# **Evaluation of the Cellfield Intervention**

# for Dyslexia:

# **Behavioural and Electrophysiological**

Outcomes

# **Jacqueline Sander**

# **Dipl. Psych. University of Wuppertal**

Submitted in fulfillment of the requirements for the degree of Master of

Science

University of Tasmania

November 2008

٨

This thesis contains no material which has been accepted for a degree or diploma by the university or any other institution, except by way of background information and duly acknowledged in the thesis, and that to the best of my knowledge and belief, the thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

This thesis may be made available for loan and limited copying in accordance with the *Copyright Act 1968*.

.

Wall Sud

Jacqueline Sander November 2008

.

#### Abstract

The current study aimed to contribute to the identification of potential intervention programs for dyslexia by comparing the effects of one commercial program, the Cellfield intervention, to those of a placebo program in adolescents with reading and/or spelling difficulties. The Cellfield intervention is a comprehensive computer-based approach to treating dyslexia, which involves visual, phonological, and visual-to-phonological exercises. The efficacy of the intervention was assessed using behavioural (reading, phonological, and spelling measures and reaction time and accuracy) and electrophysiological (P2, N4, and LPC components of the ERP) indicators of change. Twelve students (aged between 12 and 14 years) identified as experiencing reading and spelling difficulties participated, with seven students completing the Cellfield intervention and five students the placebo program. All participants completed a variety of reading and literacy tests and phonological and lexical decision tasks and an incongruent sentence ending task during which event-related potentials (ERPs) were recorded. All tasks were administered before and immediately after the completion of the Cellfield intervention and placebo program respectively. Both groups then engaged in a follow-on practice for three weeks, focusing on training in reading fluency, comprehension, and spelling. Outcome measures were assessed again after completion of the follow-on training. The Cellfield group, but not the Placebo group, showed a significant decrease in overall risk for dyslexia and a significant improvement in phonological decoding skills from pre- to post-test. These gains in phonological skills were maintained at follow-up for the Cellfield group. Higher-order literacy skills, including text reading comprehension, accuracy, and fluency did not change significantly following the Cellfield intervention. However, after the three-week follow-on practice, the Cellfield and Placebo groups showed significantly improved text reading comprehension and accuracy from pre- to follow-up-test. Spelling skills remained unaffected by either the Cellfield intervention or follow-on practice. Results from the ERP studies were less conclusive. For all three experimental tasks, neither reaction time nor latency data discriminated the two groups over time. Amplitude data indicated some neural changes

ii

within the Cellfield group, who demonstrated decreased amplitudes in the right hemisphere (LPC), and increased amplitudes in the left hemisphere (N4) from pre-, to post, to follow-uptest compared to the Placebo group. Increased left lateralised processing and its relationship to normal language processing are discussed. Overall, reading data suggest some beneficial effects of the Cellfield intervention. Neural changes due to the Cellfield intervention are tentative and need further investigation.

۰.

~

#### Acknowledgements

I would like to express my gratitude to all those who gave me the possibility to complete this thesis.

First and foremost, I would like to express my gratitude to my supervisors, Frances Martin and Nenagh Kemp who have provided professional guidance and support as well as emotional understanding. Thank you to you both for making it possible to submit this thesis on schedule. In addition, you were always accessible when needed and willing to help with my research.

Second, much respect and appreciation to Dimitri Caplygin without whose agreement this study would have not been possible. Thank you for the training sessions on the Cellfield intervention, your inspirations on the research and your willingness to allow us to evaluate the Cellfield intervention at no cost.

Thank you to the Tasmanian Ethics Committee and the Department of Education for the permission to commence this research.

Thank you to all those 'behind the scenes': Richard Thomson for computer programming support, Vlasti Broucek for IT support, and Sue Ross for administrative support and all other academic and support staff at the School of Psychology.

My special thanks to Finian MacCana who has so patiently tested the participants' visual abilities and was never short of a smile.

For financial support I would like to thank the German Academic Exchange Service (DAAD) and the School of Psychology at UTAS as this project would not have been possible without their funding contributions.

My gratitude and appreciation extends to all participants in my study who have contributed patiently so many hours of their time to this research and who trusted me. Equally I want to thank the principals, teachers and administration staff at the two co-operating high schools (names not mentioned for confidentiality purposes). In particular much respect and a big 'thank-you' goes to two literacy support teachers from one school, and one assistant principal from the other school. Thank you and you are doing an amazing job!

I want to thank my family and friends back in Germany for supporting me with their emotional encouragement despite the fact of me being so far away from home.

Last but by no means least I want to thank my partner Hakuei who has provided logistical and psychological expertise for this research, and always believed in me. Thanks for keeping me sane.

۰,

# TABLE OF CONTENTS

| Chapter 1: Overview of the Thesis                                     | 1         |
|---|-----------|
| Chapter 2: Dyslexia: An Overview                                      | 4         |
| Dyslexia: An Overview of Definitions and Terms                        | 4         |
| Prevalence of Dyslexia  |           |
| Symptoms, Developmental Course and Assessment of Dyslexia             | 5         |
| Co-morbidities of Dyslexia  | 8         |
| Chapter 3: Models of Normal Reading Acquisition and Visual Word Recog | nition 10 |
| Developmental Stage Models of Normal Reading                          |           |
| Visual Word Recognition Theories                                      |           |
| Implications for Dyslexia   |           |
| Chapter 4: Current Understanding of Dyslexia                          | 20        |
| Introduction: Current Understanding of Dyslexia                       | 20        |
| The Magnocellular, Basic Auditory and Temporal Theories               | 21        |
| The Cerebellar Theory   | 25        |
| The Balance Theory  | 26        |
| The Phonological Theories   | 27        |
| The Double-deficit Theory and other Multidimensional Approaches       |           |
| Chapter 5: Neurobiological Basis of Dyslexia                          | 35        |
| Genetic Influences on Dyslexia  |           |
| Anatomical Evidence of Dyslexia                                       |           |
| Neural Correlates of Dyslexia: Imaging Studies                        |           |
| Neural Correlates of Dyslexia: ERP studies                            |           |
| The Event-related-potential Technique                                 |           |
| ERPs and Normal Linguistic Processing                                 |           |
| ERPs and Linguistic Processing in Dyslexia                            |           |
| Developmental Considerations  |           |
| Summary: ERPs and Dyslexia  |           |

٨

| Summary: Neural Correlates of Dyslexia   | 53 |
|--|----|
| Chapter 6: Interventions for Dyslexia and Their Outcomes                       | 55 |
| Introduction: Intervention Studies in the Field of Dyslexia                    | 55 |
| Evaluation of Interventions Targeting Basic Non-linguistic Processing          | 56 |
| Interventions Targeting Visual Processing                                      | 56 |
| Interventions Targeting Auditory and Temporal Processing                       | 58 |
| Interventions Targeting Sensorimotor Processing                                | 59 |
| Interventions Targeting Lateralised Processing                                 | 60 |
| Evaluation of Linguistic Interventions   | 62 |
| Interventions Targeting Phonological Processing                                | 62 |
| Evaluation of Combined Interventions   | 63 |
| Beyond the Intervention: Other Variables that Influence Intervention Outcomes. | 67 |
| Neural Changes Following Intervention for Dyslexia                             | 70 |
| Summary: Intervention Studies and Their Outcomes                               | 72 |
| Chapter 7: Rationale and General Aims  | 74 |
| Chapter 8: Method  |    |
| Participants   |    |
| Materials  |    |
| Initial Screening Tests  | 83 |
| Pre-, post-and follow-up Psychometric Tests                                    |    |
| Experimental Stimuli   |    |
| Apparatus and EEG Recording  |    |
| The Cellfield, Placebo and Follow-on Practice Programs                         |    |
| Procedure  |    |
| Design   |    |
| Literacy Data Analyses   |    |
| Psychophysiological Data Analyses (Behavioural and ERP)                        |    |
| Chapter 9: Results   |    |
| - Treatment Fidelity   |    |
|  |    |

| Literacy Measures   | 104 |
|---|-----|
| Psychophysiological Measures  | 112 |
| Behavioural Measures  | 112 |
| ERP Measures  | 119 |
| Chapter 10: Discussion  | 152 |
| Literacy Outcomes   | 152 |
| ERP and Behavioural Outcomes  | 159 |
| Integration of the Literacy, ERP and Behavioural Outcomes                 | 166 |
| Limitations   | 170 |
| Future Research   | 171 |
| References  | 175 |
| Appendicies   |     |
| Appendix A: Medical Questionnaire   |     |
| Appendix B: Parents Questionnaire   | 208 |
| Appendix C: Word Stimuli Presentend for the Phonological and Lexical Task | 213 |
| Appendix D: Sentences Presented for the Sentence Task                     | 215 |
| Appendix E: Monitor Sheet for Home Reading Practice                       | 217 |
| Appendix F: Standard Instructions for the ERP tasks                       | 218 |
| Appendix G: Stem and Leaf Plots for the Literacy Data at Pre-test         | 219 |
|   |     |

;

.

-

## LIST OF FIGURES

| Figure 1. A dual-route model of reading (Temple, 1997)                                    |
|---|
| Figure 2. Connectionist model by Seidenberg and McClelland (1989)                         |
| Figure 3. Multiple deficit model by Pennington (2006)                                     |
| Figure 4. Gray's schematic illustration of the brain and its language-associated regions  |
| Figure 5. Time sequence for the lexical and phonological task                             |
| Figure 6. Time sequence (ms) for the sentence task  |
| Figure 7. Mean DST-S raw scores for Cellfield and Placebo group at pre- and post-test 107 |
| Figure 8. Mean External Locus raw scores for Cellfield and Placebo group at pre-          |
| and post-test   |
| Figure 9. Mean Word Attack raw scores for Cellfield and Placebo group at pre-,            |
| post- and follow-up-test  |
| Figure 10. Mean accuracy for Cellfield and Placebo group for the sentence task,           |
| at pre- and post-test   |
| Figure 11. Mean missing responses for Cellfield and Placebo group for the                 |
| sentence task, at pre- and post-test  |
| Figure 12. Mean missing responses for Cellfield and Placebo group for the                 |
| sentence task, at pre-, post- and follow-up-test  |
| Figure 13. Mean reaction time for Cellfield and Placebo group together for the            |
| lexical and phonological task at pre- and post-test                                       |
| Figure 14. Mean accuracy for Cellfield and Placebo group for the lexical and              |
| phonological task at pre- and post-test   |
| Figure 15. Group grand mean averages for incongruent endings in the sentence              |
| task at pre- and post-test  |
| Figure 16. Group grand mean averages for congruent endings in the sentence task           |
| at pre- and post-test   |
| Figure 17. Group grand mean averages for difference waveforms in the sentence             |
| task at pre- and post-test  |

-

| Figure 18. Group grand mean averages for incongruent endings in the sentence              |
|---|
| task at pre- and follow-up-test122  |
| Figure 19. Group grand mean averages for congruent endings in the sentence task           |
| at pre- and follow-up-test  |
| Figure 20. Group grand mean averages for difference waveforms in the sentence             |
| task at pre- and follow-up-test   |
| Figure 21. Mean N4 amplitude for Cellfield and Placebo group, at left, mid, and           |
| right sites, at pre- and post-test  |
| Figure 22. Mean LPC amplitude for Cellfield and Placebo group at left, mid, and           |
| right sites, at pre- and post-test  |
| Figure 23. Mean LPC latency for Cellfield and Placebo group together at frontal, central, |
| and central-parietal sites, over left, mid, and right sites, at pre- and post-test127     |
| Figure 24. Mean LPC amplitude for Cellfield and Placebo group together for incongruent    |
| and congruent endings, at pre-, post- and follow-up-test                                  |
| Figure 25. Mean LPC amplitude for Cellfield and Placebo group, at left, mid, and right    |
| sites, at pre-, post- and follow-up-test  |
| Figure 26. Mean LPC latency for Cellfield and Placebo group, at pre-, post- and           |
| follow-up-test130   |
| Figure 27. Group grand mean averages for pseudo homophones in the                         |
| phonological task at pre- and post-test   |
| Figure 28. Group grand mean averages for nonwords in the phonological task                |
| at pre- and post-test   |
| Figure 29. Group grand mean averages for real words in the lexical task at                |
| pre- and post-test  |
| Figure 30. Group grand mean averages for pseudo homophones in the lexical task            |
| at pre- and post-test132  |
| Figure 31. Group grand mean averages for the pseudo homophones in the                     |
| phonological task at pre- and follow-up-test  |

| Figure 32. Group grand mean averages for the nonwords in the phonological task   |
|--|
| at pre- and follow-up-test133  |
| Figure 33. Group grand mean averages for the real words in the lexical task  |
| at pre- and follow-up-test134  |
| Figure 34. Group grand mean averages for the pseudo homophones in the  |
| lexical task at pre- and follow-up-test134   |
| Figure 35. Mean P2 amplitude for Cellfield and Placebo group, at pre-, post- and   |
| follow-up-test136  |
| Figure 36a. Mean N4 amplitude for Cellfield and Placebo group at left frontal,   |
| left central, and left central-parietal sites, at pre- and post-test   |
| Figure 36b. Mean N4 amplitude for Cellfield and Placebo group at mid frontal,  |
| mid central, and mid central-parietal sites, at pre- and post-test   |
| Figure 36c. Mean N4 amplitude for Cellfield and Placebo group at right frontal,  |
| right central, and right central-parietal sites, at pre- and post-test   |
| Figure 37. Mean N4 latency for Cellfield and Placebo group together for the lexical and  |
| phonological task, at pre- and post-test141  |
| Figure 38a. Mean N4 amplitude for Cellfield and Placebo group at left frontal, left central,   |
| and left central-parietal sites, at pre-, post-, and follow-up-test  |
| Figure 38b. Mean N4 amplitude for Cellfield and Placebo group at mid frontal, mid central,   |
| and mid central-parietal sites, at pre-, post-, and follow-up-test   |
|  |
| Figure 38c. Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central,   |
| <i>Figure 38c.</i> Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre-, post-, and follow-up-test             |
| <ul> <li>Figure 38c. Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre-, post-, and follow-up-test</li></ul> |
| <ul> <li>Figure 38c. Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre-, post-, and follow-up-test</li></ul> |
| <ul> <li>Figure 38c. Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre-, post-, and follow-up-test</li></ul> |
| <ul> <li>Figure 38c. Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre-, post-, and follow-up-test</li></ul> |
| <ul> <li>Figure 38c. Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre-, post-, and follow-up-test</li></ul> |

| Figure 42. | Mean LPC amplitude for Cellfield and Placebo group together for the                |    |
|------------|--|----|
|            | lexical and phonological task, at central-parietal sites, pre- and post-test14     | 18 |
| Figure 43. | Mean LPC latency for Cellfield and Placebo group at left, mid, and right sites, at |    |
|            | pre- and post-test14   | 19 |
| Figure 44. | Mean LPC amplitude for Cellfield and Placebo group at pre-, post- and              |    |
|            | follow-up-test1:   | 50 |

# LIST OF TABLES

| Table 1. Proposed Stages of Reading Development                                    | 10  |
|--|-----|
| Table 2. Mean Pre-Test Screening Raw Scores for Cellfield and Placebo Group        | 82  |
| Table 3. Tests for the Assessment of Dyslexia and Reading-Related Skills           | 87  |
| Table 4. Examples of Congruent and Incongruent Sentence Endings for the            |     |
| Sentence Task.   | 90  |
| Table 5. Description and Examples of the Cellfield Exercises                       | 94  |
| Table 6. Mean Literacy Raw Scores for Cellfield and Placebo Group at Pre-          |     |
| and Post-test  | 105 |
| Table 7. Mean DST At-risk Indexes for Cellfield and Placebo Group at Pre-          |     |
| and Post-test  |     |
| Table 8. Mean Literacy raw Scores for Cellfield and Placebo Group at Pre-,         |     |
| Post- and Follow-up-test   |     |
| Table 9. Mean Literacy Standard Scores for Cellfield and Placebo Group at Pre-,    |     |
| Post- and Follow-up-test   | 110 |
| Table 10. Mean Stem and Leaf Plots for the Literacy Measures for the Cellfield and |     |
| Placebo group at Pre-test  | 219 |

#### **Chapter 1: Overview of the Investigation**

Intervention research in the field of dyslexia has confirmed the efficacy of phonological based intervention programs for many dyslexic children (Alexander & Slinger-Constant, 2004; Foorman, Francis, Fletcher, Schatschneider, & Mehta, 1998; Lovett & Steinbach, 1997; Torgesen, Wagner, Rashotte, Burgess, & Hecht, 1997a; Vellutino et al., 1996). Most significantly, gains in phonological skills, reading accuracy and comprehension have been observed following intensive phonological intervention.

However, in these studies, reading fluency remained unaffected, improvements were not consistently generalised to new reading and learning material, and some children showed resistance to intervention and did not benefit at all (Shaywitz, Morris & Shaywitz, 2008; Tijms & Hoeks, 2005; Torgesen et al., 2001).

The development of combined intervention methods, integrating different aspects such as fluency, phonology, basic auditory/visual processing, multi-sensory processing, and orthographic to phonological processing, have given hope for the treatment of dyslexia (Denton, Fletcher, Anthony, & Francis, 2006; Torgesen, Rashotte, & Alexander, 2003; Wolf, 1999). However, according to Alexander and Slinger-Constant (2004) methodologically sound studies are scarce and findings are often inconclusive. Similarly research that has evaluated commercial programs, such as the Orton-Gillingham method, Lindamood, Reading Recovery, Cellfield intervention and Dore program has revealed equivocal results (Maskel & Felton, 2001; Oakland, Black, Stanford, Nussbaum, & Balisse, 1998; Prideaux, Marsh, & Caplygin, 2005; Reynolds, Nicolson, & Hambly, 2003), in particular because some evaluations were conducted in tied cooperation with the companies promoting these commercial programs. This clearly raises some questions about the studies' validity.

A limitation of most intervention research with dyslexic individuals is the lack of a control group: The three possible types of control groups are (1) a group that does not receive the intervention, (2) a group that receives a different intervention or (3) a placebo program (with no literacy practice). Lyon and Moats (1997) review the methodological

considerations in reading intervention research and report that such research may have been hampered by design limitations such as the lack of measures of follow-up intervention gains, the study of heterogeneous samples, lack of control of potential confounding variables such as socio economic status, behavioural problems and poorly described interventions.

The primary aim of the current study was to evaluate a commercial program for dyslexia, called the Cellfield intervention, and compare its efficacy to that of a placebo program in a sample of children with reading and spelling difficulties. The evaluation was conducted as a randomised controlled trial and a large number of the methodological factors highlighted by Lyon and Moats were integrated into the study's design.

The first six chapters are concerned with setting an empirical and theoretical framework for the current thesis, emphasising the importance of intervention research in the field of learning difficulties. In Chapter 2 an overview of developmental dyslexia including definitions, prevalence, symptoms and assessment is given. Chapter 3 provides a summary of models of normal reading development (Ehri, 2005; Frith, 1985; Jorm & Share, 1983; Marsh, Friedman, Welch, & Desberg, 1981; Stuart & Coltheart, 1988) and theories on visual word recognition (Coltheart, 1978; Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001; Seidenberg, 2005; Temple, 1985). Chapter 4 outlines major theories on the causes of dyslexia discussing particularly two major areas: linguistic (phonological) and non-linguistic theories (e.g., magnocellular theory, auditory temporal processing). This chapter concludes with a description of multidimensional modelling of dyslexia. Chapter 5 provides a review of the neurobiological basis of dyslexia. Genetic and anatomical evidence is reported first, followed by neural correlates of dyslexia which have been increasingly demonstrated in imaging and electrophysiological research. The ERP components that are of particular importance within the scope of the current thesis, N4, LPC and P2, are reviewed, and findings for normal and dyslexic samples are reported. Results of intervention studies on dyslexia are outlined in Chapter 6. Contributions from phonological and combined intervention programs are contrasted with the outcomes from basic auditory, visual and sensorimotor programs. The importance of extraneous factors which influence intervention

outcomes are also highlighted in Chapter 6 followed by a review of those few studies that have investigated neural changes following various interventions. Chapter 7 provides a general rationale and outlines the general aims for the current study. Finally Chapters 8, 9 and 10 outline the method, results and discussion of this intervention study, concluding with a summary on limitations of the current study and future research possibilities for intervention research.

.

6

# Chapter 2: Dyslexia: An Overview

#### Developmental Dyslexia – An Overview of Definitions and Terms

Developmental dyslexia can be categorised as a learning difficulty. It refers to the inability to develop adequate reading and/or spelling skills despite age-appropriate education, social-cultural opportunities and average or above average intelligence (Critchley, 1970). In contrast to those with acquired dyslexia, which manifests after neurological damage and represents the loss of adequate reading and/or spelling skills prior to the brain injury, children with developmental dyslexia fail to acquire adequate reading and/or spelling skills.

Since first described in 1896 by Morgan, various definitions have been used to describe developmental dyslexia (e.g., Hinshelwood, 1907). One of the more recent definitions has been given by Lyon, Shaywitz, and Shaywitz (2003): "Dyslexia is a specific learning disability that is neurobiological in origin. It is characterised by difficulties with accurate and/or fluent word recognition and poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unexpected in relation to other cognitive abilities and the provision of effective classroom instruction"(p. 2). This definition differs from previous definitions (e.g., Critchley, 1970 as described above) in that more specific information is included, for example the well-established phonological deficits associated with dyslexia. It also acknowledges the frequently observed fluency problem among dyslexic readers. The term dyslexia will be used throughout the thesis, as it is the most broadly used term in the literature to describe individuals with reading and spelling difficulties.

### **Prevalence** of Dyslexia

In the past decade prevalence rates for dyslexia have been reported as occurring in between 4 and 17.5% of the English-speaking population (Shaywitz, 1990). The English writing system in particular puts high demands on the beginning reader because of its irregularities. Thus prevalence rates for dyslexia in English are usually higher than those reported for other languages such as German, Italian, Spanish or Japanese (Grigorenko, 2001). The

Connecticut longitudinal study by Shaywitz et al. (1994) reported that 17.5% of the students assessed (kindergarten to Grade 5) were reading below age or ability levels. The 2005 US National Assessment of Educational Progress indicated that 27% of students in Grade 12 were not achieving the most basic reading levels, defined as a minimum level of understanding of what has been read (Shaywitz et al., 2008). In a study by Lewis, Hitch, and Walker (1994), 6.2% of nine- to ten-year-old British children in an unselected sample were found to have dyslexia. The variations in prevalence rates of dyslexia across studies are due to differences in the (1) strictness of selection criterion (whether the criterion chosen is 1, 1.5, 2 *SD* below average), (2) environment, (3) grade chosen, and (4) type of screening utilised (Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992). For instance, the Connecticut longitudinal study (Shaywitz et al., 1992) followed 414 children from kindergarten to Grade 5. The authors reported a prevalence rate of dyslexia of 5.6 % in first grade, 7% in third grade and 5.4% in fifth grade using a criterion of 1.5 *SD* below expected performance. These results suggest that dyslexia is not a stable condition.

With regard to screening tests, the use of standardised screening measures, such as the Dynamic Indicators of Basic Early Literacy Skills (DIBELS, Kame'enui, Simmons, Good, & Harn, 2000) also leads to an almost equal identification of males and females as opposed to classroom-based observations, which usually identify more boys than girls (3:1) as having dyslexia (Flynn & Rahbar, 1994; Shaywitz & Shaywitz, 2003; Shaywitz et al., 2008). Shaywitz et al. infer that boys are more likely to be referred for assessment due to disruptive classroom behaviour whereas girls are commonly quieter and go unnoticed.

### Symptoms, Developmental Course and Assessment of Dyslexia

The term dyslexia is derived from the Greek word 'dys' meaning difficult and 'lexia' meaning words. The cardinal symptom of dyslexia is the reading deficit, most prominent in a much slower reading rate, but also in a lower reading accuracy and comprehension in relation to a group norm. Typical reading errors are letter reversals such as reading "b" for "d", omissions of function words (e.g., "the", "our") and misreading of longer and/or unfamiliar words such as "place" for "palace". Additionally many dyslexic readers show

persistent spelling problems, in particular dysphonetic errors (phonetically unacceptable errors) such as writing "tetr" for "tent" (Snowling, 2000). Phonological deficits are most evident during the reading of nonwords (made-up words that do not have a meaning) as these can only be read through phonological decoding (Vanijzendoorn & Bus, 1994). Phonological decoding requires the application of grapheme-phoneme-correspondences (GPCs), which represent letter-sound-relationships, in order to read a word (Coltheart, Curtis, Atkins, & Haller, 1993). GPCs are commonly weakly developed in dyslexic readers (e.g., Snowling, 2000).

