocellular activation accounts for variance in reading ability. In addition, developmental and cognitive variables were used to test alternate regression models.

Performance on the “Gray Temporal Dots” measure and performance on the corresponding control measure, “Red Temporal Dots,” were analyzed in relation to reading ability to investigate divergent validity of the visual measures. Regression was utilized with the child and adult samples to test the prediction that performance on the “Gray Temporal Dots” measure would account for more variance in reading ability.

Finally, simple and multiple regression analyses was used to determine the relationship between performance on the “Gray Temporal Dots” and performance on other measures of temporal processing. Age and cognitive development were also considered in these analyses.
CHAPTER 4

RESULTS

The purpose of this study was to investigate the relationship between temporal processing and reading ability. More specifically, psychophysical measures were administered to children and adults to test the hypothesis that developmental dyslexia is associated with a deficit in the magnocellular visual pathway.

Performance on the experimental measure designed to elicit magnocellular activation demonstrated a significant linear relationship with passage reading performance in the adult sample, but not in the child sample. Contrary to predictions, there were no significant mean differences in performance on the psychophysical measure designed to elicit magnocellular functioning and the complimentary measure that used a diffuse red background to inhibit the selective properties of the magnocellular pathway. This finding was consistent across levels of reading proficiency in the adult and child samples. Finally, when the general temporal processing deficit theory was tested, performance on the psychophysical measure designed to elicit magnocellular activation demonstrated a significant linear relationship with performance on a composite measure of phonological awareness in the child and adult samples.

The means and standard deviations for demographic and psychometric variables within the adult and child groups are described in Table 3. Analyses were performed using descriptive statistics, correlation analyses, simple regression, multiple regression
and t-tests. This study’s three hypotheses are restated below, each proceeded by statistical results.

**Hypothesis I:**

Analysis of early visual processing and reading ability was expected to support the hypothesized relationship between magnocellular deficit and reading difficulty. In the child and adult samples, performance on the magnocellular-dependent task “Gray Temporal Dots” was expected to correlate positively with, and account for a significant portion of variance in performance on a measure of passage reading (Gray-Oral Reading Test- 3rd Edition, Passage Reading Index). Analysis of this relationship considered indices of broad intellectual functioning as well as more specific neurolinguistic abilities.

For the adult sample, performance on the magnocellular-dependent task “Gray Temporal Dots” accounted for a statistically significant amount of variance in performance on the measure of passage reading (GORT-3 Passage Reading Index) at the pre-selected alpha level of .05 (one-tailed) \( (1,26) r^2 = .40; F = 4.85; p = .04 \). This association is demonstrated visually in Figure 9. Developmental, cognitive and neurolinguistic variables were then included with the psychophysical measure in a multiple regression analysis. These variables included age, intellectual ability (WASI FSIQ), and basic reading skills (WRMT-R Word Identification; WRMT-R Word Attack). Results indicated that a regression model containing all of the named variables, including performance on the “Gray Temporal Dots” measure, accounted for a significant portion of variance in passage reading performance \( (5, 21) r^2 = .62; F = 9.26; p = .00 \).
Table 3.
Means and Standard Deviations for Age and Performance Variables for the Adult and Child Groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>Adult Group</th>
<th>Child Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>38.53 (5.27)</td>
<td>10.77 (1.95)</td>
</tr>
<tr>
<td>WASI FSIQ</td>
<td>102.61 (13.15)</td>
<td>97.45 (14.41)</td>
</tr>
<tr>
<td>Gray Temporal Dots</td>
<td>.73 (.22)</td>
<td>.41 (.26)</td>
</tr>
<tr>
<td>Red Temporal Dots</td>
<td>.73 (.22)</td>
<td>.38 (.21)</td>
</tr>
<tr>
<td>CTOPP Pho. Awareness</td>
<td>80.54 (14.14)</td>
<td>89.50 (11.45)</td>
</tr>
<tr>
<td>CTOPP Rapid Naming</td>
<td>97.96 (16.57)</td>
<td>94.47 (18.95)</td>
</tr>
<tr>
<td>GORT-3 Passage</td>
<td>98.75 (20.48)</td>
<td>83.90 (15.05)</td>
</tr>
<tr>
<td>WJC-R Mem. for Words</td>
<td>95.89 (13.20)</td>
<td>94.60 (19.38)</td>
</tr>
<tr>
<td>WJC-R Mem. for Sentences</td>
<td>96.21 (18.03)</td>
<td>95.60 (21.85)</td>
</tr>
<tr>
<td>WRMT-R Word I.D.</td>
<td>93.96 (9.72)</td>
<td>90.15 (10.46)</td>
</tr>
<tr>
<td>WRMT-R Word Attack</td>
<td>99.67 (13.17)</td>
<td>87.40 (20.7)</td>
</tr>
</tbody>
</table>
Individually, performance on the test of word identification accounted for variance in passage reading performance most reliably \((1, 26) r^2 = .56; F = 34.09; p = .00\). Variance in passage reading performance accounted for by the regression model including “Gray Temporal Dots,” WRMT-R Word Identification and WRMT-R Word Attack \((3, 23) r^2 = .62; F = 12.88; p = .00\) was roughly equivalent to the model that included only WRMT-R Word Identification and WRMT-R Word Attack \((2, 24) r^2 = .59; F = 18.64; p = .00\). The age of the adult subjects alone did not account for a significant portion of variance in passage reading performance \((1, 26) r^2 = .02; F = .06; p = .80\) or performance on the “Gray Temporal Dots” measure \((1, 26) r^2 = .07; F = 2.01; p = .17\).