Less frequently investigated are the deficits in orthographic awareness among dyslexic readers. Orthographic awareness refers to the sensitivity to regular patterns of letters in a word (e.g., the combination "sud" is common in English, whereas "yxl" is not) and thus assists in the development of automatic and fluent word recognition (Vellutino & Fletcher, 2005). Dyslexic readers have also been found to have working memory deficits. Due to their slow reading style, working memory becomes overloaded, as it is limited in capacity (Cohen-Mimran & Sapir, 2007; Repovs & Baddeley, 2006; Schatschneider & Torgesen, 2004), which in turn impacts negatively on the development of fluent reading and sight vocabulary (Jeffries & Everatt, 2004; Ptok et al., 2007). In contrast, dyslexic individuals' other cognitive abilities such as thinking, reasoning, and listening comprehension are often within the normal range (Shaywitz et al., 2008). Without intervention, dyslexia persists into adulthood and over time poor and good readers tend to maintain their relative positions along the spectrum of reading disability. For many dyslexic readers the overt reading difficulty can often be overcome through intensive remediation and/or compensation strategies, whereas reading fluency and spelling often remain persistently poor (Shaywitz et al., 2008). The use of compensation strategies depends on the major problem area of the child, and often occurs as a result of continuous struggle when trying to read and/or spell. For example, a child with basic phonological problems can, to some extent, use a visual reading strategy by making use of semantic cues (meaning and similarities between words) and contextual cues (words that may fit the context) while reading to compensate for the phonological weakness. This strategy is very limited and

causes reading problems with many new, irregular, and less frequent words (Snowling, 2000).

The assessment of dyslexia is difficult due to its heterogenous nature and provides a challenge for teachers, researchers and educators (Lyon, 1995). Dyslexia is a clinical diagnosis and the minimum standard to assess dyslexia validly should include a reading test (accuracy, rate, and comprehension), spelling test, intelligence test, and phonological test (Marzola & Shepherd, 2005; Schulte-Koerne, Deimel, & Remschmidt, 2001). A variety of reliable and valid standardised tests exist to estimate a child's literacy skills. Over the past decades one of the most common diagnostic criteria for dyslexia in the research literature was and still is the ability-achievement discrepancy criterion (Meyer, 2000). Other criteria have been proposed in recent years and are discussed below.

With regard to the ability-achievement discrepancy a child performing below the 25<sup>th</sup> percentile or 1 to 2.5 *SD* below the age/grade level in relation to his/her expected performance is diagnosed with dyslexia. According to the discrepancy model, the expected performance is represented by the child's general cognitive ability, as assessed by an intelligence test. The IQ discrepancy criterion is based on the fact that generally IQ is positively correlated with reading skill in the population, which means that children with a higher IQ score tend to be better readers. A child is then classified as dyslexic if his/her reading is poor, but his/her IQ score is average or high. Subsequently children who have a low IQ score and are low achieving are generally classified as mentally challenged or backward readers, not as dyslexic readers.

However, the use of the discrepancy model has been challenged by many academics (e.g., Au & Lovegrove, 2006; Fletcher, Denton, & Francis, 2005). Some evidence suggests that backward readers (also called 'garden-variety dyslexic readers') and dyslexic readers show similar literacy deficits regardless of low or high IQ (Everatt, Weeks, & Brooks, 2007; Shaywitz et al., 2008) and both groups benefit to a similar extent from literacy interventions (Mathes & Denton, 2002; Snowling, 2000). One other disadvantage of the discrepancy model is the "wait to fail" approach. To obtain a discrepancy, children need to fall behind before they are identified (Mathes & Denton, 2002). These issues have led some researchers to propose more dynamic assessments, especially for earlier grades, to monitor progress frequently (Kame'enui et al., 2000). One approach that has generated considerable interest is called the response to intervention (RTI) (Vaughn & Fuchs, 2003). According to the RTI approach, identification of reading disability follows a response to intervention model, so that those who are failing to respond to early interventions are diagnosed as learning disabled regardless of their intelligence score. Interventions and outcome measures are conducted as early as kindergarten age and monitored on a regular basis. The advantage of this model is its dynamic, developmental and prevention approach (Snowling, 2000). Nevertheless, the implementation of this model places high demands on human resources to conduct frequent literacy tests and interventions. In addition, clinical criteria to classify interventions as successful still need to be developed and evaluated (Kavale, 2005).

## Co-morbidities of Dyslexia

The most commonly associated disorder which is co-morbid with dyslexia is attention deficit/hyperactivity disorder (ADHD). For children, estimates of co-morbidity range from 15-50% (Biederman et al., 1996, Shaywitz et al., 1994). ADHD is characterised by a lack of concentration, hyperactivity, short attention span, easy distraction and impulsiveness (DSM-IV, 1994). However, whether attentional problems in dyslexic readers are secondary or primary to the reading/spelling difficulties has not been satisfactorily answered. For example, a study by Pennington, Groisser, and Welsh (1993) compared three groups (one with ADHD, one with dyslexia, and one with both) on phonological tasks and executive tasks. They found that performance on executive tasks was mostly impaired in ADHD individuals. They further reported that the co-morbid group performed at the same level as the dyslexic-only group, with normal executive functioning and impaired phonological functioning. The authors concluded that the co-morbid group developed attention deficits as a secondary consequence of their learning difficulties. This is not surprising as effortful slow reading puts an enormous demand on attentional resources as the text is not read automatically (Shaywitz et al., 2008). In conclusion, both primary and secondary attentional problems have been associated with dyslexia.

Other less frequently investigated co-morbidities are oral language deficits (Demonet, Taylor, & Chaix, 2004). Sundheim and Voeller (2004) reported that delayed language acquisition including late speaking, mispronunciations, confusing words that sound alike, needing time to produce an oral response, was often observed in dyslexic children prior to their being diagnosed with learning difficulties. Shaywitz et al. (2008) added that some of those language deficits are still evident in the older reading-disabled child. Emotional co-morbidities have a higher incidence among students with learning difficulties than students without. These include antisocial and aggressive behaviour, low self-esteem, depression, and psychosomatic complaints such as stomach aches (Beitchman & Young, 1997; Kulkarni, Kalantre, Upadhye, Karande, & Ahuja, 2001; Sundheim & Voeller, 2004). These associations have been mainly considered as secondary symptoms, developing due to continuous failure in learning to read and write. For example, Sundheim and Voeller reported that somatic complaints were often used as a strategy to avoid going to school.

In summary, dyslexia is one of the most common learning difficulties in Englishspeaking societies and is characterised by a broad range of difficulties in reading (accuracy, comprehension, and rate), spelling, phonics, and memory. It has also become increasingly clear that individuals with dyslexia have a higher incidence of emotional and motivational problems, including low self-esteem, antisocial and aggressive behaviour, than those without. Various criteria have been proposed to diagnose dyslexia, but the discrepancyachievement criterion is one of the most widely used. Given that dyslexia is a heterogenous difficulty, comprehensive and dynamic assessment methods have been developed in recent years, allowing ongoing monitoring of a child's early literacy skills. The following chapter summarises developmental reading models and visual word recognition theories that are of particular importance within the scope of the thesis.

#### **Chapter 3: Models of Normal Reading Acquisition and Visual Word Recognition**

## Developmental Stage Models of Normal Reading

Our understanding of reading impairment is tightly coupled to our understanding of normal reading development. Various developmental models of normal reading development have been proposed. This section will discuss four models as representatives of the main models on reading development, namely the models by Marsh, Friedman, Welch, and Desberg (1981), Frith (1985), Stuart and Coltheart (1988) and Ehri (for a review see Ehri, 2005). The final paragraph will discuss Share's self-teaching theory that stresses the particular importance of phonology for successful reading acquisition in greater detail than the developmental stage models (e.g., Jorm & Share, 1983; Share, 1995). A shared fundamental aspect of the developmental stage models is that they describe the stages that a beginning reader goes through, with each stage representing the pre-requisite for the next stage. However, all theories include the possibility of entering the next stage before mastering the previous stage entirely and commonly refer to 'phases' instead of 'stages' of development. In this thesis, the term stage will be used to mean either stage or phase. Table 1 summarises the four models.

#### Table 1

| Proposed | Stages | of Normal | l Reading | Devel | opmeni |
|----------|--------|-----------|-----------|-------|--------|
|          |        |           |           |       |        |

| Marsh et al. (1981)      | Frith (1985)    | Stuart & Coltheart (1988) | Ehri (2002)           |
|--------------------------|-----------------|---------------------------|-----------------------|
| 1. Glance and Guess      | 1. Logographic  | 1. Partial Orthographic   | 1. Pre-alphabetic     |
| 2. Discrimination Net    | 2. Alphabetic   | 2. Complete Orthographic  | 2. Partial Alphabetic |
| Guessing                 |                 |                           |                       |
| 3. Sequential Decoding   | 3. Orthographic |                           | 3. Full Alphabetic    |
| 4. Hierarchical Decoding |                 |                           | 4. Consolidated       |
|                          |                 |                           | Alphabetic,           |
|                          |                 |                           | Automaticity          |

Marsh et al.'s model begins with the glance and guess stage. During this stage, the beginning reader is able to recognise a small set of words visually due to print exposure. Unfamiliar words cannot be read as phonological skills are not yet developed. Sometimes

the beginning reader guesses words within the context of a story, based purely on context and memory of semantically related words. The second stage is usually entered after the first year of reading instruction and involves the reading of words through graphemic features, in particular initial letters. Minimum graphemic cues are stored and remembered when trying to read a new word. The third stage involves the acquisition of grapheme-phonemecorrespondences (GPCs). The beginning reader learns how sounds in different words follow a general regular pattern and can decode unfamiliar words using the sounding-out strategy. Commonly the beginning reader is first able to distinguish broader units of sounds such as recognising that "headache" is made up of "head" and "ache". Later on, they realise that one word is often made up of syllables such as wa-ter and rhyme ability starts to develop. Further down the development the beginning reader realises that the word "pencil" can be segmented into p-e-n-c-i-l. Finally, the beginning readers will be able to analyse and manipulate sounds in that they will be able to say "football" without "ball", or say "pencil" backwards (Ptok et al., 2007). The last stage, hierarchical decoding, is reached around the middle years of childhood where skilled reading, which incorporates complex skills such as reading by analogy when encountering unknown words is mastered.

Frith has proposed three stages. During the first, logographic stage, the beginning reader uses visual and contextual features to recognise words. This is followed by the alphabetic stage, when decoding of graphemes to phonemes develops, and finally by the orthographic stage, when the reader recognises larger spelling patterns and is able to analyse words into orthographic units (especially morphemes) without phonological decoding. Ehri has generated three versions of her model. The latest version (2002) involves first a pre-alphabetic stage, with visual and contextual connections as a reading strategy, second a partial alphabetic stage, including connections between more salient letters and sounds, third a full alphabetic stage, during which complete connections between all the spelling graphemes and pronunciation phonemes evolve, and fourth a consolidated alphabetic stage, involving connections formed out of syllabic and morphemic units.

In contrast to the authors of the other three models, Stuart and Coltheart (1988) reject the existence of a visual and context-only stage. They argue that the beginning reader

needs to use phonological cues to be able to read. Their theory thus distinguishes only two important steps of development: one stage during which the beginning reader acquires letter-sound-correspondences sufficient enough to form partial representations of beginning and ending letters, and a later stage when vowel spellings provide the basis to form more complex representations of sight words in memory. This model, in contrast to the aforementioned models, also accounts for individual differences, in that it emphasises that the beginning reader will use any skill available when trying to read a new word.

One disadvantage of the four described reading acquisition models pointed out by Share (1999) is that none of them explains how phonology facilitates normal reading development. Jorm and Share (1983) proposed the self-teaching theory, which explicitly explains how phonological recoding facilitates normal reading development. Phonological recoding serves as a self-teaching function and is the primary drive for the development of fluent printed word recognition during beginning reading. Thus the development of fast word recognition is primarily a 'saying' not 'seeing' process. The 'seeing' process refers to the visual attention hypothesis, which proposes that visual attention to the word is the important factor for the development of orthographic representations. The self-teaching mechanism is regarded as particularly important when a child encounters novel words of low frequency (low print exposure) since it provides the opportunity to generate candidates of pronunciations for a novel word by applying recoding. In contrast, high-frequency words are recognised quickly by sight with no need, or minimal need, for self-teaching phonology. The model further proposes that self-teaching starts at an early age, even before graphemephoneme correspondences have been established, since letter-sound knowledge and a minimum of phonological sensitivity are sufficient abilities to trigger the self-teaching mechanism. Through increasing exposure to print, accumulating phonological and orthographic knowledge and successful decoding, adequate orthographic representations will develop. The final outcome of these processes is the ability to recognise a large number of words by sight. The models described provide an understanding of the general broad development of reading. Visual word recognition theories focus on a small detail of this

development and try to answer the question: How is a written word recognised and read aloud?

### Visual Word Recognition Theories

Visual word recognition is the foundation of efficient reading. The recognition process involves accessing information stored in memory to produce an oral response (Snowling & Hulme, 2005). Over the past decades a vast number of models seeking to explain visual word recognition have been developed. The two major theoretical influences will be discussed in this section, namely dual-route models and connectionist models. Both theoretical approaches contributed to our understanding of normal word reading and deficit word reading in acquired and developmental dyslexia.

### **Dual-Route Models**

During the 1970s and 1980s a series of dual- and triple-route models of normal adult word reading were proposed (Coltheart, 1978; Morton, 1979; Shallice, Warrington, & McCarthy, 1983; Temple, 1985). Figure 1 shows a typical dual-route model.

In early dual-route models of reading aloud two processes or routes were proposed, a lexical and a phonological/non-lexical route, with the two routes engaging in the processing in a non-overlapping fashion. The lexical route allows the reader to access and retrieve a word from visual memory, which contains a large amount of sight vocabulary. The cognitive process thus depends entirely on the visual analyses of the word and its association in memory. Depending on the model, the system for the visual analyses of a word is referred to as visual word form system (Shallice et al., 1983), input logogens (Morton, 1979), or word detectors (Temple, 1985, 1997). Subsequent to the visual word analyses, the semantic system is activated which provides information about the word's meaning. Following this process, the phonological representation or pronunciation of the word will be activated and either read aloud or held in a response buffer. Readers use this route primarily for familiar, sight, and irregular words, which have an entry in the visual memory.



Figure 1. A dual-route model of reading. Adapted from Temple (1997, p.179).

A second distinct route to the lexical route is the phonological/non-lexical route through which a word is recognised and read by being broken down into its components via the application of GPCs (Coltheart, 1978). GPCs can be simple or complex, allowing the skilled reader to read nonwords correctly. The phonological route is further assumed to require a longer processing time than the lexical route, which is supported by the experimental finding that readers need more time to read nonwords than real words (Snowling & Hulme, 2005). A third route, called the direct route, was first introduced by Schwartz, Saffran, and Marin (1980) who reported the case of a patient with dementia who could read irregular words correctly despite no longer understanding their meanings. The authors concluded that the patient read each word via a direct access from the visual word analysis to its phonological representation, bypassing the semantic system. Thus the direct route appears to be a lexical route, but without involving the semantic system to read the word. Coltheart, Rastle, Perry, Langdon, and Ziegler, (2001; see also Coltheart et al., 1993) took the early route models a step further and utilised computational modelling for the study of word recognition. The dual-route cascaded model (DRC) by Coltheart was inspired by the computational modelling technique used by connectionist advocates (e.g., Seidenberg & McClelland, 1989). The basis of computational modelling is that the cognitive task in question is executed by a computer program in the way the modeller thinks the human being performs the task. In a similar manner to the previous versions of the dual-route model (Coltheart, 1978; Coltheart, 1980), the computational model includes a lexical and a nonlexical route via which a word can be read. One important aspect that will be emphasised here is the claim that the non-lexical route does not start operating until ten cycles involving feedback and feed-forward mechanisms of the lexical route have been completed. This way conflicting input from the non-lexical and lexical routes when attempting to read a word is minimised. For example, if the non-lexical route starts operating too early, the program would have difficulty reading irregular words as conflicting input from the non-lexical route would produce incorrect results. This is the so-called regularity effect in which irregular words are read in a regular way, following GPC rules.

Phenomena that are explained by the DRC model include the frequency effect, which demonstrates that readers need longer to read low-frequency words than highfrequency words. Similarly, regular words are read faster than irregular words. Irregular words are in particular more time-consuming because the two routes produce conflicting input, even though a time lapse between the onset of the lexical and the non-lexical route is assumed as aforementioned. For example, research has shown that when readers are asked to speed up reading irregular words, regularity effects occur more frequently (Coltheart, 2005). According to Seidenberg (2005), dual-route models fail to explain partial regularities of a language. The assumption that regular words are learned by rules and exceptions are memorised ignores the fact that some irregular words share partial similarities with regular words. As an example the learning of the pronunciation of words like "pant" and "pine" would have no impact on learning "pint" under a dual-route model.

#### **Connectionist Models**

Connectionist models emerged historically as alternative models to dual-route models. Seidenberg (2005) and Coltheart (2005) state that connectionist models differ from dual-route models in the following ways (for a review see Plaut, 2005):

- Single processing system: Instead of proposing distinct parallel routes, connectionist
  models describe a single processing system as fundamental for word recognition. This
  implies that the pathways of a single processing system (orthographic and
  phonological) work together and the contribution of each pathway depend on the
  contribution of the other.
- 2. *Network structure*: Networks of neurons, which are implemented in the brain, constitute the physical hardware of the operating word recognition system.
- 3. *No word-specific lexical entries*: These networks do not contain word-specific representations, but code the visual and phonic features of a letter and phoneme respectively.
- 4. *Learning*: The neural network is developed and modified through learning experience, emphasising the developmental aspect to resemble human learning.

One of the first connectionist models was introduced by Glushko (1979). The model claims a single process through which nonwords and irregular words are read aloud. A modification of Glushko's model was presented by Seidenberg and McClelland (1989), and is commonly referred to as the triangle model (see Figure 2). It includes sets of hidden units (represented by the smaller ovals in Figure 2), which connect the three major units (layers; represented by the larger ovals in Figure 2) of the network, which are (1) the orthographic unit (codes the visual properties), (2) the phonological unit (codes the phonological properties) and (3) the semantic unit (codes meanings of words). The hidden units represent more complex mappings of orthographical and phonological units. Another element is the connection weights (represented by the arrows in Figure 2) between the three units via the hidden units, which modulate the flow of activation. Through learning experience, based on a propagation algorithm, these weights are adjusted and refined over and over and further vary with different word properties (e.g., word frequency, spelling-sound consistencies).

When operating, the system creates an output by finding the appropriate set of weights. In doing so the input activates the corresponding major units (layers) and lets activation pass to the output units via connections between them. The hidden units function as information and feedback mechanisms between the major orthographic, phonological, and semantic units.



1

*Figure 2*. Connectionist model by Seidenberg and McClelland (1989). Adapted from Seidenberg (2005, p.239).

Tests conducted on the model showed that it was able to read words accurately, with the common finding of regular words and high-frequency words being read faster. The model was also able to read nonwords, on which it was not trained. However, criticism emerged as the model failed to reach the same level of performance as normal adult readers during nonword reading (Besner, Twilley, McCann, & Seergobin, 1990; Coltheart & Leahy, 1992). These limitations led to a revised model which is better at reading nonwords (Harm & Seidenberg, 1999; McClelland & Plaut, 1993) and Seidenberg (2005) makes it clear that "the nonword generalisation problem was soon traced to the imprecise way that phonological information was represented in the (older) model ... thus the nonword problem 'falsified' the original model but not the theory it approximated" (p. 240). One major limitation of the current connectionist models is the relatively small range of vocabulary they can handle and the range of empirical issues explained through the models is lower compared to the dual-route models (Plaut, 2005). However, the two theories are not as opposite as commonly believed as the authors Seidenberg and McCellland (1989) themselves report "ours is a dual-route model" (p. 559). In summary all models have their strengths and weaknesses and have contributed to a great extent to our understanding of basic word recognition, and at this stage there is no clear winner (Lupker, 2005).

#### Implications for Dyslexia

The dual-route theories and connectionist models have implications for the symptoms associated with dyslexia. According to Coltheart et al. (1993) two subtypes of dyslexia are implicated. One is the phonological type, which shows an impaired non-lexical route resulting in phonological difficulties such as poor nonword reading. The other is the surface type, which shows primarily irregular word reading problems due to a deficit of the lexical route. Degradations of the lexical or non-lexical routes in the computational models have confirmed these subtypes. A third mixed type, experiencing difficulties with both routes, was further proposed (Coltheart et al., 1993; Castles & Coltheart, 1993; Coltheart et al., 2001). Research on the clinical validity of these subtypes has produced conflicting results, with some supporting the existence of certain subtypes of dyslexia and others not (e.g., Griffiths & Snowling, 2002; Manis, Seidenberg, & Doi, 1999a). In particular the surface dyslexia type has not been consistently found in dyslexia (e.g., Stanovich, Siegel, & Gottardo, 1997).

Although the connectionist models have primarily focussed on the modelling of normal reading and acquired dyslexia following neurological injury, some implications for developmental dyslexia have been proposed (Harm, McCandliss, & Seidenberg, 2003). Evidence strongly confirms the importance of phonics for dyslexic reading (Seidenberg, 2005) following degradation of phonological connections in the computational model. In another investigation Harm and Seidenberg (1999) tested different instructional approaches for reading (only semantics vs. semantics and phonics), with the combined version resulting in faster and more efficient reading acquisition. This highlights the possibility that some dyslexic readers may be "instructional dyslexic readers", who have not received any or sufficient phonological instruction in reading.

In summary, developmental models of normal reading have demonstrated the importance of phonological skill development for early and successful reading, providing a

framework for the next chapter on causes of dyslexia. What happens when this development of phonological skills is deficient? The phonological hypothesis of dyslexia is designed to answer this question. Visual word recognition theories, mainly dual-route and connectionist approaches, have greatly contributed to our understanding of how a word is recognised and read aloud by the skilled reader, and underline the importance of phonological and orthographic skills for efficient word recognition. The next chapter reviews theories on the causes of dyslexia and discusses the empirical findings on each of the theories. Two major theoretical influences have led the research in this field, namely theories that attribute dyslexia to basic visual and auditory function deficits and those theories which claim a linguistic phonological processing deficit as the underlying mechanism of dyslexia.

### **Chapter 4: Current Understanding of Dyslexia**

### Introduction: Current Understanding of Dyslexia

Investigation of the causes of dyslexia has been a major research interest and has attracted researchers from various fields such as neurologists, psychologists, medical researchers, ophthalmologists, speech pathologists and educational scientists. The most direct approach to investigating children with dyslexia is the linguistic approach (or high-level processing approach). Although the linguistic, phonological approach is one that follows a direct examination of the disability presented, it does carry the disadvantage that whatever linguistic disability may be demonstrated, it could in principal be secondary to a more basic functional impairment.

Until the 1960s dyslexia was understood as a visual condition, and was later conceptualised under the magnocelluar deficit theory (Lehmkuhle, Garzia, Turner, Hash, & Baro, 1993; Livingstone, Rosen, Drislane, & Galaburda, 1991; May, Lovegrove, Martin, & Nelson, 1991; Stein, 2001). In the following years basic auditory dysfunction and temporal deficits have been claimed to be responsible for higher-order deficits such as phonological weaknesses (Farmer & Klein, 1995) and basic senso-motor dysfunctions were related to the reading/spelling difficulties in dyslexia under the cerebellar theory (Fawcett, Nicolson, & Dean 1996). Another theory which has gained some interest is the balance theory by Bakker (2006) which attributes dyslexia to a disruption of connectivity between the left and right hemisphere functions.