For the child sample, performance on the magnocellular-dependent task failed to demonstrate a statistically significant linear relationship with performance on the measure of passage reading (GORT-3, Passage Reading Index) at the pre-selected alpha level of .05 (one-tailed) \((1, 17) r = -.24; p = .33\). This relationship is demonstrated visually in Figure 11. Developmental, cognitive and neurolinguistic variables were then included with the psychophysical measure in a multiple regression analysis. These variables included age, intellectual ability (WASI FSIQ), and basic reading skills (WRMT-R Word Identification; Word Attack). An overall regression model including all of the named variables, including performance on the “Gray Temporal Dots” measure, accounted for a significant amount of variance in passage reading performance \((5, 13) r^2 = .75; F = .843; p = .00\). This overall model was relatively unaffected when performance on the magnocellular-dependent task was removed from the series of predictor variables \((4, 14)\).
Figure 9.
Figure 10.
Figure 11.
\[ r^2 = .75; F = 11.35; p = .00. \] The model producing the greatest F-ratio in the child sample used WRMT-R Word Identification and subject age to account for variance in passage reading ability \((2, 16) r^2 = .73; F = 21.69; p = .00. \) Despite the use of an age-normed reading measure, subject age alone accounted for significant variance in passage reading performance \((1, 17) r^2 = .23; F = 5.06; p = .038, \) but did not account for significant variance in performance on the “Gray Temporal Dots” measure \((1, 18) r^2 = .00; F = .08; p = .93. \)

**Hypothesis II:**

In support of the divergent validity of the visual measures, mean performance on the “Gray Temporal Dots” measure and mean performance on the complimentary measure, “Red Temporal Dots,” were expected to differ within the entire sample of adults and within the entire sample of children. This difference was predicted to be more pronounced when only subjects with below average passage reading ability were included in analysis. Additionally, for the adult and child samples, it was expected that proficiency on the “Gray Temporal Dots” task would account for greater variance in reading ability than would proficiency on the “Red Temporal Dots” task. This finding was expected to be most significant for subjects with below average reading ability.