Around the same time another string of theories developed, which considered dyslexia to be a linguistic high-level phonological processing problem consequently leading to problems in reading and spelling (Shaywitz et al., 2008; Snowling, 1995). Regardless of the cause of dyslexia, phonological problems have been most evident in dyslexic readers. Due to the fact that each of these single-deficit approaches (non-linguistic and linguistic) is supported by a large amount of empirical evidence, current research points to a multidimensional disorder. One prominent theory is the double-deficit hypothesis posited by Wolf, Bowers, and Biddle (2000; see also Wolf & Bowers, 2000) which integrates the possibility of dual-deficits including phonological and naming speed deficits. Importantly it should be noted that in any individual case of dyslexia it is often very difficult to determine the individual cause (Hallahan, Llyod, Kauffman, Weiss, & Martinez, 2005). The following section summarises each of the major theoretical influences and the empirical evidence supporting each.

### The Magnocellular, Basic Auditory and Temporal Processing Theories

The *magnocellular theory* assumes a weakness in the fast-processing visual pathway, namely the magnocellular or transient system (M-system), (for a review see Stein, Talcott, & Walsh, 2000a). Together with the parvocellular, or sustained system (P-system) the two visual pathways provide the basis for efficient eye control and perception during reading. The M-system is responsible for eye movement control (saccades) and location whereas the P-system extracts the details of letters during a fixation. During one saccade, the M-system appears to inhibit the P-system, to avoid overlapping of the previous fixation with the next fixation (Breitmeyer & Ganz, 1976). Thus efficient reading relies on the time accurate interaction of these two systems, which, if failing can lead to "swimming of letters and words" (for a review see Stein, 2001;Stein & Fowler, 1981; Stein et al., 2000a). Subsequently, this phenomenon would occur most severely during the reading of connected text than when reading isolated words (Lovegrove, 1993; Vellutino, 1979).

M-system dysfunctions in dyslexic readers have been implicated in various studies including impaired coherent motion ability which describes the ability to judge whether small dots on the screen are moving in the same direction, impaired accurate localisation of small dots on a screen, and difficulty recognising several visual items presented simultaneously (Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Hansen, Stein, Orde, Winter, & Talcott, 2001; May et al., 1991; Lehmkuhle et al., 1993; Livingstone et al., 1991). Furthermore an increasing number of studies have shown that many dyslexic readers perform significantly less well than controls on tasks of rapid, temporal, visual information processing, including tests of visible persistence, flicker sensitivity, and contrast sensitivity, and on visual order tasks, as opposed to tests of static displays, aimed to stimulate the P- system (Farmer & Klein, 1995; Greatrex & Drasdo, 1995; Lovegrove, Garzia, & Nicholson, 1990; Lovegrove, Martin, & Slaghuis, 1986; May, Williams, & Dunlap, 1988; Romani et al., 2001). In contrast, Chase and Jenner (1993) showed that dyslexic readers had no difficulty in a colour sensitivity task designed to stimulate the P-system selectively.

Studies employing orthoptic measures have tested the specific ocular motor control deficits experienced by dyslexic compared to normal readers. Stein, Richardson, and Fowler (2000b) review several findings showing inferior binocular vergence control and unstable fixation in dyslexic samples, which causes the phenomenon of words "swimming" around the page (see also Adler-Grinberg & Stark, 1978; Black, Collins, DeRoach, & Zubrick, 1984; Olson, Kliegl, & Davidson, 1983). Taken together these findings support the M-system hypothesis. However, most of the positive findings are from the 1980s and early 1990s and recent studies have sometimes failed to find visual abnormalities in dyslexic readers (Kronbichler, Hutzler, & Wimmer, 2002; Schulte-Koerne, Bartling, Deimel, & Remschmidt, 2004a; Williams, Stuart, Castles, & McAnally, 2003).

In contrast to most other studies, which have used non-linguistic tasks to stimulate the M-system, a study by Hutzler, Kronbichler, Jacobs, and Wimmer (2006) compared dyslexic readers' eye movement patterns on a meaningful pseudoword-reading task and a meaningless letter-string task. The letter-string task was designed to stimulate the M-system based on the theoretical idea that a weak M-function impacts on the accurate perception of letter strings (Stein & Talcott, 1999). Whereas the perceptual and oculomotor demands of the letter-string and pseudoword task were considered constant, the pseudoword task had an explicit linguistic phonemic component. Group differences emerged only during pseudoword reading, with dyslexic readers showing significantly longer and more fixations than controls. The authors suggested that their results provide direct evidence that a weakness in the M-system does not drastically impact on visual perception and oculomotor control, as the perceptual letter-string task did not result in different eye patterns in the dyslexic group. Hutzler et al.'s results highlight the ongoing debate about whether the Msystem deficits are actually related to real word reading. Talcott et al. (1998) reported a high correlation between M-system function and nonword reading. However, in a study by Sperling, Lu, Manis, and Seidenberg (2003) an M-system deficit was not correlated with phonological problems in the dyslexic sample, but with orthographic problems. In conclusion, it is likely that at least a small percentage of dyslexic readers suffer from a visual deficit and that it may be that the interaction between P- and M-systems is deficient in dyslexic readers, not the M-system in isolation (Stein, 2001). Future studies are needed to test this hypothesis. Chapter 5 provides a review of the neural correlates of the proposed M-system dysfunction.

Weaknesses in the auditory domain, including a deficient auditory temporal processing system have also been reported. Tallal (1980), leading the research work on auditory deficits and dyslexia, proposed that dyslexia involves a low-level auditory processing deficit that impairs the ability to perceive rapidly varying sounds and is thus the core deficit underling phonological awareness weakness in dyslexic readers. The initial study by Tallal tested the error rate of dyslexic readers compared to controls in a tonediscrimination paradigm with varying inter-stimulus-intervals (ISIs). Dyslexic readers did not differ in their overall discrimination ability during the practice trials; however, when tone stimuli were presented in a rapid manner (ISIs below 350 ms), dyslexic readers showed a significantly higher error rate than controls. Furthermore, error rate correlated significantly with performance on a nonword test (r=0.81), which led Tallal to propose a fundamental auditory temporal deficit as a cause for the phonological problems of dyslexic readers. Subsequent studies provided further evidence: When stimuli are presented rapidly, dyslexic readers have been found to produce longer reaction times to pure tones (De Weirdt, 1988; McAnally & Stein, 1996) and to need longer reaction times to discriminate various tonalpatterns (for a review see Farmer & Klein, 1995). Temporal order judgment designs (TOJ) have also been employed in some studies and Kinsbourne, Rufo, Gamzu, Palmer, and Berliner (1991) reported that dyslexic readers needed longer ISIs to determine the order of two aurally presented stimuli compared to controls. Threshold designs are another popular way to investigate basic auditory functions, by testing tone discrimination sensitivity. Dyslexic readers have shown to be significantly worse at detecting differences between a
pure and modulated tone compared to controls, indicating a higher threshold when trying to differentiate two tones (McAnally & Stein, 1996; Witton et al., 1998).

Ramus (2001; 2003) reviewed studies in which no evidence for an auditory temporal deficit has been found (see also Chiappe, Stringer, Siegel, & Stanovich, 2002) and concluded that overall the evidence is equivocal. For instance, one study by Watson (1992) compared dyslexic readers' discrimination ability for tones and linguistic stimuli (sounds: ta, ka) and found diminished performance only for the linguistic stimuli. Studdert-Kennedy and Mody (1995) criticised the unclear conceptualisation of "temporal processing" and "processing of temporal frequencies" within Tallal's concept. This can potentially cause confusion for the investigation of temporal deficits in dyslexia, as the experimental manipulation requiring the "processing of temporal frequencies" is not necessarily sufficient to reflect a cognitive "temporal processing" deficit.

Very little research has been done to investigate the capacity of dyslexic readers to integrate information between the visual and auditory modality. It could be hypothesised that if the M-system and auditory deficits of dyslexic readers are additive, a multi-modal task synthesis would result in larger altered processing than single-modality designs. Although the majority of studies have investigated either the visual or auditory modality, in one exceptional study by Hairston, Burdette, Flowers, Wood, and Wallace (2005), a visual temporal order judgement task (TOJ) was combined with auditory cues. The TOJ task involved the judgement of the order of two white circles presented on the screen. The first auditory tone occurred at the same time as the first white circle and the second tone was delayed (0 to 350 ms) relative to the onset of the second circle. Dyslexic readers and controls showed differing performance in all conditions, with dyslexic readers showing a significantly larger time window for integrating multi-sensory information. Specifically, compared to controls, dyslexic readers showed increased response accuracy (more correct responses) when auditory cues were presented regardless of whether or not there was a delay to the visual stimulus. The authors interpreted the results as an indication that dyslexic readers show an altered cross-modal temporal processing and have a larger time window over which auditory cues can influence visual discrimination. With regard to linguistic

stimuli, this larger window may cause more interference and result in inappropriate mapping of letters and sounds.

The relationship of auditory temporal deficits and reading/spelling difficulties has rarely been investigated. However, a few studies have reported correlational relations between auditory and temporal weaknesses and phonological skill such as phoneme discrimination (Witton et al., 1998; Merzenich et al., 1996). In conclusion, although researchers acknowledge the existence of basic visual and auditory subtle deficits, there is still an ongoing debate whether these basic functional deficits are causal to the reading/spelling difficulties of dyslexia (e.g., Share, Jorm, MacLean , & Matthews, 2002; Vellutino, 2005).

## The Cerebellar Theory

The cerebellar deficit theory by Nicolson and Fawcett (1990; see also Nicolson et al., 1999; Nicolson, Fawcett, & Dean, 2001) gives an explanatory framework which accounts for phonological deficits as well as other related difficulties in dyslexia. The hypothesis is that a cerebellar weakness is the reason for problems with developing automaticity in language production and reception, which then leads to phonological problems. These, as a consequence, impact on higher-level processes such as reading and spelling. In general, the cerebellum is specialised for optimising motor performance and receives input from all sensory and motor centres. Accumulating evidence suggests that the cerebellum is also involved in non-motor functions, for example verbal working memory and reading (Fulbright et al., 1999) in disorders such as autism, schizophrenia, and dyslexia (for a review see Timmann & Daum, 2007). The direct impact of cerebellar dysfunction is the presence of motor skill dysfunctions which have been observed in a subgroup of dyslexic readers (Fawcett et al., 1996). For example, disruption of the reading process, has been observed in children with cerebellar tumours. The cerebellum receives input from left temporal-parietal areas of the brain associated with language processing, and so a cerebellar tumour disrupts this interconnected processing of language (Riva & Giorgi, 2000). Further supporting evidence has come from imaging studies showing decreased activation of the cerebellum

during motor learning in dyslexic readers (Nicolson et al., 1999; Rae et al., 1998). Stein (2001) linked the cerebellum under-function with the magnocellular dysfunction as it receives large input from magnocellular cells and plays a crucial role for the calibration and accurate timing of eye movements.

Two critical aspects about the cerebellar theory are highlighted here: First, cerebellar signs are not always reported in dyslexic readers and are often reported only for those showing attention deficits as well, raising the question of how important cerebellar dysfunction is to dyslexic symptoms per se (Demonet et al., 2004; Reynolds et al., 2003). Second, almost every task, motor and non-motor, involves activation of the cerebellum to some extent, as the cerebellum receives input from a variety of regions. Thus, it is very difficult to determine if an activation of the cerebellum is specific to the cognitive process under question (Timmann & Daum, 2007). Fawcett and Nicolson's assumption of the cerebellum as a major causal factor of the difficulties of dyslexia led to the development of a motor-exercise based intervention, called the Dore program (Dore & Rutherford, 2001), which aims to strengthen cerebellar function, speed and automaticity. The efficacy of this intervention for dyslexia is discussed in Chapter 6.

## The Balance Theory

The balance model by Bakker (for a review see Bakker, 2006) is based on the assumption that normal reading development includes a shift from right to left specific hemisphere processing at some stage of reading acquisition. As a study by Licht, Bakker, Kok, and Bouma (1988) showed, electrophysiological activity elicited by flashing words resulted mainly in right hemisphere activity at kindergarten age and left hemisphere activation at primary school age (Turkeltaub, Gareau, Flowers, Zeffiro, & Eden, 2003). Some children may not be able to make that shift and thus continue to process linguistic material in the right hemisphere. These are the so called P-type (P= Perceptual) dyslexic readers or "spellers" (slow but accurate). For other children left hemisphere processing may start too early, resulting in a so-called L-type (L= Linguistic) dyslexic profile or "guessers" (fast but many errors). More recently M-types, representing mixed profiles of L- and P-types have been added to the model.

To overcome the dyslexic symptoms of the P-type and L-type, Bakker argued that hemisphere specific stimulation (HSS) of the underactivated hemisphere would remodel the brain's activation patterns and lead to improvements in reading. The results of studies on HSS are discussed in Chapter 6. Whereas the HSS has been investigated in various studies, the theoretical assumptions of the model have not been tested in many studies and no independent research has been conducted to validate the proposed subtypes. For example, Hynd (1992) in his comment on the balance model, pointed out that Bakker neither specifies when the shift from right to left processing should occur nor what causes "imbalance" or "balance". Moreover, the proposed involvement of the right hemisphere during early reading is not conclusive, whereas left lateralisation of language functions in skilled reading has been consistently confirmed by empirical evidence. Finally, the conceptualisation of the left and right hemisphere under-function is very broad, and lacks details specifically concerning where and which brain functions/regions would be affected.

## The Phonological Theories

One of the major challenges and crucial steps for the beginning reader is to map written letters (graphemes) onto the elemental sounds of the spoken language (phonemes). Shaywitz et al. (2008) pointed out that this letter-sound mapping is a difficult concept to grasp, as a child who hears and says the word "bat", would not necessarily be aware that this word contains the three phonemes /b/, /æ/, and /t/. The ability to recognise, identify, and manipulate syllables and phonemes is referred to as phonological awareness, and research investigating phonological awareness in dyslexic readers has consistently shown the existence of a phonological deficit for dyslexic readers of average or above average intelligence (Ellis, 1989; Snow, Burns, & Griffin, 1998; Wagner & Torgesen, 1987). Further, the strength of phonological awareness as a predictor of reading acquisition, with those having weak phonological awareness experiencing difficulty in learning to read, has been well documented (e.g., Hatcher, Hulme, & Ellis, 1994). Measures of phonological

awareness reliably discriminate between good and poor readers (e.g., Goswami & Bryant, 1990).

Interventions including phonological awareness intervention have been found to be beneficial for beginning readers in general across different languages (e.g., Danish: Lundberg, Frost, & Peterson, 1988; German: Schneider, Roth, & Ennemoser, 2000; English: Torgesen et al., 1999), and also improve reading in dyslexic readers (Alexander, Andersen, Heilman, Voeller, & Torgesen, 1991; Foorman et al., 1998). Studies with adult dyslexic readers have shown robust evidence for the persistence of the phonological deficit. Adult dyslexic readers showed significantly worse phonological awareness even compared to younger readers with similar reading skill (Bruck, 1992; Schulte-Koerne, Deimel, & Remschmidt, 1997). Moreover, compensated dyslexic readers, who have achieved a normal reading level after intervention, still demonstrated weaknesses in phonological awareness (e.g., Pennington, Van Orden, Smith, Green, & Haith, 1990).

Experimental studies that have investigated higher-level processing problems have also found dyslexic readers to be slower at different experimental linguistic tasks in the auditory and visual modality compared to controls (Barnea, Lamm, Epstein, & Pratt, 1994; Farmer & Klein, 1993; Snowling, 1995). Many dyslexic readers have problems with verbal short-term memory (Jorm, 1983), with repeating multi-syllabic words (Miles & Miles, 1990), with tasks involving phonemic segmentation (Bradley & Bryant, 1978), and when asked to generate and discriminate rhymes (Snowling, Stackhouse, & Rack, 1986). Additionally, research has found a nonword reading deficit in dyslexic readers (for a review see Rack, Snowling, & Olson, 1992). Baddeley, Ellis, Miles, and Lewis (1982) presented dyslexic readers and controls with single-syllable words and nonwords. The groups performed similarly on words, but the dyslexic readers made significantly more errors on nonwords than did controls. Rack (1985) demonstrated that dyslexic readers performed poorly on a visual cue task, when the cue and the target word rhymed (cue: fruit, target: shoot), compared to when the cue and the target were visually-orthographically similar (cue: boat, target: shoot). Rack suggested that dyslexic readers rely on a more visual reading strategy than phonological decoding to compensate for their phonological weaknesses.

Taken together, these results support the hypothesis that dyslexic readers suffer from a specific deficit in the phonological language domain that leads to problems in reading and spelling. Most researchers propose that no deficits should be evident when an individual is processing non-linguistic stimuli and suggest that the reading problems are probably attributable to this phonological deficit (Miles & Miles, 1990; Stanovich, 1988a, 1988b; Vellutino, 2005). Nicolson and Fawcett (1994) tested the hypothesis that the phonological deficit would not be evident during non-linguistic tasks. A variety of tasks were used, gradually changing from non-linguistic to linguistic tasks. The finding was that the more linguistic and complex the task, the more profound the deficits of the dyslexic group, who showed significantly worse performance on lexical and phonological tasks but similar performance to controls on non-linguistic tasks. Given the conclusive evidence indicating a phonological deficit in dyslexic readers, we can now ask how this deficit impacts on reading and spelling. Research on normal reading development (see Chapter 3) implicates the importance of phonological awareness and subsequent learning of graphemephoneme correspondences (GPCs) for successful reading. Thus, a failure will drastically impact on the quality of phonological representations. Vellutino (2005) suggested that initially weak phonological awareness impacts on the storage and retrieval of words and the bonding between sounds and letters. As a consequence, inefficient and poorly developed phonological representations of words will result, leading to problems in word identification. Subsequently, fluency will be impaired. With regard to reading comprehension, Vellutino proposed that effortful reading of the phonologically deficient reader has a drastic impact on working memory, causing overload, and thus leaving few resources for reading comprehension. Ptok et al. (2007) highlighted the relationship between phonological awareness and spelling. If the beginning reader is able to detect the three different sounds in 'cat', he/she will be more likely to be able to spell the word correctly.

One critical aspect of the phonological theory was noted by Morais (1991) who reported that complex levels of phonological awareness (including GPC rules) usually develop during the first year of reading instruction, raising the possibility that complex phonological awareness is a consequence rather than a causal factor for reading development. Stanovich (1992) emphasised the reciprocal relationship of phonological skill and reading in that a certain threshold phonological awareness may be necessary for reading development. However, as soon as reading instruction starts, this impacts on the development of phonological awareness. This aspect has been neglected in the one-way causal phonological hypothesis described in this section.

### The Double-deficit Theory and other Multidimensional Approaches

Each of the theoretical approaches described above is supported by a large amount of empirical evidence indicating that multiple underlying deficits are associated with dyslexia. One prominent theory is the double-deficit hypothesis proposed by Wolf et al. (2000; see also Wolf & Bowers, 2000; for a review see Vukovic & Siegel, 2006). This theory postulates that some individuals with reading disability have a deficit in phonological awareness whereas others have a rapid naming deficit and a third group has both phonological coding deficits and rapid naming deficits (Voeller, 2004). This combined (double-deficit) type is most severe as phonological and naming deficits are additive. The double-deficit hypothesis was supported by empirical evidence showing that children with the double deficit were more impaired than those without it, and rapid naming appeared to be correlated with speed/fluent related task responses, whereas phonological awareness was more involved in decoding and related phonological processing (Compton, DeFries, & Olson, 2001).

To understand the rapid naming subtype a brief summary of the rapid naming concept is necessary. According to Wolf et al. (2000) there is no single definition commonly used in the literature and the authors suggested the following definition: "naming speed is conceptualised as a complex ensemble of attentional, perceptual, conceptual, memory, phonologic, motoric, semantic subprocesses that places heavy emphasis on precise timing requirements within each component and across all components" (p. 395). Measures of rapid naming include naming of a series of randomly presented objects, numbers, letters or colours in a speeded way. The time taken to name them is the score, and 1 *SD* below the mean has often been used as an indicator for a naming speed deficit. According to Wolf (1999), rapid naming deficits impact on reading difficulties through a slowing of perceptual, motoric and reading fluency.

Previous research on naming deficits and studies on the validity of the proposed subtypes confirmed the occurrence of naming deficits in dyslexic readers (Denckla & Rudel, 1976; Fawcett & Nicolson, 1994) and some studies have identified all three subtypes of the double-deficit theory (e.g., King, Giess, & Lombardino, 2007). However, in other studies, a rapid naming only type was not revealed or limited to a very small percentage (Badian, 1997; Morris et al., 1998; Pennington, Cardoso-Martins, Green, & Lefly, 2001). Research investigating the independent contribution of rapid naming deficits and phonological deficits, has led to inconclusive results. According to the theory, rapid naming and phonological skill should ideally be additive, non-correlated factors for the prediction of reading ability. Manis et al. (1999a) reported that rapid naming and phonological awareness assessed in Grade 1 contributed independent variance to scores on reading measures in Grade 2. Other supporting evidence came from Bowers and Swanson (1991), Neuhaus and Swank (2002) and Hammill, Mather, Allen, and Roberts (2002). Pennington et al. (2001) demonstrated that whereas rapid naming contributed to the prediction of oral reading rate only, phonological awareness was related to word attack, spelling and comprehension. However, the overall contribution of rapid naming was modest compared to that of phonological awareness. It has also been argued that the predictive strength of phonological awareness is maintained until adulthood, as opposed to rapid naming which loses its predictive ability after Grade 2 (Torgesen et al., 1997b).

Recently some researchers have argued that naming deficits can be conceptualised as a subtype of phonological deficits and thus can be integrated in the phonological hypothesis. Some studies have indicated that phonological awareness and rapid naming are not perfectly independent, and correlate modestly with each other (r= 0.30, Hamill et al. 2002; r= 0.28, Wolf, 1999). For instance, in their validation of the three subtypes Schatschneider, Carlson, Francis, Foorman, and Fletcher (2002) found that (1) rapid naming and phonological awareness correlated and accounted for 24% of shared variance for word recognition, as opposed to 13% alone, showing that their shared variance was at least as predictive as each one alone, (2) the double-deficit group had larger phonological awareness deficits than the phonological-only group, and (3) the larger the phonological awareness deficits, the greater the reading difficulties. The authors raised the possibility that the more severe reading impairment of the double-deficit type may result from the larger phonological awareness deficits and not from the combined influence of rapid naming and phonological awareness deficits. This has serious implications for the double-deficit theory and highlights the need for studies with similar levels of phonological awareness deficits in phonological-only and double-deficit groups (see also Vellutino, 2005). Based on the research findings it is difficult to draw any conclusion, highlighting the need for more studies on the importance or otherwise of rapid naming deficits in dyslexia (Schatschneider & Torgesen, 2004).

The last section of this chapter is dedicated to a multidimensional model of dyslexia, which provides an explanatory framework to guide future research investigations. Although multi-dimensional models have been proposed by various researchers (Badian, 1997; Frith, 1997; Pennington, 2006; Vellutino, Scanlon, & Tanzman, 1991), a full discussion of all proposed multidimensional models is outside the scope of the current thesis. One of these models was proposed by Pennington (2006) and underpins the importance of examining dyslexia on multiple levels and with potentially multiple causes (Figure 3).

Pennington's model includes four levels of analysis: etiologic, neural, cognitive and symptom/behaviour. As can be seen in Figure 3, the highest level, the etiological level, is fundamental for each learning disorder, with multiple and interactive environmental and genetic risk and preventive factors impacting on neural development. On a cognitive level, the neural developments alter cognitive functions, which become overt in the behavioural symptoms. The bi-directional arrows between the factors on each level as shown in Figure 3 indicate these interactive impacts. For instance, on an etiological level the genetic (G1, G2, G3) and environmental (E1, E2) risk and protective factors most likely interact with each other. Another example is that on the cognitive level, the cognitive constructs C1, C2 and C3 interact because cognitive constructs are interactive in nature and their developmental pathways overlap. The final aspect of the model is that the liability distribution of a disorder

is often continuous and quantitative, not discrete and categorical, so that the threshold for having the disorder is somewhat random.



G = genetic riskor protective factor, E = environmental risk or protective factor, N= neural system, C= cognitive process, D= disorder

*Figure 3*. Multiple deficit model by Pennington (2006). Adapted from Pennington (2006, p.404).

The author noted that a full understanding of dyslexia will only be possible with a multiple model like the one he proposed, as single-deficit theories have the following problems:

- As dyslexia represents a multi-faceted and developmental disorder, no individual with dyslexia will present with a single deficit.
- Brain-behavioural relations are not constant, but vary with individual differences and development.
- 3. Single-deficit research tends to select pure samples based on a theory, thus confirming the theory in a circular manner.

In summary, there is strong empirical support underlining the importance of core phonological deficits for the reading/spelling difficulties found in dyslexia. In contrast, evidence for theories of basic visual and auditory processing deficits has been equivocal with some studies confirming the occurrence of visual and auditory deficits, and others not. Taken together, to account for the heterogeneity of dyslexic readers, the current evidence leads to a multidimensional framework for dyslexia. Future research is needed to integrate multidimensional levels of investigation. Although the described theories have largely contributed to our understanding of the cognitive phenotype of dyslexia, the increase of neurophysiological techniques to investigate dyslexia has provided knowledge concerning the neurobiological phenotype of dyslexia. The following chapter will describe genetic, anatomical and neurobiological (imaging and electrophysiological studies) evidence, supporting the neurobiological phenotype of dyslexia.

## **Chapter 5: Neurobiological Basis of Dyslexia**

## Genetic Influences on Dyslexia

Genetic studies of dyslexia have indicated the importance of family history as a risk factor with 23-65% of children who have a parent with dyslexia reported to have the same condition (Pennington & Gilger, 1996; Scarborough, 1990). Results of twin studies have demonstrated a higher concordance rate in monozygotic twins (84-100%) compared to dizygotic twins (20-35%). Moreover, research has indicated a relatively high heritability index of  $h_g^2$ = 0.6, with both autosomal dominant and recessive genetic transmission (e.g., Olson & Byrne, 2005). In addition, replicated linkage studies have shown heterogeneity of dyslexia indicating diverse chromosome loci (1, 2, 3, 6, 15, and 18) involvement in the disorder (e.g., Fisher & DeFries, 2002). Importantly, Shaywitz et al. (2008) have pointed out that if a child had a parent or sibling with dyslexia the child should be considered at-risk and early intervention or prevention should be conducted. It should also be stressed that the evidence for a genetic abnormality does not imply that a child cannot benefit from intervention.

# Anatomical Evidence of Dyslexia

Several regions of the brain are important for the processing of language and in particular, for reading and spelling. One well-established research finding is that the left hemisphere of the brain, including a left anterior network and two left posterior networks (see Figure 4), serves language functions (for a review see Shaywitz et al., 2008). The *left anterior network* (in particular Broca's area around the inferior frontal gyrus; red oval in Figure 4) is associated with articulation, silent reading and naming, and appears to act as an executive system controlling access, retrieval, selection, and gating of information (Cao, Bitan, Chou, Burman, & Booth, 2006). The *two left posterior networks* are critical for fluent reading and involve one parietal-temporal network (in particular planum temporale, Wernicke's area and peri sylvian region around the middle temporal and angular gyrus; green oval in Figure 4), which serves word analysis and phonological processing, and one occipital-temporal

network (around the inferior temporal gyrus; blue oval in Figure 4), which is associated with visual word form processing. In addition, the occipital-temporal network appears to be specifically responsive to well-learned visual word forms (Cohen et al., 2000; Price, Wise, & Frackowiak, 1996; Tarkiainen, Helenius, Hansen, Cornelissen, & Salmelin, 1999).



*Figure 4*. Gray's schematic illustration of the brain and its language-associated regions. Adapted from Clemente (1985, p.1038).

Findings from post-mortem analyses of dyslexic brains have shown anatomical differences compared to non-dyslexic brains (for a review see Habib, 2000), including lack of asymmetry of the planum temporale (normally left larger than right) (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Hynd & Semrud-Clikeman, 1989), ectopic neurons (small neuronal congregations in an abnormal layer location) particularly in the left hemisphere (Galaburda & Kemper, 1979), decreased anisotropy (cells are organised in a certain direction) of the white matter in the peri sylvian region (Klingberg et al., 2000), and focal dysplasia (loss of characteristic architectural organisation of cortical neurons) in language regions of the brain (Kaufman & Galaburda, 1989). With regard to the early findings on the lack of asymmetry of the planum temporale, Eckert and Leonard (2000) argued in their review of 20 studies, that the asymmetry has not been consistently found. The search for anatomical differences between dyslexic readers and controls has also been extended to the cellular level. Within the framework of the magnocellular theory Galaburda

and Livingstone (1993) reported that dyslexic readers had more disorganised and smaller magno cells, but no anomalous parvo cells (see also Livingstone et al., 1991). Furthermore, Galaburda and Livingstone found smaller and disorganised cells in the auditory channel, providing some neural evidence for the auditory temporal theory by Tallal (1980). In sum, the findings from anatomical research have indicated anomalous left-hemisphere posterior regions in the dyslexic brain. Results of imaging studies and electrophysiological studies have further enlightened our understanding of deficient neural systems in dyslexia and are reviewed and discussed in the following sections.

## Neural Correlates of Dyslexia: Imaging Studies

A vast number of neuroimaging studies have demonstrated the atypical activation pattern of dyslexic readers during reading (for reviews see Goswami, 2004; McCandliss & Noble, 2003; Shaywitz et al., 2008; Zeffiro & Eden, 2000). Neuroimaging techniques such as fMRI and PET are based on the principle that cognitive tasks produce change in blood flow in brain regions and images of this changed regional brain activity can be obtained.

Compared to normal readers, dyslexic readers have been found to have decreased activity in the posterior left hemisphere, indicating deficient processing in the two left posterior language networks (parietal-temporal and occipital-temporal, as described earlier) of the brain (Brunswick, McCrory, Price, Frith, & Frith, 1999; Helenius, Tarkiainen, Cornelissen, Hansen, & Salmelin, 1999b; Horwitz, Rumsey, & Donohue, 1998; Paulesu et al., 2001; Rumsey et al., 1997; Shaywitz et al., 1998; Shaywitz et al., 2002). These two networks are crucial for sound-symbol linkages (phonological processing), fluent reading, and word analyses. For example, a study by Rumsey et al. (1992) demonstrated that dyslexic readers had reduced activity in the left parietal-temporal region (peri sylvian area) during a phonological rhyming task compared to controls and that phonological skill correlated with recruitment of this brain area (see also Rumsey et al., 1997). An fMRI study by Cao et al. (2006) also demonstrated the importance of the left middle temporal gyrus (part of the posterior left parietal-temporal network) for semantic processing. In their study dyslexic readers and controls made rhyme judgements. It was found that controls recruited the left

middle temporal gyrus to use semantic information to assist in their judgment on the rhymes, whereas dyslexic readers failed to show enhanced activity in this region. In other words, they failed to access and/or use semantic information (see also Friederici, Opitz, & von Cramon, 2000; Pugh et al., 1996). However, semantic processing and reading comprehension have not been investigated in many imaging studies. More complex semantic tasks, such as sentence processing, have been intensively studied with electrophysiological techniques, in particular event-related potentials (ERPs). These studies are discussed below.

Imaging findings for the second posterior network, the left occipital-temporal region, indicate that dyslexic readers show a lack of responsiveness during the presentation of words compared to controls. Activation of this region was further correlated with reading skill (Brunswick et al., 1999; Cao et al., 2006; Shaywitz et al., 2002). The occipital-temporal region is highly specialised for visual word processing as indicated by a higher activity during visual word presentation compared to non-word stimuli such as checkerboards (Cohen et al., 2000; McCandliss, Cohen, & Dehaene, 2003), auditory words, and false font characters (Dehaene, Le Clec, Poline, Le Bihan, & Cohen, 2002). This region develops continuously through print exposure as soon as reading acquisition begins (Aghababian & Nazir, 2000; McCandliss, Posner, & Givon, 1997; Posner, Abullaev, McCandliss, & Sereno, 1999) and the skilled reader usually develops visual expertise, which allows for rapid word recognition within 200 ms (for a review see Rayner, Juhasz, & Pollatsek, 2005). Shaywitz et al. (2008) review the neurobiological evidence for dyslexia and refer to the disruption of the two posterior networks as the neural signature of dyslexia, reflecting the evidence to date.

Investigation of the third language network, the left anterior system (inferior frontal gyrus) has revealed less conclusive results. Some studies have found this network to be overactive in dyslexic readers (Brunswick et al., 1999; Corina et al., 2001; Georgiewa et al., 2002; Shaywitz et al., 1998). Other studies have found no differential activation between dyslexic and controls (Paulesu et al. 2001; Rumsey et al, 1997), and still others revealed under-activation (Cao et al., 2006; Georgiewa et al., 1999). The study by Cao and colleagues showed that dyslexic children had decreased activation during a phonological rhyming task

(see also Poldrack et al., 1999) in the left inferior frontal gyrus compared to relatively increased activation in controls. Furthermore, the posterior networks (occipital-temporal and parietal-temporal) also showed diminished activation in dyslexic readers during task execution. The authors concluded that dyslexics have deficient orthographic representations and difficulties with orthographical-phonological mapping. In contrast, Shaywitz et al. (1998) reported over-activation of the anterior network in adult dyslexic readers and argued that over-activation may indicate a compensatory mechanism, showing an increased reliance by dyslexic readers on phonological decoding. As reported earlier, activity in the left anterior network is normally enhanced during phonological tasks compared to semantic tasks (Mummery, Patterson, Hodges, & Price, 1998). The reason for these divergent results appears to be the age of the study participants. Taken together, these findings suggest that adult dyslexic readers show over-activation of the left anterior network, whereas most dyslexic children show under-activation of this network (Shaywitz et al., 2002).

Other findings on compensative and relative over-activation of brain areas in dyslexic readers included a larger-right-than-left activation, in particular increased activation of the homologous area of the occipital-temporal network in the right hemisphere, which was found to correlate negatively with reading skill (Shaywitz et al., 2003). In addition, compensated dyslexic readers show over-activity in the left frontal areas of the brain while reading text (Horwitz et al., 1998; Shaywitz et al., 2002) and increased activity in the right frontal area during phonological decision tasks (Shaywitz et al., 2003). Shaywitz et al. (2003) associated the over-activity of the right frontal regions with working memory and retrieval demands. These findings have been interpreted as indicators of compensatory mechanisms, which occur due to ongoing failure to read and serve to overcome some of the difficulties. Shaywitz et al. (2008), for example, pointed out that a compensated dyslexic may well read accurately, but will still be a very slow reader.

It is important to remember that a large amount of this research has been conducted with adult dyslexic readers, raising the question of whether the neural abnormalities are primary or secondary to the symptoms of dyslexia. Nevertheless, as Shaywitz et al. (2008) made clear, several studies with children have now confirmed the existence of abnormalities in children. These findings reduced the possibility that the brain abnormalities seen in adults are due to ongoing failure to read (Seki et al., 2001; Shaywitz et al., 2002; Simos et al., 2000; Temple et al., 2000). McCandliss and Noble (2003) noted that to verify the causal assumption more rigorously, more studies are needed to investigate children who are at risk for dyslexia but who have not had any formal reading instruction. The authors highlighted another potential problem for the interpretation of imaging findings, which they refer to as the "task performance confound". Dyslexic readers commonly perform more poorly than controls on cognitive tasks during imaging recoding. Thus there is a possibility that the differing brain activation is due to lower levels of task accuracy. However, in a study by Paulesu et al. (1996) the task accuracy of dyslexic and control readers was matched during practice trials before the actual scanning was conducted and dyslexic readers still showed reduced activity in the peri sylvian region during a letter-rhyming task.

In sum, the majority of imaging studies have made dyslexia a visible condition and identified the disruption of left posterior language networks in dyslexic readers. They have also identified some areas of over-activation which have been suggested to serve as compensatory mechanisms. Moreover, in cross-language imaging studies, these deficits have further been replicated for diverse languages which suggest a shared biological mechanism for dyslexia (Shastry, 2007; Paulesu et al., 2001). High correlations between neural abnormalities and phonological and reading skills have been established (Eckert, Lombardino, & Leonard, 2001; Habib & Robichon, 1996). The development of these important language networks of the brain appears to be disrupted at some stage of development in dyslexic children. A description of how these language networks develop would exceed the scope of the thesis but for a full discussion the reader is referred to the articles by Maurer et al. (2007) and McCandliss and Noble (2003).

## Neural Correlates of Dyslexia: ERP Studies

A number of studies have used the ERP technique to investigate the neurophysiological basis of normal linguistic processing and to investigate how these functions might differ in dyslexic readers. First, the ERP technique will be briefly explained, followed by a review of the findings from ERP studies of normal linguistic processing. The last section reports empirical studies that have investigated ERP differences between dyslexic readers and controls.

# The Event-related-potential Technique

ERP measures allow analysis of neural changes related to information processing with a time-resolution in the order of milliseconds. They are therefore particularly useful in examining questions of the speed and time order of cognitive operations at different stages of information processing. Although the spatial resolution of ERPs is limited since multiple neural generators are likely to be involved in the activation (Fabiani, Gratton, & Coles, 2000), multichannel recordings provide an estimation of the intracerebral locations of the cerebral processes (Picton et al., 2000). An ERP is of smaller amplitude (5-15  $\mu$ V) than the background noise EEG activity (50-100  $\mu$ V) and is extracted by computer averaging. ERP components are referred to as the negative or positive peak visible in the average waveform and are labelled according to their polarity and time distribution (e.g., N1 = negative deflection occurring around 100 ms after stimulus onset) or functional meaning (e.g., MMN = Mismatch Negativity). The amplitude of a component is held to reflect the activation strength and resource allocation during cognitive processing and the latency of a component represents the speed of cognitive information processing (Kok, 2001; Kramer, Strayer, & Buckley, 1991).

The different components in an ERP waveform reflect various neural processes that occur at different time points in response to a given event. Task manipulations influence the ERP components at different time points. As a general rule, the earlier (or exogenous) components of the ERP such as N1 and P1 are associated with sensory activity related to physical stimulus processing and are robust and individually replicable over sessions. With regard to psycholinguistic processing, however, the more informative ERP components are the later (endogenous) components, which occur after the initial sensory processing (about 100 ms) and depend on the cognitive operations within the individual. The review presented below focuses on tasks in the visual modality and on language-related endogenous ERP components. ERP research on language processing has widely focussed on the N4 and Late Positive Component (LPC) components and investigated these components in single-word and sentence tasks. Another line of research is concerned with earlier linguistic processing, associated with the P1, N2 and P2 components of the ERP. The review will focus on studies that have investigated the N4, LPC and P2 components as these are of particular importance within the scope of the current thesis.

## **ERPs** and Normal Visual Linguistic Processing

N4. ERP studies of linguistic processing have typically investigated the N4 component, a negative-going wave occurring between 250 to 600 ms after stimulus onset (for reviews see Kutas & Van Petten, 1994; Kutas, Van Petten, & Kluender, 2006). The initial study by Kutas and Hillyard (1980) compared ERP components for congruent semantic and congruent physical final words in a sentence, semantic incongruent sentence endings (strong and moderate incongruence), and physically deviant endings (sentence endings written in large capital letters). Their finding was that the semantically incongruent endings elicited a large negative component around 400 ms which was distributed across the scalp but more pronounced in the right central-parietal area for the strong incongruent endings (e.g., "He took a sip from the transmitter"). The strongly incongruent endings showed larger negative amplitudes than the moderate incongruent endings (e.g., "He took a sip from the waterfall"). An LPC was observed following physically deviant endings. The negative wave observed after semantic incongruence was named N4 and was argued to "reflect the interruption of ongoing sentence processing by a semantically inappropriate word and the reprocessing ... that occurs when people seek to extract meaning from senseless sentences" (Kutas & Hillyard, 1980, p.204).

This finding has stimulated a vast amount of research into the semantic specificity of the N4 component in various task designs (for a review see Pritchard, Shappell, & Brandt, 1991). Subsequent research on sentence processing replicated the original finding of Kutas and Hillyard, showing larger N4 amplitudes to semantically incongruent than congruent sentence endings both in the visual (e.g., Friederici, Steinhauer, & Frisch, 1999; Gunter, Stowe, & Mulder, 1997; Hagoort, 2003; Kuperberg, Sitnikova, Caplan, & Holcomb, 2003; Kutas & Hillyard, 1983, 1984, 1989; Nobre & McCarthy, 1994; Osterhout & Nicol, 1999) and auditory (e.g., McCallum, Farmer, & Pocock, 1984) modalities. N4 amplitude further varied with different task manipulations including (1) word position, that is, if the semantically incongruent word appeared in the middle of a sentence, the N4 amplitude was larger than when the sentence was completed with a semantically inappropriate word (Hinojosa, Martin-Loeches, & Rubia, 2001; Van Petten & Kutas, 1990), (2) word frequency (normative count of usage frequency of a word in a language) with low-frequency words eliciting larger N4 amplitudes than high-frequency words (Allen, Badecker, & Osterhout, 2003; Van Petten, 1993), and (3) word category, that is, N4 was larger when the semantically incongruent word belonged to the open-class category (content words: nouns, verbs, adjectives), than closed-class category words (function words: e.g., prepositions "by", conjunctions "but"), which Van Petten and Kutas (1991) explained in terms of the higher frequency of usage for closed-class words. A large number of studies have also observed N4 in response to congruent endings with N4 amplitude being inversely proportional to the goodness-of-fit in a given sentence (e.g., Kutas & Hillyard, 1984). Kutas et al. (2006) argued that differences in Cloze probability (defined as the proportion of a large sample of persons using a given word to complete or "close" a sentence) accounted for these effects: Less expected final words elicit larger N4 amplitudes than more predictable completions (Kutas, Lindamood, & Hillyard, 1984), with semantically incongruent completions representing one of the lowest Cloze probabilities.

Although the N4 has been mainly studied in sentence task designs, studies using word designs have reported N4 for tasks such as semantic oddball (Shappell, Pritchard, Brandt, & Barratt, 1986), semantic priming (Kutas & Hillyard, 1989), categorical mismatch (Harbin, Marsh, & Havey, 1984), lexical decision (Boddy, 1986), and new-old memory tasks (Neville, Kutas, Chesney, & Schmidt, 1986). In particular, semantic priming designs demonstrated that N4 amplitude was larger when semantic cues were unrelated to the target stimuli in a given task, as opposed to semantically related cues. In addition, N4 amplitude decreased with increasing semantic priming (Hinojosa et al., 2001; Radeau, Besson, Fonteneau, & Castro, 1998).

Lexical decision task designs originated from psycholinguistic research and commonly involve the presentation of real words in contrast to pronounceable nonwords (pseudowords) or unpronounceable nonwords, requiring the participant to judge if the presented word is spelled correctly (Gernsbacher, 1994). Findings indicated larger N4 amplitudes for pseudowords and similar or somewhat smaller N4 amplitudes for words compared to unpronounceable nonwords, which elicited little or no N4 activity (Anderson & Holcomb, 1995; Chwilla, Brown, & Hagoort, 1995; Fonseca, Tedrus, & Gilbert, 2006; Hauk, Davis, Ford, Pulvermueller, & Marslen-Wilson, 2006; Ziegler, Besson, Jacobs, Nazir, & Carr, 1997). An additional result reported by Kounios and Holcomb (1994) was that pseudowords, which are close to real words, were more likely to elicit the same N4 activity, suggesting that pseudowords may access semantic memory. These findings suggested that the absence of the N4 to unpronounceable nonwords is due to the nonwords not following orthographical and phonological rules and thus having no semantic relevance.

Finally, the N4 in single-word task designs has been frequently found over central, parietal, and central-parietal regions. However, a semantic decision task by Bentin, Mouchetant-Rostaing, Giard, Echallier, and Pernier (1999) showed enhanced left frontal N4 amplitudes in response to pseudowords, smaller N4 to words and no N4 for unpronounceable nonwords. The authors related the frontal activity to semantic memory processes. In addition, whereas in sentence task designs N4 has been found to be maximal in the right hemisphere, in the semantic word study by Bentin et al. (1999) the N4 amplitude was left lateralised (see also Nobre & McCarthy, 1994). The N4 also does not seem to be limited to verbal stimuli, as N4 has also reportedly been elicited by pictorial stimuli ending a sentence anomalously (Nigam, Hoffman, & Simons, 1992), by meaningful line drawings, photos, and environmental sounds (Ganis, Kutas, & Sereno, 1996; Holcomb & McPherson, 1994; Plante, Van Petten, & Senkor, 2000).

Taken together, the findings suggest that N4 amplitude is not an indicator of semantic abnormalities per se, but associated with violations of expectancy based on any kind of semantic priming. In line with this finding is the decrease in N4 amplitude with stimulus repetition (Kutas & Van Petten, 1994). In addition, different task manipulations

including word category used (open-class, closed-class), word frequency (low, high) and word position in sentence designs (middle, terminal) impact on N4 amplitude and need to be taken into consideration when designing experimental tasks.

N4 latency has not been investigated in many studies, as it has proved to be relatively stable under diverse task manipulations (for a review see Kutas et al., 2006). However, a few studies have reported longer N4 latency in a lexical ambiguity task when the prime was contextually inappropriate to the ambiguous word as opposed to contextually appropriate to the ambiguous word (Van Petten, 1995; Van Petten & Kutas, 1987) and when incongruent words in a sentence task were presented rapidly (10 words per second), (Kutas, 1987).

LPC. The LPC (also called P6) occurs between 500 and 800 ms after stimulus onset, but can occur as early as 200 ms following the P2 component. The LPC is broadly distributed but often maximal at central-parietal sites. It has often been observed following syntactic, grammatical, or physical violations during linguistic tasks (e.g., syntactical anomalies involving phase structure, subject-verb agreement), in sentences (e.g., syntactical incorrect completions), or word pair tasks (e.g., syntactical incorrect pairs) as opposed to syntactical correct sentences/word pairs. This component has been interpreted as an indicator of orthographic-syntactic processing (Garnsey, Tanenhaus, & Chapman, 1989; Kutas & Hillyard, 1983; Kutas et al., 2006; Neville, Nicol, Barss, Forster, & Garrett, 1991; Osterhout, Holcomb, & Swinney, 1994). Studies that have investigated the LPC component in word task designs without overt syntactical violations are scarce, probably because most studies have researched the N4 component. Nevertheless, a study by Ziegler et al. (1997) found significantly larger LPC amplitudes for unpronounceable nonwords than for words and pseudowords and a study by Lovrich, Kazmerski, Cheng, and Geisler (1994) reported later LPC latency for a letter rhyming task compared to a less linguistic letter form task. Some authors refer to the LPC as an SPS (Syntactic Positive Shift) component to emphasise its relation to syntactical processing since the SPS is commonly observed following syntactical violations in sentence and word tasks. The component is held to reflect the inability of the information processing system to assign a preferred syntactical structure in

response to syntactical violations (Hagoort, Brown, & Groothusen, 1993; Osterhout, McKinnon, Bersick, & Corey, 1996). Other researchers have argued that the LPC should be included in the family of P3 components since it showed a similar scalp distribution, indicating solely a delayed P3 occurring at 600 ms after stimulus onset due to more complex task requirements (Coulson, King, & Kutas, 1998). The P3 component is one of the most intensively studied components of the ERP and is commonly elicited by an unexpected but task-relevant stimulus (Coles, Smid, Scheffers, & Otten, 1995; Kok, 2001).

In sum, research results on the LPC are not as conclusive as those on N4. As Kutas et al. (2006) pointed out, the LPC has also been found during complex syntactic tasks and syntactically well-formed sentences with non-preferred structure, suggesting that the LPC is not specific to syntactic violations per se. Moreover, some research suggested that the LPC also occurs following semantic incongruity (Besson, Kutas, & Van Petten, 1992; Kuperberg, 2007; Kuperberg, Sitnikova, Caplan, & Holcomb, 2003; Muente, Heinze, Matzke, Wieringa, & Johannes, 1998). This finding questions the specificity of the LPC as a solely syntactical-orthographical indicator.