For the entire adult sample, a paired samples t-test indicated that there was not a statistically significant difference between mean performance on the “Gray Temporal Dots” measure and mean performance on the “Red Temporal Dots” measure \((1, 26) t = -.25, p = .80. \) There was also no significant difference in means between the visual tests when only below average passage readers (GORT-3 Passage Index \(\leq 80) \) were assessed \((1, 6) t = .42, p = .69. \) Subjects who were relatively proficient passage readers (GORT-3
Passage Index $\geq 90$) also did not demonstrate significant mean differences between the two visual measures (1, 19) $t = -.64, p = .53$. As reported in the testing of hypothesis I, performance on the magnocellular-dependent measure “Gray Temporal Dots,” by the entire adult sample accounted for a significant amount of variance in passage reading performance (GORT-3 Passage Index) (1, 26) $r^2 = .16; F = 4.85; p = .04$. As illustrated in Figure 10, performance on “Red Temporal Dots,” the visual measure designed to inhibit magnocellular functioning, accounted for a significant amount of variance in passage reading performance (1,26) $r^2 = .22, F = 7.14, p = .01$.

When analysis included only adult subjects who demonstrated below average passage reading ability (GORT-3 Passage Reading Index $\leq 80$), performance on the “Gray Temporal Dots” task did not account for a significant amount of variance in passage reading performance (1,6) $r^2 = .08; F = .45; p = .53$. For adult subjects with below average passage reading ability, performance on the comparison measure “Red Temporal Dots” task was also did not account for a significant amount of variance in passage reading performance (1, 6) $r^2 = .29; F = 2.10; p = .53$.

For adults considered relatively proficient readers (GORT-3 Passage Reading Index $\geq 90$), performance on the magnocellular-dependent “Gray Temporal Dots” accounted for a significant amount of variance in passage reading performance (1, 9) $r^2 = .37; F = 11.22; p = .00$. For this non-reading disabled group of adults, performance on the “Red Temporal Dots” task also accounted for a statistically significant portion of variance in passage reading performance (1, 8) $r^2 = .20; F = 4.5; p = .05$.

For the overall child sample, a paired samples t-test indicated that there was not a statistically significant difference between mean performance on the “Gray Temporal
Dots” measure and mean performance on the “Red Temporal Dots” measure (1, 17) $t = .81; p = .43$. There was also no significant difference in means between the visual tests when only below average passage readers (GORT-3 Passage Index $\leq 80$) were assessed (1, 7) $t = -.62; p = .55$. For non-reading disabled children (GORT-3 Passage Index $\geq 90$) there was also no significant mean difference between the two visual measures (1, 8) $t = 2.05; p = .07$.

As reported in the testing of hypothesis I, performance on the magnocellular-dependent measure “Gray Temporal Dots,” by the entire child sample did not account for a significant portion of variance in passage reading performance (GORT-3 Passage Index) (1, 19) $r^2 = .05; p = .33$. As illustrated in Figure 12, performance by the entire child sample on “Red Temporal Dots,” the measure designed to inhibit magnocellular activity, also did not account for a significant amount of variance in passage reading performance (1, 16) $r^2 = .11; F = 2.09; p = .17$.

For child subjects who demonstrated below average passage reading ability (GORT-3 Passage Index $\leq 80$), the amount of variance in passage reading accounted for by performance on the “Gray Temporal Dots” task was not significant (1, 7) $r^2 = .12; F = 2.09; p = .17$. For the children with below average passage reading ability (GORT-3 Passage Index $\leq 80$), the amount of variance in passage reading performance accounted for by performance on the visual measure, “Red Temporal Dots,” was not statistically significant at the pre-selected alpha level of .05 (1, 6) $r^2 = .45; F = 5.50; p = .06$.

When analysis was limited to children who demonstrated relative proficiency on the measure of passage reading (GORT-3 Passage Reading Index $\geq 90$), performance on the “Gray Temporal Dots” measure did not account for a significant amount of variance
in passage reading performance (1, 8) $r^2 = .05; t = -.63; p = .55$. For this non-reading disabled group of child subjects, performance on the complimentary visual measure, “Red Temporal Dots” also did not account for a significant portion of variance in passage reading performance (1, 7) $r^2 = .00; t = .01; p = .91$.