Some studies have investigated the combination of syntactic and semantic violations in sentence and word pair tasks and reported both larger N4 and LPC components for combined violations (Kutas & Hillyard, 1980, 1983), or larger N4 but same LPC (Hagoort, 2003), or a left anterior negativity-LPC complex but no N4 (Friederici, Gunter, Hahne, & Mauth, 2004). These results are far from conclusive but they appear to indicate some kind of interactive processing between syntactic and semantic processes. The discovery of syntactic and semantic associations in the ERP led to two distinct theoretical approaches to understanding the temporal order of syntactic and semantic processing. One set of approaches (referred to as connectionist approaches) advocate a continuous integration of syntactic and semantic information as a sentence is processed (e.g., MacDonald, Pearlmutter, & Seidenberg, 1994; McClelland, St. John, & Taraban, 1989; Mitchell & Holmes, 1985). This is in contrast to the other set of approaches (referred to as early visual word recognition theories), which claim that syntactic analysis is distinct from and occurs before semantic processing of a word (Clifton, Speer, & Abney, 1991; Muente, 1993; Rayner, Garrod, & Perfetti, 1992; for a review see Lupker, 2005) (See also Chapter 3).

Finally, a methodological aspect relevant for studying the N4 and LPC, the "overlap issue" (Pritchard et al., 1991; Kutas & Van Petten, 1994), should be mentioned. As the N4 and LPC components occur in a similar time window, especially in sentence task designs, component overlap has been the subject of considerable debate. Overlap between N4 and LPC commonly occurs when an overt decision task is involved (Kutas & Van Petten, 1994). One way of dealing with overlap is thus to avoid task-related decisions (Kutas & Van Petten, 1994). A second way is to avoid response-related P3 activity during the time window of the N4 (200 to 500 ms) by delaying the motor response to the final word in a sentence (Holcomb, Coffey & Neville, 1992) and a third option is to compute difference waveforms by subtracting the congruent from the incongruent waveforms (Pritchard et al., 1991), based on the assumption that the P3 does not vary significantly between the congruent/incongruent conditions.

P2. The P2 component has been classified as both an endogenous and an exogenous component and seems to reflect feature detection, selective attention, and stimulus encoding (Dunn, Dunn, Languis, & Andrews, 1998; Hackley, Woldorff, & Hillyard, 1990; Luck & Hillyard, 1994; McDonough, Warren, & Don, 1992; Shibasaki & Miyazaki, 1992). Visual word recognition studies that have investigated P2 and other earlier linguistic ERP components (e.g., P1, N2) have reported word processing differences in the brain as early as 160 ms after stimulus onset (Dehaene, 1995; Hinojosa et al., 2001; Landi & Perfetti, 2007; Martin-Loeches, Hinojosa, Gomez-Jarabo, & Rubia, 1999). For example, Landi and Perfetti (2006) reported larger P2 amplitudes for homophone pairs versus non-homophone pairs in a phonological task, and larger P2 amplitudes for semantically unrelated word pairs as opposed to semantically related word pairs in a semantic word task, with the effects most pronounced over frontal and central electrode sites. Similarly Ziegler et al. (1997) found larger P2 amplitudes in the left anterior region for words as opposed to pseudowords and nonwords, which did not differ. However, a study by Fonseca et al. (2006) did not find differential P2 components for pseudowords and real words but pseudowords and real words

were distinguished at a later time window (N4). P2 amplitude has also been shown to vary with both word frequency and word length. Specifically, low-frequency words and longer words are associated with larger P2 amplitudes.

During the time window of the P2 at frontal sites, polarity is reversed at occipital sites selectively. Most commonly found is a P1-N2 complex in the waveform, which has been associated with initial word form analysis (surface features of a word) in visual word recognition tasks. For example, it has been shown that N2 amplitude is larger following visual linguistic stimuli as opposed to visual non-linguistic stimuli at occipital sites (Simon, Bernard, Largy, Lalonde, & Rebai, 2004). However, these early components are not the major interest of this study. For a more detailed review of these early linguistic ERP components see Simon et al. (2004) and Hauk et al. (2006). In conclusion, Hauk et al. state that, "although a pattern is emerging ... that the earliest electrophysiological effects, around 100 ms, are related to surface features of written words, which are subsequently followed by lexicality and semantic word properties, the results are still partly inconsistent and electrophysiological data on early word recognition is still sparse" (p.1384). It should be noted that most studies of earlier linguistic processing have focussed on word task designs and P2 and other earlier components have rarely been investigated in sentence task designs, which is probably attributable to the main interest of the research in N4 and LPC components during sentence processing (Landi & Perfetti, 2007). The following section will discuss findings in relation to linguistic processing in dyslexic samples.

# ERPs and Linguistic Processing in Dyslexia

ERPs have been widely applied to the investigation of dyslexic and control samples. Early ERP studies of dyslexia were mostly concerned with early and non-linguistic processing in dyslexic readers. For instance, it has been reported within the validation of the magnocellular hypothesis that dyslexic readers have smaller amplitudes and delayed latencies for the N1, P1 and N2 components (e.g., Brannan, Solan, Ficcara, & Ong, 1998; Lehmkuhle et al., 1993; Livingstone et al., 1991). Of particular importance for the current thesis are ERP studies that have compared the ERPs of dyslexic and control readers in linguistic tasks. Dyslexic readers commonly show P2, P3, N4, and LPC components which diverge from those of controls in the auditory and visual domains. They have been shown to have longer ERP latencies (e.g., Breznitz & Meyler, 2003; Taylor & Keenan, 1990) and smaller amplitudes (e.g., Ackerman, Dykman, & Oglesby, 1994; Barnea et al., 1994; Holcomb, Ackerman, & Dykman 1985, 1986) or larger amplitudes than controls (e.g., Ackerman et al., 1994; Lovrich, Cheng, & Velting, 2003; Ruesseler, Johannes, Kowalczuk, Wieringa, & Muente, 2003). Diverging findings on ERP amplitudes are mostly due to variations in linguistic task manipulations and ERP components investigated (see later in this chapter for details). ERP studies in which the electrical response distribution has been investigated indicate atypical electrical distribution for dyslexic readers during linguistic tasks. Normal readers exhibit more pronounced amplitudes over the left hemisphere while processing linguistic stimuli (Brunswick & Rippon, 1994; Geschwind, 1970; Shaywitz et al., 2008). In contrast, dyslexic readers variously showed similar ERP amplitudes over both hemispheres or a larger-right-than-left asymmetry, thus confirming, in general, results from imaging studies.

*N4 and Dyslexia.* The N4 is most commonly evoked in sentence tasks, but studies investigating electrophysiological sentence processing in dyslexic readers are scarce. Brandeis, Vitacco, and Steinhausen (1994) found delayed N4 latencies for dyslexic children for semantically incongruent sentence completions compared to controls, who showed no latency differences between the incongruent/congruent conditions. In addition, N4 amplitude for the incongruent endings was significantly smaller for dyslexic readers than for the control group. Moreover, within the dyslexic group the effect of ending was not significant, indicating that incongruent and congruent endings elicited N4 amplitudes of similar magnitude within this group. In contrast, a study by Neville, Coffey, Holcomb, and Tallal (1993) reported larger N4 amplitudes and longer N4 latencies for language-impaired children in response to both semantic incongruent and congruent sentences. The authors interpreted this as "compensatory increases in the effort required to integrate words into context" (p. 248). These results were confirmed for a dyslexic adult sample in a study by Robichon, Besson, and Habib (2002). Helenius, Salmelin, Service, and Connolly (1999a) compared semantic incongruent, and combined syntactic and semantic violations

in a sentence task in dyslexic and control samples. Results were in contrast to the other studies; no N4 amplitude differences between controls and dyslexic readers (see also Sabisch, Hahne, Glass, Suchodoletz, & Friederici, 2006, for confirming result for auditory sentences). However N4 latency was delayed for the dyslexic group in response to incongruent endings.

ERP studies that have investigated N4 in single-word tasks designs have revealed smaller N4 amplitudes for dyslexic readers during a visual memory task (acquisition and recognition), (Stelmack, Saxe, Noldy-Cullum, Campbell, & Armitage, 1988), a memory priming task (Stelmack & Miles, 1990), a rhyme/no-rhyme decision task (Ackerman et al., 1994), and in response to words as opposed to pictures in a semantic naming task (Greenham, Stelmack, & van der Vlugt, 2003). An interesting result was obtained by Miles and Stelmack (1994) who found no decrease in N4 amplitude for dyslexic readers following priming, whereas controls showed the common effect of reduced N4 amplitude to primed versus unprimed words. Similarly, Landi and Perfetti (2007) found that less-skilled comprehenders yielded N4 amplitudes of similar magnitude to semantically related and unrelated word pairs, which appears to reflect less linguistic sensitivity in poor comprehenders. N4 latency differences between dyslexic readers and controls have also been implicated in a study by Breznitz (2003), with dyslexic readers showing longer N4 latencies following orthographic, phonological, and rhyme tasks. With regard to distributional differences of the N4 between dyslexic readers and controls, Penolazzi, Spironelli, Vio, and Angrilli (2006) indicated a more broadly distributed N4 in dyslexic readers, whereas controls showed a more pronounced left anterior N4 amplitude in response to orthographic, phonological, and semantic word tasks (see also Gruenling et al., 2004).

LPC and Dyslexia. With regard to the LPC component, ERP studies on dyslexia are rare. Lovrich, Cheng, and Velting (1996) reported enhanced LPC (referred to as P800) amplitude and delayed LPC latency at frontal electrode sites for dyslexic readers in a rhyme discrimination task compared to controls, and Stelmack et al. (1988) found a larger LPC (referred to as P6) for dyslexic readers compared to controls in a word recognition task at left temporal, central and frontal sites. Similarly Ackerman et al. (1994) observed larger

50

LPC (referred to as P6) amplitudes for dyslexic readers than controls during a rhyme/no rhyme decision task. Schulte-Koerne, Deimel, Bartling, and Remschmidt (2004b) reported smaller LPC amplitudes for pseudowords in their dyslexic sample compared to controls. Interestingly studies on normal linguistic processing have mainly focused on LPC in relation to syntactic anomalies in sentence and word tasks. However, within the field of dyslexia, researchers have investigated LPC among dyslexic readers more in lexical and visual word recognition tasks than in tasks involving syntactic violations per se.

In summary, only a handful of studies have investigated LPC among dyslexic readers and results so far appear to indicate either larger or smaller LPC amplitudes for dyslexic readers, depending on the lexical word task design. Larger LPC amplitudes have been held to reflect increased processing efforts of dyslexic readers during complex linguistic tasks (e.g., Ackerman et al., 1994; Lovrich et al., 2003) whereas smaller LPC amplitudes have been associated with diminished resource allocation and quality of information retrieved for linguistic tasks (e.g., Schulte-Koerne et al., 2004b).

*P2 and Dyslexia.* Earlier linguistic ERP components in dyslexic samples, in particular the P2 component, have been investigated in a few studies and have revealed longer P2 latencies for words and larger P2 amplitudes for pseudowords for dyslexic readers compared to controls (Miller-Shaul & Breznitz, 2004). Similar results were obtained for a memory-recognition task (Stelmack et al., 1988) and a lexical decision task (Breznitz & Misra, 2003; Taylor & Keenan, 1990). Larger P2 amplitudes for less-skilled comprehenders have further been reported for a semantic probe-target task (Landi & Perfetti, 2007). The P2 amplitude to semantically related pairs was generally much higher compared to unrelated pairs. However, this difference was much less marked for less-skilled comprehenders compared to skilled comprehenders. The authors suggested that this larger P2 following semantically unrelated probe-target pairs might be the initial and more effortful access of semantic information in less-skilled comprehenders.

The reported P2 variations in dyslexic readers appear to occur across the scalp, indicating a lack of left asymmetry for dyslexic samples as opposed to control samples. In contrast to studies using word designs, a study by Neville et al. (1993) used a congruent/incongruent sentence task. They showed smaller P2 amplitudes for languageimpaired children compared to controls in response to incongruent and congruent sentence completions. However Robichon et al. (2002) did not find diminished N1-P2 amplitudes in their dyslexic sample during a sentence task.

# **Developmental Considerations.**

The findings of developmental ERP studies are of direct relevance for the present study, as the participants will be adolescents between 12 to 14 years of age whose skills and brain functions are still developing. In general, developmental ERP research has consistently indicated decreases in P3 latency from five or six years of age through to the early twenties (Courchesne, 1978; Friedman, Boltri, Vaughan, & Erlenmeyerkimling, 1985; Johnson, 1989) with a few studies also revealing a reduction in P3 amplitude with age (Johnson, 1989; Mullis, Holcomb, Diner, & Dykman, 1985). Studies that have investigated N1 and P2 have produced inconsistent results. The study by Johnson reported age effects with decreases in N1 and P2 latency from seven to 20 years, but no amplitude variations and others have found no age-related changes (Courchesne, 1978). The effects of development on N4 amplitude and latency were investigated in a sentence task (semantically incongruent/congruent completions) by Holcomb et al. (1992). Results revealed that the younger group (seven to12 years) showed N4 activity to both congruent and incongruent endings (with incongruent N4 larger), whereas the older group (15 to 26 years) only demonstrated N4 following incongruent endings. Further, the younger group showed a left focus and the older group a right focus of the N4. This study also revealed decreases in latency and amplitudes for N4, N1 and P2 from age five to 16 following which it was stable.

# Summary ERPs and Dyslexia

In summary, research indicates larger P2 amplitudes and delayed P2 latencies in word task designs and smaller P2 amplitudes in sentence task designs for dyslexic readers. These results indicate earlier linguistic processing differences between dyslexic readers and controls. With regard to the later, linguistic ERP components, LPC amplitude appears to be enhanced in dyslexic readers in various linguistic tasks and a delayed LPC latency has been

found in one study. With regard to N4, word task designs have shown diminished N4 amplitude, whereas sentence task design studies have variously revealed both larger and smaller N4 amplitudes and, in addition, longer N4 latencies for dyslexic readers. Further research with dyslexic samples, comparing tasks of different levels of complexity is needed to explain the diverging findings. Distributional differences in brain activity have further been implicated by some studies, showing that dyslexic readers have a broader or larger right than left activity pattern during linguistic processing in general. With regard to the N4 in word tasks, dyslexic readers seem to show a lack of activation in frontal areas. In contrast, LPC activity appears to be enhanced in frontal and central areas compared to controls. However, results are far from conclusive.

### Summary Neural Correlates of Dyslexia

Findings from ERP and imaging studies have largely contributed to our understanding of the neural phenotype of dyslexia. In particular, imaging studies have identified three language networks in the left hemisphere of the brain, one anterior and two posterior, which are deficient in dyslexic readers. These have been used as explanations for the phonological and reading difficulties in dyslexia, as well as compensated reading behaviours (accurate but persistent slow readers). Whereas imaging studies have mainly been able to locate deficient language systems in the brain, ERP studies have the advantage of a high time-resolution, allowing insight into the temporal order of linguistic processing in the brain. ERP findings on linguistic processing indicate that different linguistic features in simple (e.g., lexical, phonological word tasks) and complex (e.g., sentence tasks) tasks can be distinguished as early as 160 ms after stimulus onset and that dyslexic readers show delayed latencies in linguistic-sensitive components such as P2, LPC, and N4. Studies showing amplitude differences between dyslexic readers and controls in diverse linguistic tasks have been less conclusive since some studies report larger amplitudes and others smaller amplitudes for dyslexic readers compared to controls. The findings from neurobiological research have helped us to understand normal and deficient-dyslexic brain functions and can allow the evaluation of intervention programs on a neural level. One of the most important questions

is whether dyslexic brain function can be "re-organised" following interventions. The following chapter reports the most common intervention programs for dyslexia and their peer-reviewed efficacy. Although not many researchers have investigated neural changes after an intervention program, this chapter also reports the findings of these few studies that have used neural markers as intervention efficacy indicators in the last section since ERP components were investigated in the current study to evaluate neural changes after an intervention.

. .

.

### **Chapter 6: Interventions for Dyslexia and Their Outcomes**

# Introduction: Intervention Studies in the Field of Dyslexia

Dyslexia is of great social relevance since a person's success in our society depends largely on adequate written communication and thus is addressed and investigated by different disciplines. The shared goal of all disciplines is to treat the difficulties associated with dyslexia. Those involved in achieving this goal are the affected children themselves and their parents, schools and teachers, and researchers from different disciplines such as psychology, medicine, neurology, and education. On a more general level, educational and political efforts are increasing to improve literacy skills in many countries. The approach used to address the issue of literacy problems differs according to the discipline. Once literacy problems are detected in schools, the struggling students are most commonly referred to special education classes, with some of the programs being conducted within the classroom setting and others requiring the withdrawal of the students from normal classes. If services offered by the school are not sufficient then commercial education providers, outside the schools, offer a variety of programs to help overcome dyslexia. Political initiatives commonly focus on the development, monitoring, and evaluation of teaching approaches and special education services in schools. Using scientific methodologies, intervention research aims to investigate intervention programs for dyslexia. Most commonly researchers have evaluated programs offered in schools, commercial programs, or programs the researchers have developed themselves.

Each of the variety of theories on the causes of dyslexia has different implications for remediation. Based on the different theories the intervention programs can be divided into (1) basic perceptual interventions which usually include non-linguistic stimuli, (2) linguistic intervention which works with linguistic stimuli to stimulate certain aspects of reading and writing, and (3) integrated approaches which include basic perceptual and different aspects of linguistic skill interventions (phonological, fluency, comprehension). Due to the fact that educators may feel "bombarded" by a variety of multimedia intervention programs and devices for remedial instruction, there is an urgent need to evaluate the efficacy of different intervention methods for the dyslexic child. According to Alexander and Slinger-Constant (2004), evaluation studies in the field of dyslexia can be divided into two types of studies: Interventions targeted at preventing reading difficulties in at-risk younger children (children in kindergarten who have had minimal exposure to reading: prevention studies) and approaches to treating older reading-disabled children (those who have had exposure to adequate reading instruction and have not learned to read: intervention studies). The following report will focus intervention studies for the already readingdisabled child because the findings of these studies are of particular importance within the scope of the current thesis. It should be noted that overall gains made by older children (after Grade 2) are much less frequent and smaller than the gains made by younger children (Shaywitz et al., 2008). The literature includes intervention studies from basic and linguistic interventions as well as the combination of both. Most of the intervention studies discussed below have been conducted as classroom-based programs or commercial, out-of-class programs.

#### **Evaluation of Interventions Targeting Basic Non-linguistic Processing**

Individuals with dyslexia have been reported to have co-occurring non-linguistic deficits in the visual and auditory domain (see Chapter 4). A shared principle of the interventions targeting basic functions such as visual, auditory and sensorimotor functions is that they claim to treat the underlying fundamental processes involved in dyslexia rather than the symptoms. This is in contrast to linguistic interventions which target the overt symptoms such as phonological and reading problems, directly.

# Interventions Targeting Visual Processing

According to von Suchodoletz (2007), visual intervention includes training of (1) visual differentiation ability, (2) eye movement control, and (3) binocular vision. Studies evaluating the effects of these intervention programs are rare. The most frequently evaluated intervention is the use of Irlen lenses to treat dyslexia (Irlen & Lass, 1989). Irlen lenses are coloured glasses, most commonly red, blue, green, yellow or orange, which are used to enhance the function of the visual system and improve the timing of the sustained and

transient visual pathways. Another common remediation technique for visual deficits is the use of occlusion of one eye through patching, which is aimed at minimising instability while reading (the letter moving phenomenon). In a study by Clisby et al. (2000) some dyslexic children were given coloured lenses aimed at making small print clearer for them, and others, who showed unfixed ocular dominance, were given monocular occlusion (patching). This study reported considerable gains in reading age for participants. A similar study by Stein et al. (2000b) reported a 16-month gain in reading ability for a group of dyslexic readers showing visual instability at pre-test who were treated with monocular occlusion and tinted lenses compared to an eight-month gain over a nine-month period in a group treated with tinted lenses only. However, as pointed out by Alexander and Slinger-Constant (2004), when dividing the groups according to their visual stability post-intervention, those who had normal visual stability after the intervention and those who still showed visual instability both gained in reading age. Moreover, all participants were still lagging behind in their reading ages compared to the normal achieving comparison sample. A study by Martin, Mackenzie, Lovegrove, and McNicol (1993) did not find evidence in support of the use of Irlen lenses to treat dyslexia. Their sample comprised dyslexic readers without previous visual difficulties, in contrast to the study conducted by Stein et al. which exclusively selected participants with demonstrated visual problems.

In conclusion, in a small subset of dyslexic readers visual processing problems might account for some of the difficulties. Irlen lenses may then provide useful assistance in achieving higher visual stability, which may facilitate the reading process by stabilising visual input (Alexander & Slinger-Constant, 2004). An alternative to the use of Irlen lenses has been suggested by Williams, Lecluyse, and Rock-Faucheux (1992). These researchers investigated the impact of a meta-contrast program, using red, blue and acetate coverings of white-written words and sentences presented on a computer screen.. Both blue and red writing resulted in gains in reading comprehension for the dyslexic sample, with blue having a greater impact than red. It should be noted, however, that reading comprehension was measured by a test developed by the researchers, rather than by an already published test. The authors suggested that the lower contrast produced by the coloured writing slowed down the sustained (parvocellular) system allowing a greater temporal separation from the transient (magnocellular) system processes, resulting in less interference. In conclusion the authors stated that the use of coloured text may assist some dyslexic readers and can be implemented almost at no expense as opposed to the high-cost Irlen lenses.

# Interventions Targeting Auditory and Temporal Processing

The development of basic auditory interventions has been theoretically influenced by the work of Tallal (1980) who argued that the difficulties associated with dyslexia are attributable to a basic temporal auditory weakness. This temporal weakness hinders dyslexic readers in their attempts to perceive and discriminate the sounds of language in a fast and efficient way, thus impacting on the development of adequate phonological skills, which then leads to reading difficulties. To target these basic processing problems, tone and time discrimination intervention programs, such as FastForWord, have been developed. Other intervention programs involve direction hearing training and high-pitched tone training (von Suchodoletz, 2007).

Interventions for basic auditory processing deficits and scientific evaluations of these interventions are relatively uncommon. According to a review by Alexander and Slinger-Constant (2004), only two basic-auditory computer programs, namely the FastForWord Program (Scientific Learning Cooperation, 1996) and the Earobics program (Cognitive Concepts, 1998) have been investigated in studies and these have produced inconsistent results. Agnew, Dorn, and Eden (2004) investigated the FastForWord program with language-impaired children. The program uses modified speech but also includes some exercises on phonological skills and syntactic and semantic comprehension. Use of the program resulted in improved auditory discrimination ability, but did not transfer to better phonological skills. In contrast, Pokorni, Worthington, and Jamison (2004) did not find any intervention gains following FastForWord in comparison to Earobics and the Lindamood Phoneme Sequencing program after a 20-day summer program (three 1-hour sessions daily) in their dyslexic sample. Bischof et al. (2002) evaluated a computer program designed to teach tone and phoneme discrimination and found a significant correlation between auditory discrimination performance and orthographic skill following the intervention. In conclusion, whereas gains in basic auditory and language processing have been noted with basic auditory programs, the gains in reading skills have been inconsistent and need further study. For example, it remains an open question which aspects of reading skill are associated with basic auditory functions. Moreover the gains observed have not been as large as gains achieved following linguistic interventions, highlighting the need for critical evaluation of these additions for the treatment of dyslexia.

#### Interventions Targeting Sensorimotor Processing

Observations of dyslexic children who demonstrate poor sensorimotor coordination, poor postural stability, low tone in the upper body, and difficulties in a variety of skilled motor tasks (Alexander & Slinger-Constant, 2004) have been the basis for the development of the cerebellar theory by Nicolson et al. (2001). These authors have developed an exercise-based intervention program derived from the cerebellar theory which includes visuo-motor activities. This program was initially called DDAT (dyslexia, dyspraxia and attention-deficit treatment) but is now known as the Dore program (Dore & Rutherford, 2001). In a similar manner to other interventions of basic auditory and visual functions, the Dore program aims to treat the cause of the presented learning difficulty, which within the cerebellar theory is an under-functioning of the cerebellum. As part of the program participants engage in a broad variety of motor exercises such as dual tasking, throwing and catching of beanbags, and balance board exercises. The exercises are individually tailored, frequently monitored and adapted, and can be conducted at home for ten minutes twice a day over a period of six months to two years. The aim is to enhance cerebellar functioning.