**Hypothesis III:**

There is a hypothesized convergence between the neuroanatomical pathways that enable visual motion processing and auditory processing of language. The former innervates the inferior parietal cortex and the latter traverses regions proximal to the inferior parietal cortex, such as the posterior superior temporal gyrus, the inferior parietal lobule, and the angular gyrus. Consistent with the theory that a common developmental perturbation may account for deficiencies in the temporal processing of visual and auditory stimuli, performance on indexes of phonological processing (CTOPP Phonological Awareness Composite), verbal fluency (CTOPP Rapid Naming Composite) and short-term auditory processing and retrieval (WJC-R Memory for Words; WJC-R Memory for Sentences) were expected to correlate positively with performance on the “Gray Temporal Dots” task. Age was included as a variable since there is a developmental curve associated with the attainment and decrement of perceptual and processing speed.

For the overall adult group, results of correlation analysis indicated that there were three statistically significant associations at the .05 alpha level (2-tailed). The “Gray Temporal Dots” measure correlated positively with phonological awareness (1, 27) $r = .43; p = .02$, phonological processing ability was strongly associated with passage reading performance (1, 27) $r = .49; p = .01$, and performance on the WJC-R Memory for
Words and WJC-R Memory for Sentences subtests were positively correlated (1, 27) $r = .48, p = .01$. Performance on the “Gray Temporal Dots” measure and the phonological awareness index combined to account for significant variance in passage reading performance (2, 25) $r^2 = .24; F = 8.04; p = .00$. This model accounted for variance in passage reading at a level comparable to the measure of phonological processing alone (2, 25) $r^2 = .28 ; F = 4.8 ; p = .02$

The results of this correlation analysis for the child group were statistically significant for five linear associations at the pre-selected .05 alpha level (2-tailed). Like the adult sample, performance on the WJC-R subtests Memory for Words and Memory for Sentences were positively correlated (1, 19) $r = .65, p = .00$. Subject age and performance on the composite measure of phonological processing (CTOPP Phonological Awareness) correlated negatively (1, 19) $r = -.46, p = .04$. Performance on the WJC-R Memory for Words subtest and performance on the CTOPP Phonological Awareness demonstrated a significant positive correlation (1, 19) $r = .49, p = .30$. Phonological awareness and passage reading performance demonstrated a significant positive association (1, 19) $r = .48; p = .04$. Finally, performance on the “Gray Temporal Dots” measure and performance on the measure of phonological awareness combined to account for significant variance in passage reading performance (2, 16) $r^2 = .23; F = 5.08; p = .04$, which was comparable to the variance accounted for by the measure of phonological processing alone (1, 17) $r^2 = .32 ; F = 3.73; p = .05$. 
CHAPTER 5

DISCUSSION

The general purpose of this study was to investigate the relationship between temporal processing and reading ability. The more specific aim was to test the hypothesis that developmental dyslexia is associated with a deficit in the magnocellular visual pathway. Standardized psychometric instruments were used to assess children and adults with a wide range of reading ability. In addition, experimental psychophysical measures were utilized that replicated visual stimuli described by Eden and colleagues (1995). A complimentary psychophysical measure designed to inhibit a degree of magnocellular activation was introduced in this study. Finally, to investigate the notion that diffuse neurological irregularities associated with developmental dyslexia (Galaburda et al., 1985) are inconsistent with a single core processing deficit (e.g., phonological awareness, verbal fluency or early visual processing), the hypothesized co-occurrence of temporal processing deficits and reading ability was examined.

Relationship Between Magnocellular Functioning and Reading Ability

The results of this study indicate that magnocellular functioning may be related to reading ability. Interestingly, this significant association was found in the adult sample, but not in the child sample. The non-significant relationship in the child group suggests possible limitations of these visual stimuli. Further research is needed to determine whether these visual stimuli can be useful for identification of young dyslexics in need of pre-reading intervention.
One possible explanation for the non-linear relationship between performance on the magnocellular-dependent task and passage reading ability in the child group takes into account chronological age. In the adult sample, age was not significantly associated with either performance on the magnocellular-dependent task or with performance on the measure of passage reading ability. Conversely, age was significantly associated with the latter in the child sample. This was unexpected since the measure of passage reading was normed by age; however, the mean passage reading score for the child sample was approximately one standard deviation below the mean for the normative sample (see Table 3). This may have been due to the significant proportion of relatively young clinic-referred children in comparison to several older siblings who were normal readers. Thus, as passage reading scores varied significantly by age, and psychophysical performance did not vary by age, a non-linear relationship emerged between performance on the magnocellular-dependent visual task and the measure of passage reading ability (see Figure 9).

**Utility of a Complimentary Visual Measure with a Diffuse Red Background**

The results of this study demonstrated no meaningful difference between group performance on the “Gray Temporal Dots” task, designed to elicit predominant magnocellular activation, and the complimentary “Red Temporal Dots” task designed to inhibit a degree of magnocellular activation. In fact, there was a significant linear relationship between performance on the “Red Temporal Dots” task and the measure of reading ability in the adult sample, just as there was a significant linear relationship between the “Gray Temporal Dots” task and passage reading ability. Conversely, there was no significant linear relationship between performance on either
the “Gray Temporal Dots” task or the “Red Temporal Dots” task and the measure of passage reading ability in the child sample.

Since much of the preceding literature has used separate “dyslexic” and “non-dyslexic” groups, the adult and child samples were split depending upon passage reading ability. Unexpectedly, the only significant relationships between magnocellular-dependent visual processing and passage reading ability was evident when regression analyses were limited to adult subjects with average to above average reading proficiency (GORT-3 Passage Reading Index > 90). Conversely, there was no significant linear relationship between magnocellular-dependent visual processing and passage reading ability for the adult subjects in this sample whose passage reading was relatively poor (GORT-3 Passage Reading Index < 80). This is inconsistent with Eden and colleagues’ (1995) finding that dyslexic adults displayed significant difficulty on a measure of magnocellular-dependent visual processing in comparison to normal reading subjects.

Global Temporal-Processing Deficits

Some researchers contend that magnocellular dysfunction in developmental dyslexia is one characteristic of a more global temporal processing deficit. In addition to psychophysical (Eden et al., 1995; Demb et al., 1998; Cornelissen, 1997) and functional neuroimaging (Eden et al., 1996; Demb et al., 1998) results that support a visual processing speed deficit in dyslexia, there is evidence that children with language disabilities process (Tallal et al., 1993) and retrieve (Wolf & Bowers, 1999) speech sounds more slowly than the average child. Anatomically, there is a hypothesized convergence between the neuroanatomical pathways that enable visual motion processing and auditory processing of language. The former innervates the inferior parietal cortex
and the latter traverses regions proximal to the inferior parietal cortex, such as the posterior superior temporal gyrus, the inferior parietal lobule, and the angular gyrus.

In this study, performance on the magnocellular-dependent measure and performance on the composite of phonological awareness were significantly related in the adult sample. Phonological awareness accounted for a significant portion of variance in passage reading ability, as did a regression model that included phonological awareness and performance on the measure of magnocellular processing. Phonological awareness was also significantly related to passage reading in the child sample. While the magnocellular-dependent task did not account for significant variance in passage reading independently, a regression model including both phonological awareness and magnocellular processing produced a significant result.

These results support a link between temporal processing deficits in vision and audition. Further, reading ability was compromised when inefficiencies impacted both sensory processing systems. Since performance on the psychophysical instruments alone only significantly accounted for variance in reading ability in the adult sample, their utility as “markers” of developmental dyslexia may be limited. To reflect current theory, and the results of this study, assessment of young children should measure a more global index of temporal processing ability. Researchers should begin to develop integrated batteries that assess temporal processing of both visual and auditory stimuli in order to estimate the likelihood that a child will present with reading difficulty at school age.

Limitations of this Study

Efforts were made to control for non-experimental error in this investigation; however there are limitations inherent in stimulus design and subject sampling that
warrant discussion. The most pressing concern from the outset of this study was the need for highly precise psychophysical stimuli. Although great care was taken to program and calibrate the computerized display to match the stimuli described by Eden and colleagues (1995), some variability in luminance was expected between computer monitors. This was evident when luminance levels were measured with a photometer at nine points along the perimeter and interior of the monitor screen. Some regions demonstrated up to 8% difference in luminance (cd/m²) from other points on the monitor, while all nine zones contributed to an overall mean luminance within the scotopic range. It is unknown whether these slightly unequal luminance values across the monitor screen effected performance on the psychophysical tasks.