Reynolds et al. (2003) assessed the efficiency of the Dore program and found significant benefits in cerebellar functioning for an intervention group compared to a nonintervention control group after a six-month intervention period. Benefits included gains in posturography (refers to the ability to keep a stable body balance), visual tracking, and in literacy-related functions such as reading, semantic fluency, and phonemic segmentation. The results of this study were controversial due to the researchers' affiliation with the Dore Company and methodological concerns (e.g., inclusion of non-dyslexic readers in the study, initial literacy imbalance between intervention and control group, Hawthorne effects as the
control group did not receive an alternative program) and five members of the editorial board of the journal *Dyslexia* resigned to protest against the publication (for various commentaries see McPhillips, 2003; Rack, 2003; Richards et al., 2003; Snowling & Hulme, 2003; Stein, 2003). A follow-up study by Reynolds and Nicolson 9was published 2007. Results of the follow-up study take the criticisms of the 2003 study into account by adjusting the methodological design and analyses. The follow-up study evaluated whether gains were maintained after 18 months and adjusted for initial group differences. Significant gains in motor skill, speech/language, phonology and working memory were still observed, indicating a long-lasting effect. However, gains in reading were reported to be small. Rack, Snowling, Hulme, and Gibbs (2007) criticised both studies stating, "We argue that the design of the study is flawed, the statistics used to analyse the data are inappropriate, and reiterate other issues raised by ourselves and others in this journal in 2003. Current evidence provides no support for the claim that DDAT is effective in improving children's literacy skills" (p. 97). Despite all criticisms of the Dore program, the International Dyslexia Association encourages future research to evaluate the program's efficacy (Peer, 2003).

# Interventions Targeting Lateralised Processing

Another intervention approach for dyslexia has been proposed and evaluated by Bakker (for a review see Bakker, 2006) who has developed the balance model of dyslexia (see Chapter 4). Bakker, Moerland, and Goekoop-Hoefkens (1981; see also Bakker & Vinke, 1985) utilised hemisphere-specific stimulation (HSS) to treat their proposed subtypes of dyslexia, the P-type dyslexic (slow but accurate and relying on right hemisphere processing) and the L-type dyslexic (fast but with many errors and relying on left hemisphere processing). The HSS program has a strong neurological basis assuming that stimulation of the left hemispheric for P-types and right hemispheric stimulation for L-types can help to minimise the impact of dyslexia. Using a HEMSTIM-program (specific computer software to accomplish the HSS; HEMSTIM, www.pits-online.nl, PITS, Leiden, Netherlands) words are flashed for a duration of not longer than 300 ms in either the left or right visual field to stimulate the right or left hemisphere selectively. The child is instructed to fixate the centre of the screen when the word appears. Although the visual HSS method is most commonly used, a tactile HSS method also exists, during which plastic letters are presented to the left or right fingers, to stimulate the right or left hemisphere, respectively.

Although this approach has not received much attention by academics the evaluations conducted by Bakker and colleagues (Bakker et al., 1981; Bakker, Bouma, & Gardien, 1990; Bakker & Vinke, 1985) suggested beneficial impacts of the HSS. The selection of dyslexic participants followed standard criteria such as average IQ and a reading lag of  $\geq$  1 year. The sub-classification of L- and P-types followed their error profile when reading texts. Selection criteria for the P-types was a larger number of fragmentation errors (e.g., word repetition, hesitations), and for the L-types a larger number of substantive errors (e.g., word mutilations, omissions, additions) on the text reading test (TRT; Van den Berg & Te Lintelo, 1977) in comparison to the group mean (the overall screened sample involved 174 subjects). Each session lasted 45 minutes and stimulation was applied once a week over 20 to 22 weeks. Differential reading results at post-test were obtained for L- and P-type Dutch dyslexic readers: L-types achieved improved word reading accuracy and comprehension and, as expected, P-types showed a faster reading rate. The neural changes following intervention are discussed in the last section of this chapter. Similarly Kappers (1997) reported outstanding intervention gains following HSS, but equally for both L- and P-type dyslexic readers, with 91% improving in text reading, and 55% of the children achieving a normal text reading level. The number of intervention sessions varied with some children receiving intervention for up to two years.

Studies in different languages have reported similar gains after HSS. A study conducted by Lorusso, Facoetti, Paganoni, Pezzani, and Molteni (2006) with Italian dyslexic readers compared the HSS to a phonics-based reading intervention and demonstrated superior gains for the HSS intervention group on measures of reading speed, reading accuracy, phonemic awareness and memory after four months of intervention twice a week. The authors suggested that apart from the strengthening of the neglected hemisphere, a more automatised processing due to the time pressure on information processing during stimulation (words flashing only for 300 ms) may be the underlying mechanism for the observed gains. Most of the studies reported support for the efficacy of the HSS on reading performance. However, more evaluation studies are needed to evaluate the significance of this addition to the intervention possibilities for dyslexia and more specifically research is needed to determine which intervention aspects of the HSS are related to which aspects of reading.

#### **Evaluation of Linguistic Interventions**

#### Interventions Targeting Phonological Processing

Alexander and Slinger-Constant (2004) reviewed the research supporting the efficacy of various interventions for dyslexia. They concluded that direct and systematic phonological awareness and phonics instruction intervention produced significant effects for at-risk readers as well as disabled readers and could close the gap for reading accuracy and often also for comprehension (e.g., Foorman et al., 1998; Lovett & Steinbach, 1997; Torgesen et al., 1997b; Vellutino et al., 1996). Apart from a few variations for the different phonological programs, most of them share the principle of explicitly teaching grapheme-phoneme correspondences, blending and manipulation skills. These skills are usually taught in an oral and written way simultaneously as this has been proved most effective (Beck, 2005).

Nevertheless, improvements in reading fluency and automaticity due to phonological intervention programs have not been reported often in children after Grade 2 (Shaywitz et al., 2008; Tijms & Hoeks, 2005; Torgesen et al., 2001). One possible explanation why the fluency gap cannot be narrowed by phonological interventions was offered by Snowling and Hulme (2005). By late primary school most children have developed a relatively large sight vocabulary, which means they can read words rapidly and automatically. In contrast, dyslexic children have limited sight vocabulary and need more repeated exposure to a word before it becomes part of their sight vocabulary. In addition, vocabulary increases rapidly for children after Grade 3 and an increasing number of lowfrequency words need to be learned. For dyslexic readers it is difficult to catch up, as they are still trying to memorise and automatise words they have learned earlier on. Another issue that has been stressed in only a small number of studies is the long-term effect of phonological intervention programs. In one study by Torgesen et al. (2001), which compared two intervention approaches, only about 40% of the participants sustained or increased their gains during a two-year follow up period.

In summary, the phonologically driven linguistic intervention studies indicate that the younger the child, the more explicit the intervention must be; the older the child and the more severe the impairment, the more intense the intervention and the longer its duration must be. A systematic phonics approach leads to robust results in word reading accuracy but it is not effective in developing fluency in more severely affected dyslexic readers after Grade 2 (Foorman, Breier & Fletcher, 2003; Torgesen, Wagner & Rashotte, 1997a). The following metaphor from Bakker (2006) emphasises the potential shortage of phonological interventions and leads us into the next section, which discusses the claims of combined intervention programs and the scientific evidence regarding their efficacy. "Imagine that a wheel of a farm cart breaks down, preventing the driver to continue his journey. Repairing the wheel would do. However, possibly the driver is aware of the fact that the road is very rough. Consequently, another breakdown may follow. The wheel is part of the cart, the road rather is subserving the cart. Phonological analysis similarly is part of the reading process. In case that is the whole story about reading and dyslexia, appropriate intervention of phonological processing might do. However, in case one or more subserving mechanisms appear to fail, it seems more appropriate to address these mechanisms in order to establish enduring improvement" (p. 11).

#### **Evaluation of Combined Interventions**

Bearing in mind that current research suggests a multidimensional deficit in dyslexia, a transfer of this theoretical approach results in a combined remediation method. Multidimensional interventions are believed to produce superior outcomes and establish more robust long-term effects than interventions that focus on one deficit. Many combined programs (e.g., Alphabetic Phonics, Project Read, the Spalding Approach, the Herman Approach, the Wilson Approach; Alexander & Slinger-Constant, 2004) are based on the Orton-Gillingham method, which has been the forerunner in the field. This is a multisensory explicit phonics method with emphasis on visual and auditory feedback for sounds and on tactile-kinesthetic input of letter formation. Unfortunately, only a few methodologically sound studies exist to validate its efficiency (for a review see Alexander & Slinger-Constant, 2004). The few studies that have evaluated the Orton-Gillingham approach have reported gains in phonological decoding, word-level reading, and comprehension (Maskel & Felton, 2001) as well as in word identification (Oakland et al., 1998).

As reported in the previous section (linguistic intervention programs) a common research finding is that reading rate remains unaffected by intense intervention. Thus, some researchers have endeavoured to improve reading fluency by combining phonological programs with explicit fluency interventions. In a study by Torgesen et al. (2003) the "Spell, Read Phonological Auditory Training" (MacPhee, 1998), which combines fluency-practice and phonological instruction, was utilised to close the fluency gap for mildly (30<sup>th</sup> percentile), moderately (10<sup>th</sup> percentile), and severely (2<sup>nd</sup> percentile) slow readers. Significant gains in fluency after the intense intervention (between 50 to 100 hours) were reported for the mildly and moderately slow readers, but not for the most severely slow readers. Moreover, the children in the moderate group still remained below average with a standard score of 79 at post-test compared to 65 at pre-test. Similarly, Denton et al. (2006) evaluated eight weeks of phonological intervention, followed by eight weeks of fluency intervention in a group of persistently reading-disabled children who had not benefited from previous interventions at their schools. The findings showed significant improvements in decoding, fluency, and comprehension. However, as for the Torgesen et al. (2003) study, the students did not achieve average fluency levels. The authors also pointed out that the gain in fluency could not be attributed to the fluency program alone, because it was always conducted following the phonological program, thus leaving the possibility of an accumulative effect.

Another relatively new combined intervention approach is the RAVE-O (Retrieval, Automaticity, Vocabulary, Evaluation, Orthography) program by Wolf, Miller, and Donnelly (2000), which has been derived from the double-deficit theory (see Chapter 4). The double-deficit theory claims the existence of three subtypes of dyslexia: One with a phonological deficit, one with naming deficits and a mixed type having both and consequently being more severely affected. The RAVE-O incorporates training in reading fluency (e.g., repeated reading of connected text), phonological skills (e.g., phonological analysis and blending), and automaticity in underlying component skills (e.g., left to right scanning of letters at different temporal rates) and was initially developed to treat the naming subtype and mixed type. In contrast to many other intervention programs, the RAVE-O also integrates motivational and emotional aspects in the intervention to change children's perception towards a more positive attitude of themselves as language learners, which in turn encourages greater risk-taking during reading challenges. The RAVE-O is conducted in 70 one-hour sessions over half a year.

Studies evaluating the RAVE-O program are rare. A pilot study by Wolf and Segal (1999) on an earlier version of the RAVE-O found significant improvement in measures of word retrieval accuracy and vocabulary depth in a dyslexic sample with naming deficits. However, the study did not include an untreated control group. A case study by Deeney, Wolf, and O'Rourke (2001) conducted the RAVE-O program with a student who had only a rapid naming deficit and reported marked improvement in naming speed and phonological skills. However, as Vukovic and Siegel (2006) pointed out, the student also appeared to have weak phonological skills as he could identify only 10 out of 25 rhyme patterns. Further, due to the integrated nature of the RAVE-O program it cannot be determined whether the training gains in naming speed tests could be attributed to the naming speed aspect of the training, the phonological aspect, or the combination of both. An intervention study by Lovett, Steinbach, and Frijters (2000a) classified dyslexic children into the three types according to the double-deficit theory, and compared a phonological intervention, word identification intervention, and a control program (study skills). All subtypes achieved significant gains, mainly in phonological skills and word reading, in response to the two interventions. These results question the double-deficit theory as the children in the naming and double-deficit group made phonological gains after the intervention. In conclusion, according to Vukovic and Siegel (2006) it is difficult to identify children with naming deficits who do not have co-occurring phonological problems and who do not benefit from phonological interventions. More studies are needed to demonstrate the additional or

superior efficacy of the RAVE-O compared to other intervention programs, in particular for the proposed naming deficit only subtype. Regardless of the type of combined intervention, with regard to fluency intervention effects, Shaywitz et al. (2008) noted that a crucial element of successful intervention is the need for scaffolding support from parents and peers. That is, the more reading practice is undertaken, the more likely the improvements in fluency will endure.

Another recently developed integrated approach is the Cellfield intervention, which takes into account the possibility that dyslexia might be attributable to a multidimensional deficit. The Cellfield Intervention was developed by Caplygin (2001) and involves 10 computer-based activities designed to remediate multiple deficits concurrently. The intensive intervention emphasises three deficits of dyslexia, namely phonological, visual, and visual to phonological processing. The development of the Cellfield intervention has been influenced by various theories on dyslexia, including the visual and auditory temporal theories, phonological theory and their neurobiological correlates. For instance the magnocellular theory for the auditory and visual system (see Chapter 4) and its neural correlates (Galaburda & Livingstone, 1993) suggested reduced auditory and visual processing speed in dyslexic readers. These theories led to the integration of visual motion graphics and aural modified speech into the intervention (for details on the Cellfield exercises see Chapter 8), designed to alter visual and auditory processing through a visualto-auditory-bonding strategy. In addition, the theories on visual eye movement control have influenced the development of the intervention in that red coloured lenses and monoocclusion (patching of one eye) are used for children showing eye movement difficulties (Stein et al., 2000b). Finally, the phonological hypothesis has had a broad impact and resulted in the inclusion of exercises aimed at strengthening grapheme-phoneme correspondences and sound segmentation ability (Castles & Coltheart, 1993; Ehri, 2002). Recently the Cellfield Company has developed a follow-on training program targeting reading fluency that is conducted for three weeks (flexible) with a trained tutor at the Cellfield clinics. This training aims to strengthen any gains made following the computer sessions.

To date, the Cellfield intervention (computer sessions) has been evaluated in only one published paper. This evaluation, conducted by Prideaux et al. (2005) in a clinical setting associated with the Cellfield Company, showed outstanding intervention gains. Significant gains were made in all three sets of dependent variables analysed (readingrelated skills, oral reading proficiency and ocular measures) providing support for the Cellfield intervention. Follow-up measurements were not conducted. The following project aimed to provide a preliminary critical evaluation of the potential contribution of the Cellfield intervention to the field of interventions for dyslexia. The project was conducted independently of the Cellfield clinic and further aimed to assess potential follow-up benefits of the Cellfield intervention three weeks after its completion.

#### Beyond the Intervention: Other Variables that Influence Intervention Outcomes

Gains following an intervention program depend on a variety of variables in addition to the intervention itself. A meta-analysis conducted by Swanson and Hoskyn (1998) on intervention research reported the following additional methodological impacts on effect sizes of intervention outcomes: Larger effect sizes were revealed when (1) different teachers administered the control and intervention program, (2) the intervention took place in resource rooms rather than in normal classrooms, and when intervention and control groups participated in different rooms, instead of the same room (3) studies used experimental measures rather than standardised measures, (4) studies had a smaller sample size (25 or less) rather than a larger one (25 to 100). Additional methodological influences have been reported by the US National Reading Panels (Langenberg et al., 2000; see also Snow et al., 1998) who have published a comprehensive report on efficient reading instructions and influencing factors. For instance, phonological awareness programs of between five and 18 hours total instruction were more beneficial than shorter or longer programs and larger gains were observed when the program included one to two aspects instead of three or more aspects of phonological awareness intervention simultaneously.

Besides these methodological variables, research has shown that socio-demographic variables such as low socioeconomic status (SES) affect reading gains negatively. For

example, Phillips, Noppeney, Humphreys, and Price (2002) reported that low SES also correlates with low language input at home, impacting on a child's speech production and vocabulary (see also Hart & Risley, 1992). A similar relationship has been identified between the home literacy environment and SES: Families from lower SES background seem to engage in practices such as shared reading with their children less often and this impacts negatively on their early literacy skills (Baker, Fernandez-Fein, Scher, & Williams, 1998, Dunning, Mason, & Stewart, 1994). Snowling (2000) reported that the more print exposure a child had the greater gain he/she could achieve from interventions. Family beliefs and values have also been shown to influence a child's academic achievement in reading and maths, even after influences of income, ethnicity, and parental education were controlled (Phillips et al., 2002). Snowling (2000) emphasised the importance of considering the child's family background to maximise intervention gains, because any intervention needs to transfer to the more natural context the child is interacting with, at home and at school. Al Otaiba and Fuchs (2006) reported in their study on non-responsiveness to intervention that those children who have been assigned to previous special education are more likely to be non-responsive to another intervention than those who have not engaged in intervention before.

Various child characteristics have also been shown to influence intervention outcomes in various intervention studies. These characteristics include (1) age, (2) verbal ability, (3) rapid naming skills and other reading-related components such as phonological awareness, initial reading and spelling level, and (4) motivational, behavioural and emotional characteristics (Al Otaiba & Fuchs, 2006; Snow et al., 1998; Wolf, Bally, & Morris, 1986). Whereas gender did not impact on intervention outcomes as indicated in the meta-analysis conducted by Swanson and Hoskyn (1998), a large body of research has confirmed the impact of the child's age on intervention gains, with younger children benefiting more from interventions than older children, especially for phonology-based programs (e.g., Snowling, 2000; Shaywitz et al., 2008). A deficit in rapid naming seems to mark a deficit in the rate of learning and a higher verbal IQ seems to facilitate, in particular, benefits in reading comprehension after intervention (Snowling, 2000). Additionally, as reported by Tijms and Hoeks (2005), the initial level of reading and spelling impacted on the intervention outcomes, with greater progress for those having larger pre-test deficits (see also Snowling, 2000). Tijms and Hoeks reported no effects of initial phonological awareness on intervention outcomes, but in contrast, Snowling in her review on intervention studies stated that the better developed the phonological awareness, the greater the gain (see also Al Otaiba & Fuchs, 2006). Taken together, these findings suggest that differences in learning to read initially may account for some individual differences in response to interventions (Crain-Thoreson & Dale, 1992).

With regard to emotional and motivational aspects, Casey, Brown, and Brooks-Gunn (1992) investigated the relationship between reading impairment and emotional health and found that reading-disabled children had a lower positive well-being score and a higher anxiety score than their control age-matched peers. This stresses the importance of integrating these aspects into interventions to help to prevent negative impacts on the children's self-esteem. Positive correlations between behavioural problems (e.g., conduct problems and antisocial behaviour) and learning difficulties have also been implicated. Beichtman and Young (1997) stated that it is not established whether conduct problems develop secondarily to learning difficulties (e.g., Grande, 1988) or are a primary cause of the development of learning issues (e.g., Larson, 1988). In the evaluation study of the Cellfield intervention (Prideaux et al., 2005) the authors suggested that the child's motivation might have influenced the intervention outcomes. Clinicians working with the children during the intervention program reported lower participant motivation at the beginning of the intervention than by the middle of the sessions. However in this study the children's motivational characteristics were not directly assessed. Research on the relationship between motivational characteristics and academic outcomes indicates that early problems in learning to read and spell are related to motivational-emotional vulnerability in learning situations in the school context (Poskiparta, Niemi, Lepola, Ahtola, & Laine, 2003). Research has also shown that the child's motivation to read can be influenced by reading instruction programs (Wigfield, Guthrie, Tonks, & Perencevich, 2004). However the relationship between intrinsic motivation to read, self-efficacy and selfesteem and academic outcomes of the child remains an open question and needs further study.

# Neural Changes following Intervention for Dyslexia

In relation to intervention studies, neuroimaging and electrophysiological measures have been used in combination with behavioural measures to develop an understanding of the neurobiological responses to interventions. Those studies are rare, and involve different types of intervention, making comparisons across studies speculative. Some interventions have targeted basic cognitive functions. For example, Temple et al. (2003) evaluated the FastForWord program, which aims to strengthen tone and phoneme discrimination skills. They included the measurement of neural activity before and after the intervention of dyslexic readers compared to an untreated non-dyslexic control group. The results showed increased activity in the left temporal-parietal cortex and left frontal gyrus, alongside significant improvements in oral language and reading for dyslexic readers. However, overall neural changes in the dyslexic group were widespread and even the brain activity patterns of some controls changed. Noble and McCandliss (2005) also noted that there was no reading impaired control group.

Simos and colleagues (2002) used magnetic source imaging to evaluate changes in spatiotemporal brain activation profiles after a phonological based intervention. The eight dyslexic and eight control children performed a pseudoword reading task during the brain scanning, which was conducted before and after the intervention. In contrast to controls, dyslexic readers showed little or no left temporal activation prior to the intervention during the pseudoword reading. After intervention the cortical activation patterns of the participants with dyslexia resembled much more closely those of the normal controls, with increased activity in the left superior temporal gyrus in dyslexic readers. Alexander and Slinger-Constant (2004) questioned the validity of these findings as six out of the eight participants also had ADHD and were on medication. However, a study by Shaywitz et al. (2004) confirmed the findings of Simos et al. and further showed that within their dyslexic sample the neural changes were larger than the reading-related outcomes.

The dyslexic readers studied by Aylward et al. (2003) engaged in two hours of intervention a day for two weeks. The program included intervention in linguistic awareness, alphabetic principle, fluency, and reading comprehension. A standardised measure of reading performance as well as two experimental tasks, a phonological and a morphological task were performed before and after the intervention. The fMRI results for dyslexic readers at pre-test indicated overall less brain activation, engagement of distinct brain regions during the two language tasks, and a more right focused or bilateral activation compared to non-dyslexic controls. Following intervention, dyslexic readers showed higher overall activation with more left-focused processing. The authors concluded that these findings demonstrate the plasticity of the brain. The dyslexic readers' reading performance also improved significantly from a standard score of 87 to 97, as measured by the Woodcock Reading Mastery Test. Noble and McCandliss (2005) criticised the fact that the control group also showed changes in brain activity during the second scanning, with overall decreased brain activation. Another criticism is that the study did not include an untreated dyslexic control group.

More specific changes were achieved by the HSS method in various studies by Bakker and colleagues (Bakker et al., 1990; Bakker et al., 1981; Bakker & Vinke 1985) where specific hemisphere stimulation resulted in increased activity in the left (with right hemispheric stimulation) and right (with left hemispheric stimulation) hemisphere as measured by the P250 amplitude of the ERP for P-type and L-type Dutch dyslexic readers respectively, compared to controls. However results of studies showing that dyslexic readers sometimes had enhanced activity of the non-stimulated hemisphere question the specificity of the intervention (Dryer, Beale, & Lambert, 1999; Grace & Spreen, 1994; Kappers, 1997). Future studies are needed to test whether the left or right single hemispheric stimulation is a crucial factor in the intervention or if any stimulation left, right or of both hemispheres would achieve brain activity changes. Within the auditory domain Santos, Joly-Pottuz, Moreno, Habib, & Besson (2007) have investigated auditory ERP-related changes following an intervention for temporal processing in a sample of dyslexic readers. Before the intervention dyslexic readers had a significantly smaller late positivity (P3) than controls in response to aurally presented incongruent sentence endings. Following intervention the dyslexic readers showed significantly increased P3 amplitude, resembling the pattern of the normal readers.

In summary, the few existing physiological findings generally indicate a reorganisation of functional brain activity following intensive intervention (see also Richards et al., 2002) and are a promising addition to the field of intervention studies. Shaywitz et al. (2008) noted that "still to be determined is the precise relationship among the type of intervention, changes in brain activation, and clinical improvement in reading" (p. 459).

# Summary: Intervention Studies and Their Outcomes

In summary, a large body of evidence exists to support the efficacy of direct and explicit training of phonological skills for the remediation of dyslexia. Gains in phonological skills, reading accuracy, and comprehension have frequently been reported. In contrast, pure phonological programs have less often led to transfer to reading fluency skills. Theories on dyslexia highlight the fact that dyslexia is a heterogeneous learning difficulty. Thus, interventions targeting various deficits concurrently associated with dyslexia are a promising addition to the field and are supported by recent empirical evidence. In contrast to the phonological and combined programs, the efficacy of interventions targeting basic auditory, visual, and sensorimotor processing is yet to be reported. In addition to improvements in reading skills, the neurobiological evidence to date suggests reorganisation of the brain following intensive intervention, which gives some indication of the plasticity of the brain. Equally important for all intervention programs are the findings from research on potentially influential variables other than the intervention itself. These include instructional-methodological variables (e.g., intervention setting, intervention administration), socio demographic variables (e.g., socio-economic status, home literacy environment) and child characteristics (e.g., age, verbal ability, rapid naming ability, initial level of reading and spelling, phonological awareness, behavioural, emotional and motivational aspects). In conclusion, the science of intervention studies is increasing and has already enlightened our understanding of the impact of various intervention approaches. Nevertheless, the more interventions are evaluated in scientific studies, the more likely they will have an impact on educational practices and become a useful information source for parents, teachers, and educational practitioners.