Other possible limitations associated with this study relate to the composition of the child and adult samples. While recent fMRI investigations of the magnocellular deficit hypothesis have relied predominantly upon young adults between 17 and 25 years of age (Demb et al., 1998; Eden et al., 1996), this specific subset of the population was not included in this study. This was due to the fact that recruitment for the subject pool was guided by a N.I.H. grant that targeted children ages 8 to 12 years of age, their siblings, and their parents. Analyses determined that subject age did not significantly effect performance on the psychophysical task; however it should not be assumed that these results may not generalize to age groups who were not included in this study.

The manner in which the psychophysical measures in this study were administered may also limit reliability and validity. While a uniform field of vision was maintained with a chin rest and an adjustable chair, feedback from several subjects suggested that the chin rest may have initially been a distraction during the assessment.
Subjects were permitted to adjust the height of the chair to their preference and to adjust the height of the chin rest within a 2 cm. vertical range relative to the center of the monitor screen. While most participants responded that they were relaxed and not concerned about keeping their head still, a few others mentioned that it took several minutes to become accustomed to the chin rest. A brief trial period may have helped to ensure that all subjects were sufficiently accustomed to the chin rest prior to data collection.

In an effort to limit the effect of sustained visual attention, a significant modification in administration was made relative to the original investigation (Eden et al., 1995). Rather than using a standard latency period between visual presentations, and assuming that all subjects were adequately focused to perform 72 consecutive trials at this rate, subjects in this study were empowered to initiate each trial when they were ready to proceed by pressing the spacebar. The time required for subjects to respond to the preceding trial typically lasted several seconds, which was sufficient to prevent backward masking. Subject-initiated trials might have controlled for significant variance in auditory sustained attention; however, this modification is also considered a limitation to this study since it is not universal feature in the literature and may hinder direct comparison with other results.

Similarly, a modification was also made relative the original investigation (Eden et al., 1995) to control for variance in motor ability between subjects. It was felt that the subjects position in the chin rest, particularly within the darkened room, might be conducive to manual errors. Rather than asking participants to input their answer by pressing a number key, verbal responses were elicited which were recorded by the
administrator. This enabled subjects to maintain visual fixation on the monitor instead of having to glance downward towards the keyboard. While this modification in administration might have controlled for significant variance in motor ability; it is also considered a limitation to this study since it is not consistent with most previous designs.

Finally, by avoiding reliance upon group differences in “dyslexic” and “non-dyslexic” samples, and instead focusing on the relationships between temporal processing variables and reading-related variables, the results of this experiment are not amenable to direct comparison with the predominance of literature in this area of study. Further, the selection of variables considered in regression models was intended to be broad, but is by no means exhaustive of possible developmental or neuropsychological factors of temporal processing or reading ability.

**Directions for Further Research**

This study provided three principal findings: 1) There was a significant positive relationship between magnocellular pathway functioning and reading ability in adults, 2) the magnocellular-dependent measure and the complimentary measure incorporating a diffuse red background did not produce significantly divergent results, and 3) magnocellular processing, phonological processing, and passage reading abilities were significantly related in the child and adult samples, consistent with the temporal processing deficit theory.

Too often in the visual processing literature there is significant heterogeneity between similarly termed “dyslexic” and “non-dyslexic” groups. As was demonstrated in this investigation, future researchers are encouraged to depart from the common analysis of group differences in order to more accurately reflect the continuous nature of variables
related to neuropsychological performance and reading ability. Also consistent with this study, the list of developmental and neuropsychological variables considered relevant in regression analyses must continue to expand in order to test the limits of the global temporal processing hypothesis. Variables considered within a broad analysis should be examined across several studies until the particular developmental characteristic or processing ability is repeatedly shown to be insignificant to the development of reading ability.