#### **Chapter 7: Rational and General Aims**

Dyslexia is one of the most common learning difficulties in society and is especially common in English-speaking countries (Shaywitz, 1990). For this reason intervention research in this area is of great social relevance – a person needs to develop reading and spelling skills to be able to lead a "normal" life in our society. By examining behavioural measures (reading and related skills) as well as psychophysiological measures (latencies and amplitudes of ERP components P2, N4 and LPC, and reaction time and accuracy data) in response to an intervention; the Cellfield intervention program, the study aims to investigate the behavioural and psychophysiological concomitants of this commercially available intervention for dyslexia. The Cellfield intervention aims to assist the dyslexic individual through concurrent treatment of visual, phonological, and visual to phonological deficits in 10 one-hour computer sessions.

To date, the Cellfield intervention has been evaluated in one study (Prideaux et al., 2005) and significant gains in reading comprehension, accuracy, and nonword reading were reported following the intervention. However, the study included non-dyslexic and dyslexic children, the age range was broad (range seven to 17 years), no control/placebo group was included, the evaluation was conducted in co-operation with Cellfield clinics, and no follow-up assessment to investigate maintenance of gains was conducted. As recommended by Prideaux et al. (2005) the present study will be conducted independently from the Cellfield clinic and dyslexic participants will be chosen carefully on a broad range of tests to include only those who actually present with reading/spelling difficulties. The age range will be kept to a minimum (12 to14 years) to avoid developmental confounds with potential intervention gains. Moreover, a placebo intervention, conducted for the same duration as the Cellfield intervention, will be implemented to control for Hawthorne effects. Furthermore, the intervention will be offered free of charge, to exclude monetary investment as a motivational factor to "do well" on the program. Participants will be randomly assigned to the Cellfield or Placebo group.

The outcomes of the Cellfield and Placebo program will be primarily evaluated using various literacy tests to assess reading, spelling, and phonological skills. Group outcomes before and after engagement in the Cellfield and Placebo program respectively are expected to produce group differences, with the Cellfield group showing gains in the literacy measures, at post- compared to pre-test. In line with the results of the Prideaux et al. study we expect gains in particular for phonological skills (nonword reading), text reading accuracy, and comprehension. As in the Prideaux et al. study, we may also expect reading rate to drop from pre-to post-test for the Cellfield group. Prideaux et al. inferred that the slower reading rate is due to a speed-accuracy trade-off, indicating the application of phonological decoding to read more accurately. With regard to spelling skills, we may expect smaller gains than for reading and phonological measures at post-test for the Cellfield group. Similarly Prideaux et al. found significant but small gains for spelling skills in their sample following the Cellfield intervention. No changes in these literacy measures are expected for the Placebo group.

The study will also investigate potential follow-up gains of the Cellfield intervention after a three-week follow-on practice program (involving spelling, reading fluency, and comprehension practice). Immediately after completion of the 10 Cellfield and 10 Placebo sessions, participants from both groups will participate in the three-week followon practice. This design was chosen to evaluate the impact the Cellfield intervention may have on literacy skills immediately after the 10 sessions and also to determine whether follow-on practice will impact on the Placebo group and further impact on gains for the Cellfield group. It is hypothesised that the Cellfield group will maintain or increase any literacy gains from post-test after the follow-on practice, that is improved reading, phonological, and to a smaller extent spelling skills. With regard to reading fluency we may expect the Cellfield group to increase their fluency at follow-up, after an initial decrease at post-test. The Placebo group may show some gains in the literacy measures after the followon practice, in particular in text reading accuracy, comprehension, rate, and spelling. However it is also hypothesised that the Cellfield group will show superior gains to the Placebo group due to the cumulative effect of the multi-modal Cellfield intervention and follow-on practice.

Motivation to read and locus of control are motivational aspects of academic achievement that have been related to learning difficulties in previous research (Gambrell, Palmer, Codling, & Mazzoni, 1996; Hinshaw, 1992; Poskiparta et al., 2003; Wigfield & Guthrie, 1997). Furthermore it has been reported that reading skills and reading motivation have a bidirectional relationship and thus targeting both may result in greater gains for poor readers (Morgan & Fuchs, 2007). Although the Cellfield intervention does not target reading motivation per se, we aim to investigate the influence motivational aspects have on training outcomes, by assessing reading motivation and locus of control at pre- and post-test. In line with previous research (e.g., Butkowsky & Willows, 1980), at pre-test we expect both groups to have a relatively low reading motivation due to their ongoing literacy difficulties and a relatively higher external locus than internal locus of control. It is expected that the Cellfield group will show increased motivation to read and internal locus of control at posttest. In contrast the Placebo group will not change with regard to their reading motivation and locus of control.

The study also aims to evaluate the potential impacts of the Cellfield intervention and the follow-on practice at a neural level using ERPs (P2, N4 and LPC). ERP experiments targeting lexical, phonological (single-word-level tasks), and sentence processing (sentencelevel task) will be conducted before, immediately after and three weeks after the Cellfield/Placebo program. Imaging and ERP studies of dyslexic brain activation have shown deficient language processing in the left hemisphere in dyslexic children and adults (e.g., Ackerman et al., 1994; Breznitz & Meyler, 2003; for reviews see Goswami, 2004; McCandliss & Noble, 2003; Shaywitz, 2008; Zeffiro & Eden, 2000). Of particular importance for the current study were the findings from those few intervention studies, which have also evaluated neural changes following interventions. Initial findings of these studies suggested that powerful interventions with dyslexic children do produce a normalised localisation and timing of brain functions that support reading and phonological processing in the brain, in particular a larger engagement of the left hemisphere (Aylward et al., 2003; Shaywitz et al., 2004; Simos et al., 2002; Temple et al., 2003). A few ERP intervention studies have been conducted and indicate enhanced amplitudes following interventions for dyslexia (Santos et al., 2007), which were left lateralised in some studies (e.g., Bakker et al., 1990). Larger ERP amplitudes in particular in the left hemisphere have been previously associated with more specific activation strength when processing linguistic material (e.g., Miles & Stelmack, 1994; Licht, Bakker, Kok, & Bouma, 1992; Penolazzi et al., 2006).

The Cellfield intervention has not been evaluated with a neurophysiological technique, and thus the present study will be the first to report any potential changes in neural markers following the Cellfield intervention. The findings of imaging and ERP studies showing that dyslexic readers often had bilateral or a larger right than left activation during linguistic processing and that neural changes following intervention were often observed in the left hemisphere led to the following general hypotheses: For ERP amplitudes and latencies, at pre-test we expect either bilateral activation of the hemispheres, so we predict either no pre-test differences in amplitudes/latencies, or larger amplitudes and longer latencies right than left at pre-test for both groups. At post-test we expect that the Cellfield group will show increased amplitudes and longer latencies in the left hemisphere following the intervention, which may be accompanied by a decrease in amplitudes and shorter latencies for respective components in the right hemisphere, commonly referred to as the 'normalisation hypothesis' in the imaging intervention literature (e.g., Aylward et al., 2003; Temple et al., 2003). We anticipate that the Placebo group will not show these changes at post-test. At follow-up we expect that the Cellfield group will maintain left focussed processing, with larger amplitudes and longer latencies, and decreasing activity in the right. If no changes in amplitude or latency are observed at post-test for the Cellfield group, first changes may emerge at follow-up-test.

Our final hypothesis for the neural changes will be referred to as the 'linguistic specificity hypothesis'. Two of the experimental tasks (lexical and phonological task) require single-word processing and one task (sentence task) requires ongoing word processing in a sentence task paradigm. The single-word-level tasks may show the proposed changes in ERP amplitudes and latencies to a greater extent than the sentence task, as they are less complex. Within the two word-level tasks, the phonological task may show larger changes as the Cellfield intervention has a strong phonological component.

In addition, within each of the three tasks, two stimuli types are included, that is (1) incongruent versus congruent sentence completions for the sentence task, (2) real words versus pseudo homophones for the lexical task, and (3) pseudo homophones versus nonwords for the phonological task. Previous research with normal reading controls has indicated that ERP amplitudes and latencies discriminate the linguistic features of these stimuli. For example, the incongruent sentence completions commonly evoke a N4 amplitude of much larger magnitude than congruent completions (for a review see Kutas et al., 2006) and some studies have found N4 amplitudes of similar magnitude for incongruent and congruent sentence endings for dyslexic readers compared to controls (e.g., Brandeis et al., 1994).

For the lexical and phonological task, research has indicated larger N4 amplitudes towards pseudo homophones and real words compared to nonwords, which elicit little or no N4 activity in normal-reading samples (for a review see Kutas et al., 2006). At pre-test we expect the investigated dyslexic sample to be less sensitive in detecting and discriminating these linguistic features of the presented stimuli for all three tasks, showing similar N4 amplitudes and latencies in response to the different stimuli types. At post-test, following the Cellfield and Placebo program respectively, we expect that the Cellfield group will show diverging amplitudes and latencies for the different stimuli types, resembling more closely the activation pattern found in normal-reading samples. In contrast the Placebo group will show no differences. At follow-up-test the increased ability of the Cellfield group to discriminate among the linguistic features of the different stimuli types will be maintained or more pronounced.

Reaction time and accuracy data for the experimental tasks will be obtained, allowing investigation of the relationship between any neural changes and behavioural outcomes. As a general hypothesis we expect the following effects for reaction time: The Cellfield group will have a longer reaction time at post- and follow-up- compared to pre-test as the Cellfield group is expected to apply phonological decoding to perform the experimental tasks which would require a longer processing time. For all three experimental tasks the response time period is limited. If phonological decoding skills are strengthened through the Cellfield intervention and the Cellfield group starts using these skills at post-and follow-up-test, then more or the same missing responses and subsequently a lower or the same accuracy are expected. No changes in task performance are expected for the Placebo group from pre- to post-test, however after engagement in the follow-on practice some changes may occur from post- to follow-up-test. The changes in task performance for the Placebo group at follow-up-test may be distinct from the anticipated changes for the Cellfield group as the Placebo group will not have completed the Cellfield intervention.

In sum, the purpose of the current study is to provide an independent evaluation of the Cellfield intervention for dyslexia and for the first time to integrate neural indicators as outcome measures. We hypothesised that the multi-modal Cellfield intervention will improve reading and related skills as well as the development of a more left-lateralised linguistic processing associated with skilled reading. To examine these hypotheses we investigated a sample of 12 students presenting with reading/spelling difficulties, with seven students assigned to the Cellfield intervention and five to a placebo program. Literacy and ERP measures were assessed before, immediately after, and three weeks after the completion of the Cellfield/Placebo program.

#### **Chapter 8: Method**

#### **Participants**

An initial sample of 170 Grade 7 students was screened for literacy difficulties with a nonword reading test (Martin & Pratt, 2001), an irregular word reading test (Coltheart & Leahy, 1996), and a test of non-verbal cognitive abilities (Standard Progressive Matrices, Raven, 1938) at two Tasmanian high schools (see Materials section for details on screening and other tests). Previous research strongly indicates phonological deficits as one of the major problem areas associated with dyslexia. Students invited to participate further (n=15) were those who scored at least one *SD* below the average of their age group on the nonword reading test and who also obtained a non-verbal intelligence standard score between 85 and 115. Students were then randomly assigned to the Cellfield group (n=8) and Placebo group (n=7). All were native speakers of English.

The second selection criterion was the indication of a mild, strong or very strong risk of dyslexia according to the Dyslexia Screening Test- Secondary (DST-S, Fawcett & Nicolson, 2004). The DST-S was chosen for compatibility with the previous evalaution study by Prideaux et al. (2005) and because it screens for a variety of skills associated with dyslexia. One student, initially assigned to the Cellfield group, did not meet the criterion, displaying a 'not-at-risk' index according to the DST-S. The student remained in the study, as she displayed a selective impairment in reading rate (a very slow, but accurate reader) according to the Neale Analysis of Reading Ability (Neale, 1999), but was excluded from all analyses. Screening for gross behavioural problems was conducted with the Child Behavioural Checklist (CBCL, Achenbach, 2001) and children with clinical problems on any of the eight syndrome scales (see Materials) were excluded from the study. Two students, initially assigned to the Placebo group, had a diagnosed co-morbidity, one student with ADHD and the other one with Asperger syndrome. Both students completed the study, but were not included in any analyses. Of the 12 remaining participants (aged 12 to 14 years; five female and seven male; two left- and 10 right-handed), seven were in the Cellfield group (three females and four males), and five in the Placebo group (two females

and three males). None of these participants displayed any major medical conditions and all had normal or corrected-to-normal vision. The Cellfield and Placebo group completed the study over a six-month period (Terms 2 and 3 of the school year). Following the completion of the study, participants in the Placebo group were offered the opportunity to complete the Cellfield intervention, and all accepted.

Means and standard deviations of participants' characteristics or scores on all initial screening measures for each group at pre-test are presented in Table 2. The range of raw scores on the nonword test is 0 to 54, 0 to 30 for the irregular word test, and 0 to 60 for the IQ test, with higher scores representing higher ability in respective tests. Primary standard scores (M=100, SD=15) were also obtained for these measures. SES was assessed using the ANU-4 scale (Jones & McMillan, 2001), ranging from 0 to 100, with 100 representing the highest possible score. It should be noted that information on the three parental variables: known family history of dyslexia, reading home environment, and parental reading habits, was obtained using a parental questionnaire (see Materials for details) developed for the purpose of the study. The interpretation of raw scores was based on the researcher's coding of the questions. For family history of dyslexia, the higher the raw score, the higher the occurrence of dyslexia in the family; the range of possible scores is 2 to 6. For reading environment, the higher the raw score, the more stimulating the reading environment; the range of possible scores is 5 to 21. Finally, for parental reading habits, the higher the raw score, the more positive the parents' own reading engagement; the range of possible scores is 3 to 9.

For the literacy screening measures, nonword and irregular word reading, both groups had by definition a standard score of at least 1 *SD* below the mean, indicating a weakness in phonological decoding and irregular word recognition. Both groups were of average non-verbal intelligence as assessed by the Standard Progressive Matrices Test. With regard to the socio-demographic information, the parental background of both groups can be placed in the middle range of socio-economic status according to the ANU-4 scale.

Preliminary *t*-test analyses for independent samples performed on mean raw scores for the pre-test measures showed no significant differences between the Cellfield and the

Placebo group for age, t(10) = -0.95, p = 0.37, non-verbal IQ, t(10) = -0.12, p = 0.90, nonword reading, t(10) = -0.22, p = 0.83, irregular word reading, t(10) = -0.53, p = 0.61, or socio-economic background, t(10) = 0.68, p = 0.52.

## Table 2

Mean Pre-Test Screening Raw Scores and Standard Scores for Cellfield and Placebo Group

|                                      | Cellfield (n=7) |      |       | Placebo (n=5) |       |      |       |      |
|--------------------------------------|-----------------|------|-------|---------------|-------|------|-------|------|
|                                      | RS              |      | SS    |               | RS    |      | SS    |      |
| Variable name                        | M               | SD   | M     | SD            | М     | SD   | M     | SD   |
| Age*                                 | 12.99           | 0.42 |       |               | 13.23 | 0.47 |       |      |
| Nonword Reading                      | 18.29           | 4.23 | 76.20 | 4.89          | 19.00 | 6.89 | 76.13 | 9.32 |
| Irregular Word Reading               | 18.00           | 5.07 | 81.51 | 11.12         | 19.40 | 3.51 | 83.97 | 9.33 |
| Non-verbal IQ                        | 37.57           | 2.76 | 92.71 | 6.24          | 37.80 | 3.70 | 92.40 | 9.24 |
| Socio economic status <sup>1</sup>   | 38.35           | 8.59 |       |               | 34.57 | 5.86 |       |      |
| Family history dyslexia <sup>1</sup> | 3.14            | 0.90 |       |               | 2.25  | 0.50 |       |      |
| Reading environment <sup>1</sup>     | 12.42           | 3.26 |       |               | 12.25 | 3.59 |       |      |
| Parental reading habits <sup>1</sup> | 6.14            | 2.19 |       |               | 6.25  | 0.96 |       |      |

RS= Raw score; SS= Standard score; \*= Age in years; 1= SS were not available for these measures

One participant from the Placebo group did not return the parental questionnaire and thus his/her data could not be included in the analysis, leaving n=7 participants for the Cellfield group, and n=4 participants for the Placebo group. As can be seen in Table 2, the mean scores of both groups reveal a relatively small known family history of dyslexia. Secondly, both groups show a medium level of scores in terms of a stimulating reading environment. Thirdly, both groups show raw scores reflecting a medium level of positive reading engagement of the parents. Mann-Whitney U-tests were conducted on these three measures of the parental questionnaire: No significant effects of known family history of dyslexia,  $U(n_1=7, n_2=4) = 6.00, p= 0.13$ , home reading environment,  $U(n_1=7, n_2=4) = 12.50, p= 0.78$ , or parental reading habit,  $U(n_1=7, n_2=4) = 13.50, p= 0.92$ , were observed between the groups, indicating that the two groups did not differ with respect to their family reading environment, known family history of dyslexia or the parents' own reading habits.

Parents were also asked to report if their child had completed any previous interventions for learning difficulties. Within the Placebo group, three out of four participants had not engaged in intervention for learning difficulties. Similarly five out of seven participants from the Cellfield group did not report previous intervention experience. Those who reported previous intervention experience had engaged in literacy programs at school. A chi-square analysis applying Fisher's exact test showed no significant effect of previous intervention experience between the groups,  $\chi^2(1, N=11) = 0.13$ , p= 0.38.

### **Materials**

## **Initial Screening Tests**

Nonword Reading Test: Phonological Decoding Skills. The Nonword Reading Test (Martin & Pratt, 2001), a standardised measure of phonological decoding, requires the participant to read aloud pronounceable nonsense words increasing in difficulty. Raw scores were also converted into primary standard scores to allow the placement of each participant's performance with respect to his/her age peers. Norms are provided for ages 6 to 16 years. Kuder-Richardson internal reliability for Form A was reported at .96 for 12-0 to 13-11 year-olds, and for Form B, at .95. A high test-retest reliability of .96 (Form A), and .95 (Form B) was stated.

*Irregular Word Test: Irregular Word Reading Skills.* The Irregular Word Test (Coltheart & Leahy, 1996), which measures irregular word recognition skills, consists of 30 exception words which increase in difficulty. These words are read aloud by the participant. Although no test manual exists, Coltheart and Leahy provided some normative data based on a sample of 420 Australian children, aged 7 to 12 years. Raw scores were assigned to Band A (lowest score; more than 2 *SD* below the group mean) and Band B (up to 2 *SD* below the group mean) for each age. Alexander and Martin (2000) provided more recent normative data for the Irregular Word Test based on a sample of 863 Tasmanian participants, aged 6 to 15 years, allowing raw scores to be converted into age-normed standard scores, which were utilised in the current study.

*Standard Progressive Matrices: Non-verbal Intelligence.* The Standard Progressive Matrices Test (Raven, 1938) consists of matrix patterns which measure spatial abilities and provides an estimate of non-verbal intelligence. The participant has to complete patterns printed in the test booklet by choosing one out of six to eight possible missing pieces. There are five test sets increasing in difficulty, and for the timed administration used in the current study, time is limited to complete each of the five sets, with a total time of 20 minutes. Splithalf reliability is reported at .91 in the manual. Australian norms from 1989 for Grades 7 and 8 were used (de Lemos, 1989a, 1989b).

*Child Behavioural Checklist: Gross Behavioural Problems.* The Child Behavioural Checklist (CBCL, Achenbach, 2001) consists of two major sets of scales: the Competence scales (including the activities, social and school scales) and the Syndrome scales (including the anxious/depressed, withdrawn/ depressed, somatic complaints, social problems, thought problems, attention problems, rule-breaking behaviour, aggressive behaviour, and other problems scales). Parents estimate the presence of the listed problems for their child. Cronbach's alpha reliability is reported to range from .63 to .79 for the competence scales, and from .78 to .97 for the syndrome scales. Normalised *T*-scores were derived from the raw scores. Separate norms exist for males and females, ages 6 to 18 years.

*Medical Questionnaire*. A medical questionnaire was used to screen for medical (e.g., physical conditions, medication), neurological (e.g., brain injuries, epilepsy) and hearing/vision problems (Appendix A). Parents were required to complete the form.

*Parents' Questionnaire.* This questionnaire required participants' parents to provide information on their family's socio-economic background, home reading environment, parental reading habits and known family history of dyslexia (Appendix B). The parental questionnaire was developed for the purpose of the study after an extensive literature review. However, it does not represent a standardised measure of the reported aspects and no norms are available. Parents were asked if they, or any member of their own families, have or had reading problems, and about the reading home environment of their children (e.g., *How many age-appropriate books do you have?*) and parental reading habits (e.g., *How often do you, the parent, read a book for pleasure?*). *Ocular Examination.* Participants were screened for visual deficits, as required by Cellfield Pty. Ltd. A qualified optometrist conducted several visual eye exercises with each participant to determine if he or she displayed any specific visual weaknesses. Of particular interest were the ratings of visual stability and visual eccentricity. Normal visual stability was given if no discernable movement from the fovea is achieved and a steady focus could be maintained, in which case a score of zero was given. If the steady focus could not be hold, the participant was said to have fixation instability, and received a score of 0.5, 1 or 2, depending on the extent of the instability, with the higher score indicating a larger instability. Visual eccentricity referred to the inability to align both eyes so that their vision is exactly centred on the fovea, the sharpest point of vision in each eye. Scores of 0.5, 1 and 2 were given to express visual eccentricity and a score of 0 if the vision was centred on the fovea (Prideaux et al., 2005). Participants having 0.5 or above on instability and/or eccentricity were required to wear special glasses (with a red lens on one eye, and patch on the other eye) for some of the intervention sessions as directed in the Cellfield manual. For the Cellfield group, two participants (1 female, 1 male) wore these glasses.

## Pre-, post- and follow-up- Psychometric Tests

Table 3 gives an overview of the tests used in the assessment of dyslexia and reading-related skills at pre-, post- and follow-up-test. Details of each test are then explained in the text.

*The Dyslexia Screening Test- Secondary: Overall Risk for Dyslexia.* The Dyslexia Screening Test- Secondary (DST-S, Fawcett & Nicolson, 2004) provides an at-risk index for dyslexia along with an individual profile and consists of 13 subtests described below. Raw scores are converted to "at-risk-index" scores, which are based upon the stanine scale (M= 5, SD= 1.96). An overall "at-risk-index" is calculated. An at-risk index of 0.9 or greater is interpreted as a strong indication for the participant being at risk of dyslexia. The 13 subtests consist of:

 One-minute reading: The number of single words that can be read in 1 minute; a composite test of single word reading fluency and accuracy.

- Nonsense passage reading: A passage mixing real words and pseudowords.
  Pseudowords require knowledge of grapheme-phoneme correspondences to be read correctly. Scoring takes into account both accuracy and fluency.
- Two-minute spelling: The number of words the participant can spell correctly in 2 minutes. The tester dictates the next word as soon as the participant finishes the previous one; a combined test of spelling accuracy and fluency.
- 4. One-minute writing: The number of words copied in 1 minute, with adjustments made for errors. A test designed to assess speed of writing.
- Phonemic segmentation: Words need to be segmented into their constituent sounds (e.g., Say breakfast without fast).
- Spoonerisms: Word pairs are presented that require the participant to swap the sounds of the two words (e.g., Teddy Bear becomes Beddy Tear); a test of phonemic manipulation.
- 7. Backwards digit span: A string of single digits is presented on tape, and the participant has to repeat the string of digits in the reverse order. The tape starts with two digits and increases up to eight; a standard test of verbal working memory.
- Bead threading: The number of beads that can be threaded in 30 seconds; a standard test of manual dexterity as one aspect of cerebellar functioning.
- 9. Postural stability: How much the participant sways when pushed gently in the back using a pre-calibrated stability tester; a test of cerebellar/vestibular function.
- Rapid automatised naming: Involves the time taken to say the name of pictures on a page full of common objects; a test of general linguistic fluency as part of the memory retrieval fluency tests.
- 11. Verbal fluency: Simply how many words beginning with 'S' the participant can think of and say in 1 minute.
- 12. Semantic fluency: How many animals the participant can think of and say in 1 minute.
- 13. Non-verbal reasoning: Sequences of patterns have to be completed by pointing out the correct pattern; a test which requires application of non-verbal reasoning skills.