The stimuli described in most psychophysical (Eden et al., 1995; Mason et al., 1993; Martin & Lovegrove, 1987) and neuroimaging (Eden et al., 1996; Demb et al., 1998) studies in this field were based upon sound visual science. Until results become more consistent; however, questions will remain regarding the differential effects of each stimulus upon performance. In the future, researchers are urged to collaborate more closely with basic visual scientists to facilitate the creation of a visual stimulus that achieves broad consensus and widespread application in the measurement of early visual pathway functioning.

The need for consistent stimuli between studies also applies to complimentary psychophysical measures. To date, functional neuroimaging studies have not included enough participants to conclusively assert the relationship between psychophysical performance and neurophysiological activation. Until the sample sizes in fMRI studies increase, performance on complimentary psychophysical measures provides important information. Only when magnocellular functioning is inhibited to some degree in a complimentary measure can researchers make a comparison that determines whether the measure of magnocellular functioning is valid.
In this study, a diffuse red background was used for this purpose, consistent with evidence in the visual science literature that this psychophysical property diminishes magnocellular activation (Brown & Koch, 2000). Although the results of this study did not support the effectiveness of one particular application of a diffuse red background, further applications are needed to determine its overall utility in the study of perceptual processing and reading. Future researchers are encouraged to examine evidence from recent gender studies suggesting that other applications of a diffuse red background have provided significant information about the early visual pathways (J. M. Brown, personal communication, January 15, 2002).

Finally, while there will always be a need for refinement of the classic studies that spurred interest in the biological bases of developmental dyslexia (Geschwind & Levitsky, 1968; Galaburda, 1985), future breakthroughs in the study of temporal processing and reading ability, and more specifically early visual processing, will most certainly occur through the application of functional neuroimaging.

A logical application of fMRI following this study would seek to determine the neurophysiological processes that underly these behavioral results. Such an investigation might include analysis of cerebral regions believed to mediate visual and auditory temporal processing and reading performance. With regard to reading, detection of hemodynamic activity should focus upon cerebral activation within the inferior parietal cortex, including the posterior superior temporal gyrus, the inferior parietal lobule, and the angular gyrus. At the same time, analysis of nearby perceptual processing areas should include the inferior parietal cortex and subcortical pathways connecting the medial geniculate nucleus of the thalamus to the primary auditory cortex in the temporal
lobe. Characteristic under-activation of several of these neuroanatomical regions during psychophysical stimulus presentation, occurring in association with significant reading difficulty, would support the global temporal processing hypothesis.
REFERENCES


Semrud-Clikeman, M., Hynd, G. W., & Novey, E. S. (1991). Dyslexia and brain morphology: Relationships between neuroanatomical variation and


CENTER FOR CLINICAL AND DEVELOPMENTAL NEUROPSYCHOLOGY

CONSENT FORM

I, ________________________________, agree (or give my consent) for (child’s name) ______________________________ to be administered a visual/perceptual test at the Center for Clinical and Developmental Neuropsychology under the supervision of Dr. George Hynd (542-4265). I understand that this participation is entirely voluntary; I can withdraw my consent (or my child may withdraw consent) at any time without penalty and have the results of the participation, to the extent that it can be identified as mine (or my child’s), removed from research records.

1) The purpose of this test is to study the relationship between visual/perceptual processes and reading ability. Thus, I understand that at some later date the data from my (or my child’s) evaluation may be included in a research project. I also understand that in no fashion will these results be used in any identifiable manner. Thus, I am assured that should these results be used in later research, my confidentiality is protected.

2) Participation involves no known physical, psychological, social or legal risks.

3) Dr. Hynd will answer any questions about the clinic procedure either now or at any later time.

_______________________
Participant
[or parent or guardian]

_______________________
Date
ADMINISTRATOR:

“The purpose of this test is to study how people see dots on this computer screen. If you decide that you don’t want to do this test please let me know at any time, even if we have already started. Again, it is no problem if you decide that you do not want to do this test. In order to do this test I will turn off the lights for about 5 minutes at a time. Please let me know at any time if you want me to turn the lights back on and I will quickly turn them back on for you. Remember, you can ask me or Dr. Hynd if you have any questions. Would you like to try this test?”