#### Table 3

Name of test Assessment of Post Pre Fol<sup>1</sup> Dyslexia Screening Test-Secondary Reading skills x\* х Wide Range Achievement Test 4: Spelling Skills х х x Spelling subtest Neale Analysis for Reading Reading Comprehension, х x х Fluency, Accuracy Woodcock Reading Mastery Test-Decoding skills and lowх х х Revised: Word Attack and Word frequency word reading Identification subtests skills Motivation to Read Profile **Reading Motivation** х х Review of Personal Effectiveness and Locus of Control х х Locus of Control

Tests for the Assessment of Dyslexia and Reading-Related Skills

1= Fol refers to Follow-up-test; \*= x indicates at which testing times a test was administered

*Wide Range Achievement Test-4: Basic Spelling Skills.* The spelling subtest of the Wide Range Achievement Test-4 (WRAT-4, Wilkinson, 2006) was used to assess basic spelling skills. This test requires the participant to write to dictation words that increase in level of difficulty. The WRAT-4 is a widely used and well-normed instrument. It provides measures of the performance of participants in relation to their same aged peers. Derived scores utilised in the present study are primary standard scores. Age norms are provided. Internal reliability coefficients range from .88 for the 8 year-old normative sample to .90 for 13 year olds.

Neale Analysis for Reading: Oral Reading Proficiency. The Neale Analysis for Reading (Neale, 1999) consists of six narratives with six levels of increasingly difficult vocabulary and complex grammar. The passages are read aloud by the participant. Following the completion of each passage the examiner asks the participant comprehension questions. Standard scores and reading ages are provided for reading accuracy, comprehension and rate for a norming sample from 6 to 12 years. Raw scores and reading ages were used for the current study. The internal Kuder-Richardson reliability coefficients for seven years of schooling (based on Australian schools) are reported at .96 for rate scores, .96 for accuracy scores and .89 for comprehension scores.

Woodcock Reading Master Test-Revised: Phonological Decoding and Irregular Word Recognition. Two subtests were used from the Woodcock Reading Mastery Test-Revised (WRMT-R, Woodcock, 1987). The Word Attack subtest measures participants' ability to apply phonological skills to read aloud 45 nonsense words. The Word Identification subtest requires the participant to read aloud words that appear less and less frequently in written English as the test progresses. Both measures' raw scores can be converted into grade and age equivalents and standard scores (M= 100, SD= 15) with the latter being used for the current study. Split half reliability (odd and even items) with the Spearman-Brown correction for Grade 8 for the word identification subtest is reported at .99 and for the word attack subtest at .95. Updated norms (1998) exist, but according to Pae et al. (2005) an inflation of 5 to 9 standard score points was indicated when using the updated norms.

*Motivation to Read Profile: Students' Motivation to Read.* The Motivation to Read Profile (MRP, Gambrell et al., 1996) was developed for the use in the classroom and to be administered by teachers. Twenty statements covering aspects of value of reading and selfconcept as a reader are read aloud to the participant. The participant estimates his/her own reading motivation. Gambrell et al. report guidelines for scoring and interpretation. Raw and percentage scores were calculated for overall reading motivation, the value of reading, and self-concept as a reader. Cronbach's alpha reliability is .75 for the self-concept scale and .82 for the value of reading scale. Pre- and post-test reliability was reported at .68 for the selfconcept scale and at .70 for the value of reading scale respectively. No norms have been published for the MRP.

Review of Personal Effectiveness and Locus of Control: Students' Perceived Life Effectiveness and Locus of Control. The Review of Personal Effectiveness and Locus of Control (ROPELOC, Richards, Ellis, & Neill, 2002) consists of 14 scales to assess individuals' perception of their own life effectiveness in different areas of their life. For the purpose of this study four scales were assessed, including the two locus-of-control scales (internal and external locus), the self-confidence scale, and the overall effectiveness scale. The participant's self-perception is assessed by reading statements to them which are rated on an 8-point scale (1 = it isn't like me at all to 8 = it is very much like me). Internal reliability for the 14 subscales ranged between .79 and .93, based on a normative sample of 1250. Norms have not been published for the ROPELOC.

## **Experimental Stimuli**

Participants completed three experimental tasks during which behavioural data and EEG activity was recorded. The experimental tasks were conducted three times; before, immediately after, and three weeks after the completion of the Cellfield intervention and the Placebo program.

Phonological and Lexical Decision Task. The phonological decision task (PDT) consisted of 80 visual word stimuli presented one word at a time, of which 40 were pseudo homophones (e.g., thaute) and 40 were nonwords (e.g., thaups). Participants were required to decide whether the presented word sounded like a real word ('yes' response to the pseudo homophones) or not ('no' response to the nonwords) (see Appendix C for complete list of word stimuli). The lexical decision task (LDT) consisted of 80 visual word stimuli presented one word at a time with 40 real words (e.g., queens), whereas 40 words were pseudo homophones (e.g., quenes). Participants were required to decide whether the presented word was spelled correctly ('yes' response to the real words) or not ('no' response to the pseudo homophones) (see Appendix C for complete list of word stimuli). Both the LDT and PDT word stimuli were presented in black 48 point Times New Roman font on a light grey background on a computer screen. The words were presented in a pseudo random order with the caveat that the same response ('yes', 'no') was not required more than five times in a row. All word stimuli were matched for frequency (from a minimum Standard Frequency Index of 10 to a maximum of 600; Kucera & Francis, 1967) and word length (six letters). Pseudo homophones and nonwords were generated from the ARC Nonword database (Rastle, Harrington, & Coltheart, 2002) and real words from the MRC database (Coltheart, 1981).

The presentation duration for each word stimulus was up to 4000 ms or until the participant responded, immediately followed by a fixation point '+' (duration 500 ms) which was followed by the beginning of the next word stimulus with an ISI of 500 ms. Figure 5 illustrates the time sequence for the stimuli.



Figure 5. Time sequence for the lexical and phonological task.

\*Note. Maximum response time was 4000 ms. However, if the response occurred earlier, the subsequent events (+, Blank, Next word) started as soon as the response was entered.

Sentence Task. The sentence task (ST) involved the processing of 90 six- to sevenword sentences, 45 of which ended in a semantically incongruent word ('no' responses) and 45 in a semantically appropriate word ending ('yes' responses) (see Appendix D for the complete list of sentences). Participants had to judge for each sentence whether it made sense ('yes' responses) or not ('no' responses). Presentation of the congruent and incongruent sentences was pseudo randomised, so that either congruent or incongruent sentences would not occur more than five times in a row. Table 4 shows some example sentences.

# Table 4

## Examples of Congruent and Incongruent Sentence Endings for the Sentence Task

| Sentence type | Sentence                 | Terminal word |
|---------------|--------------------------|---------------|
| Congruent     | He buys dog food for his | dog.          |
|               | She cooked it on the     | stove.        |
| Incongruent   | He buys dog food for his | story.        |
|               | She cooked it on the     | beard.        |

The words of each sentence were written in black 48 point Times New Roman font on a light grey background presented on a computer screen. Each sentence was written with appropriate sentence case and punctuation. The sentences were presented one word at a time with a duration of 500 ms for each word presentation and an ISI of 500 ms between words (see Figure 6 for time sequence of the sentence task). A slide showing XXXX following the final word of each sentence signalled the start of a waiting period of 1500 ms, with an ISI of 500 ms between the last word of a sentence and the beginning of the waiting period. The end of the waiting period and beginning of the response period (maximum response time 2 seconds) was marked by a slide showing a question mark, with an ISI of 250 ms. The ISI between the end of the response period and onset of the next sentence was 250 ms. The delayed response was chosen to prevent overlapping ERP components to the final word with response-related potentials like P3 in the utilised sentence task design (Holcomb et al., 1992; Nobre & McCarthy, 1994).



Figure 6. Time (ms) sequence for the sentence task.

*Note.* 'Word 1' and 'Word 2' refer to the words in each sentence (between six and seven words), 'Word X' to the final word of each sentence, and 'Next word' to the first word of a new sentence. The three black circles indicate that the time sequence is repeated until the final 'Word X' in each sentence is presented and the response (resp) required.

The 90 sentences were taken from Pratt, Kemp, and Martin (1996) who developed a list of sentences from three standardised reading tests: the Woodcock Reading Mastery Test-Revised (Woodcock, 1987), the Peabody Picture Vocabulary Test – Revised (Dunn & Dunn, 1981), and the National Adult Reading Test – Second Edition (Nelson, 1991). The existing list of congruent sentences was intended to provide highly predictive contexts for the word endings. The existing list of congruent sentences was rated again, by 20 adults, for the current study following the cloze procedure: "Please complete the following sentences. Write down the word which comes to your mind first". The sentence-final words selected were predicted by 77% to 100% of the raters and thus the sentences can be described as "highly predictive" sentences. The sentence-final words were further matched for frequency: all words had a Kucera-Francis frequency from a minimum of 10 to a maximum of 1000 and it was ensured that children in Grade 7 and 8 would have encountered the chosen words

by checking the corresponding frequencies from the "Words Children Know List" from the "Educator's Word Frequency Guide" from Ivens et al. (1995). After the finalisation of the congruent sentence list, a list of 45 incongruent word sentences was generated by crossing sentence-final words, matched for syntax. The incongruent word sentences list by Pratt et al. (1996) served as a guideline.

### Apparatus and EEG Recording

A Neuroscan 32 channel synamps system with Scan 4.3 software and Stim 3.1 software was used to record EEG, accuracy and reaction time data continuously while participants performed the tasks. Participants were fitted with an electrode quick cap to hold the electrodes to the scalp. The locations were 12 standard positions (F3, Fz, F4, C3, Cz, C4, CP3, CPz, CP4, TP7, TP8, Oz) from the international 10-20 system of electrode placement (Jasper, 1958). All electrode sites were referenced to left and right mastoids. Horizontal and vertical electro-oculargraphic (EOG) activity was recorded from the outer canthi of both eyes and above and below the left eye. Electrode impedance was kept below 5 K $\Omega$ . EEG activity was sampled continuously at 1000 Hz, and amplified with a DC (direct current) high pass, and a 300 Hz low pass filter. EEG data was merged with the behavioural data, continuous data files were then band pass filtered with 0.5 Hz high pass and 30 Hz low pass filter and ocular artefact reduction was conducted. The cut-off for artefact rejection was set between -100  $\mu$ V and 100  $\mu$ V. ERP data for correct responses were epoched offline for a 1100 ms epoch commencing 100 ms before stimulus onset and baseline corrected for the word stimuli from the lexical and phonological task and for the final-word stimuli for the sentence task. EEG activity for correct responses was averaged for each participant and averages including 15 trials or more were accepted for further analyses. Grand mean averages were calculated for each electrode site for each group and each time of testing (pre-, post-, and follow-up) for the two stimuli types in each task separately ('yes' and 'no' responses). In addition, for the sentence task difference waveforms were calculated by subtracting the congruent waveforms from the incongruent waveforms. This gives an index of incongruence over and above what would be expected under congruent conditions. ERP waveforms for each stimuli type were quantified by calculating the peak amplitude and

latency values for the voltage points in specific time windows which were decided after visual inspection of group grand mean ERP averages. For all three experimental tasks P2 (120-250 ms), N4 (250-500 ms) and LPC (500-800 ms) components were identified, scored, and used for further analyses.

# The Cellfield, Placebo and Follow-on Practice Programs

The Cellfield intervention consists of 10 one-hour computer-based sessions administered over two weeks with one session a day. Within each session there are 10 exercises targeting various deficits associated with dyslexia. Some of the exercises target phonological processing, requiring the concurrent activation of visual and auditory processing. Exercises involve decoding and encoding activities using tasks such as finding text embedded in continuous random text without spacing. There are three levels of difficulty: A+ (difficult), A (medium) and A-(easy), to allow more individual tailoring.

Motion graphics designed to stimulate the magnocellular pathways (transient system) are incorporated in each session. As described earlier, participants with visual problems were required to wear red lenses and eye patches. The stimulation of the transient system aims to enhance eye movement control, working memory, sequencing, and peripheral vision. Thus the motion graphics of the Cellfield intervention progressively change from translucent, so that words can be seen through the motion graphics, to opaque, so that words can only be read between the gaps of the motion graphics. The auditory presentation of the word stimuli is stretched for most sessions to allow participants to detect the phonemes in each word and to discriminate sound segments from each other. Table 5 describes the exercises included in each session.

As part of the program the participants had to complete two short homework sheets prior to each session. The homework sheets prepared the participants for the computer exercises. One sheet listed homophone pairs (words that are pronounced the same way, but have a different meaning and spelling, e.g., our - hour) and participants needed to find their meanings and write them down on the sheet. The other sheet contained the "Pidgin English" exercise which consists of "funny-made up words" where the first letter of a word is shifted to the end of the word and an "a" is added at the end of each word. The participants then had to delete the "a" at the end of each word and shift the last letter in front to make a real word again (e.g., omeha – home). Once completed the words were read aloud by the participants.

# Table 5

| Description of | and Example | es of the Co | ellfield Exerc | eises. |
|----------------|-------------|--------------|----------------|--------|

| Name of exercise | Exercise description                             | Example                             |
|------------------|--|-------------------------------------|
| Letter sounds    | The participant must say the sounds of letters.  | a,e,i,o,u                           |
| Rhyming          | The participant must choose the right word       | floss-dross-boss and the right word |
|                  | among similar sounding, rhyming words.           | was floss.                          |
| Pidgin English   | The participant must change funny-made up        | Change 'atca' back into 'cat' by    |
|                  | words into the right words.                      | deleting 'a' and putting 'c' at the |
|                  |  | beginning                           |
| Homophones       | These are words that sound the same, but are     | hour-our                            |
|                  | spelled differently. The participant has to pick |                                     |
|                  | the correct word spoken in a sentence.           |                                     |
| Embedded text    | The participant must scan an embedded text       | thekinghadatheevening               |
|                  | for words.                                       |                                     |
| Mosaics          | The participant must complete a black and        |                                     |
|                  | white mosaic pattern.                            |                                     |

The Placebo group engaged in a computer game called Zuma deluxe, (Zuma, 2002, http://www.realarcade.com, RealNetworks Inc.). The game required participants to manipulate a frog and gain points by shooting a colour-matched ball among other balls with different colours. The balls which appeared on the top of the computer screen at the start of each game and then moved down towards the frog, needed to be cleared before any ball could reach the frog. Different levels of difficulty existed. The game was non-violent. The visual graphics of this program are similar in complexity to the Cellfield exercises.

After completion of the ten Cellfield sessions or the Placebo sessions, all participants entered the reading and spelling practice phase of the program which included a three-week follow-on practice of reading and spelling. The follow-on practice consisted of the reading/spelling home practice and two to four sessions at school over the three weeks. Materials used for the sessions at school included individual reading material chosen by the participants or if preferred Dynamic Indicators of Basic Early Literacy Skills (DIBELS, 2000) which are standardised graded reading texts downloadable from the Internet (DIBELS, 2000, https://dibels.uoregon.edu/measures/materials.php, University of Oregon Center on Teaching and Learning). Repeated reading of these texts as well as comprehension training (e.g., asking questions about the text) were conducted. Spelling games were utilised either on a computer or by using the Look-Say-Cover-Write-Check method. This method involved:

- Look: The participants look at the word and then close their eyes and imagine the word
- 2. Say: The participants say the word out loud and write the letters "in the sky"
- 3. Cover: The participants cover the word
- 4. Write: The participants write the word
- 5. Check: The participants check their spelling and rewrite it if not correct.

Spelling words were individual chosen by the participants. Phonological games such as "syllasearch" and word building games were utilised as additional elements for the practice (Beck, 2005; Westwood, 2002).

The home practice program involved ten minutes' reading practice every day, with seven minutes' silent reading, three minutes' reading aloud to someone, and five to ten minutes' spelling practice if the participant wished to engage in this. Participants were encouraged to choose enjoyable reading materials and parents were asked to monitor the practice using a monitor sheet (Appendix E).

#### Procedure

# Phase 1: Pre-Screening and Testing for Dyslexia

Ethics approval for the study was obtained from the University of Tasmania's Ethics Committee and the Department of Education. Following these approvals schools were contacted and consent to participate in the project was obtained from two Tasmanian high schools. The initial screening of all Grade 7 students in the two Tasmanian high schools was organised and conducted in co-operation with the Grade 7 teachers and supervisors. The non-verbal intelligence test (Standard Progressive Matrices; Raven, 1938) was administered as a group test in class. The Nonword Reading Test (Martin & Pratt, 2000) and Irregular
Word Test (Coltheart & Leahy, 1996) were conducted in individual sessions with each student at their respective schools. During the individual testing sessions a brief welcome and introduction was given by the researcher before the two tests were administered. Student responses were audio-taped. This session lasted about 10 minutes.

Following the screening, parents of those students meeting the inclusion criteria, as outlined under the Participants section, were contacted and received a detailed briefing of the study and the proposed involvement of the students. After parental and student consent were obtained, each participant underwent the reading/spelling testing battery during school hours. This testing took between 90 to 120 minutes to complete. Tests were conducted in a standardised manner by the researcher following a testing protocol to prevent examiner effects. The administration of the tests was counterbalanced, so that participants received a different order at pre-, post- and follow-up-test. Some of the standardised tests, including the Neale, WRAT-4, and WRMT-R, provide two parallel test forms. These were used in a counterbalanced order to avoid gains due to repeated testing.

Following the screening and reading/spelling assessment, participants were taken to the optometrist to assess their visual performance. Teachers at the two schools provided transport assistance. Parents completed the parents' questionnaires, a medical questionnaire and the Child Behaviour Checklist (Achenbach, 2001) and were asked to return the forms prior to the commencement of the intervention.

### Phase 2: Pre-EEG Experiment

In the second phase all participants individually completed the three experimental tasks (PDT= Phonological Decision Task, LDT= Lexical Decision Task, ST= Sentence Task) during which EEG activity was recorded. The experimental tasks were conducted in a small quiet room at the children's schools. Participants were asked if they had any skin sensitivity or needle phobia and briefed on the EEG procedure. Participants then had the electrodes attached, were seated on a chair in front of the STIM computer, and given the instructions for the first task. The standard instructions for all three tasks are presented in Appendix F. Participants were given practice trials prior to each task to ensure that they understood the instructions. The order of the three tasks was counterbalanced to ensure participants did not

receive the same order of tasks at pre-, post- and follow-up-test. As mentioned earlier, participants had to press one of two buttons if the presented word sounded like a real word or not (PDT), spelled a real word or not (LDT), or if the presented sentence made sense or not (ST). As described previously, the required response for the ST was delayed by 1500 ms to avoid early responses: participants were instructed to wait during the presentation of the XXXXX, and respond when the '?' showed. The LDT and PDT tasks took eight minutes each, and the ST took 20 minutes. The whole procedure took between 90 minutes and two hours for each participant. After the completion of the three tasks participants were offered the opportunity to wash their hair.

## **Phase 3: Cellfield Intervention and Placebo Program**

Following the pre-reading/spelling assessment and the pre-ERP experiment, the intervention programs commenced. The technical set-up for the Cellfield intervention involved the installation of the Cellfield software on one university computer which was set up at the high schools while running the intervention. The intervention software was loaded into a computer of high-level graphic processing specification, with an optical mouse for good eye-hand control. A set of headphones was connected to the computer. Reflections on the CRT monitors were avoided by closing the blinds in the rooms. The Placebo program was installed on a laptop made available by the two schools.

Cellfield sessions and Placebo sessions took place during school hours (9 am to 3 pm), in a quiet separate room. Cellfield sessions required between 60 to 90 minutes and Placebo sessions were conducted for between 50 and 60 minutes. Participants were told which group they were in, but great effort was made not to raise any expectations about possible outcomes. Participants in the Placebo program were not informed about the actual purpose of their program, but knew that they would be doing the Cellfield intervention afterwards. The Cellfield group was told that we were mainly interested in what this program could or could not do for them. With the exception of two participants, the participants were organised in pairs, with one participant from each group, so that the two programs were conducted at the same time. Each participant was seated in front of a

computer with the Cellfield or Placebo program installed. A brief introduction was given at the first session.

Following this the homework sheets for the intervention (homophones and Pidgin English exercises) were completed with the participants from the Cellfield group. The participants always completed the homework sheets together with the researcher in order to ensure a methodologically consistent procedure. Each participant in this group was guided through the Cellfield intervention sessions by the researcher. As much as possible, the experimenter ensured that each participant maintained a distance from the screen of between 475mm and 525mm by asking him or her to move back into this distance range whenever he or she appeared to move too close or too far away. After each session each participant's score was written down on a monitor sheet. Participants in the Placebo group received instructions on the computer game and were told to report their scores at the end of each session on a monitor sheet. The intervention and Placebo program were completed within six months for all participants. After the completion of the Cellfield intervention and the Placebo program respectively, two appointments were arranged with each participant for the post- reading/spelling assessment and post-ERP experiment.

### Phase 4 and 5: Post Dyslexia Assessment and ERP Experiment

Immediately following the completion of the 10 sessions, all participants took part in the post-test consisting of both the ERP experiments and the reading/spelling assessments (DST-S, MRP, ROPELOC, WRMT-R, WRAT-4, Neale) from the pre-test (Phase 1 and 2). Reading tests and experimental tasks were conducted in the same way as at pre-test.

### **Phase 6 and 7: Follow-on Practice Sessions and Home Practice**

Once the post-tests were completed all participants engaged in further tutorial sessions at their schools. The sessions at the schools were conducted by the researcher in one school and by two literacy support teachers in the other school. Some participants were organised in pairs and some received one-on-one sessions. The sessions lasted for one school lesson and were conducted in a semi-structured way. The teachers and researcher adapted the exercises to suit the participants' learning needs and the pairs of participants received spelling, reading, and phonological skill practice that varied in both length and intensity.

With regard to the home practice, the procedure involved brief instructions on the intention of the home practice and the importance of completing the monitoring sheet. Participants and their parents were asked to write down on the monitor sheet when and for how long they engaged in the reading and/or spelling practice at home. Most participants chose a book or DIBELS reading material to take home and read, while others had books at home they wanted to practice with. How the actual practice was conducted at the participants' respective homes was beyond the researcher's control. At the end of the three-week follow-on practice, participants were asked to return the monitor sheets.

## Phase 8 and 9: Follow-up Dyslexia Assessment and ERP Experiment

The follow-up-test followed the same procedure as described in Phase 1 and 2 and was conducted with all participants from the Cellfield and Placebo group for the literacy measures. The tests for the follow-up-data included the WRMT-R, WRAT-4 and Neale. For the experimental measures data from five Cellfield participants and three Placebo participants were obtained.

#### Design

Overall the study followed a 2 [Group: Cellfield, Placebo] x 3 (Time: pre, post, follow-up) x 3 (Task: lexical, phonological, sentence) x 2 (Stimuli Type: 'yes' responses, 'no' responses) repeated measures design. The electrode sites led to two further within-subjects factors of Sagittal (frontal, central, central-parietal) and Coronal (left, mid, right). Efficacy of the Cellfield intervention compared to the Placebo program and the Follow-on practice was investigated on the following dependent variables: Literacy measures (DST-S, MRP, ROPELOC, WRAT-4, WRMT-R, Neale test data) and experimental measures (ERP and behavioural data). The dependent variables for the literacy data were mean raw scores on the pre-, post-, and follow-up-tests, for the experimental behavioural data mean reaction time, accuracy, and missing responses, and for the ERP data mean amplitudes and latencies of the P2, N4 and LPC components of the ERP.

# Literacy Data Analyses

Means and standard errors of the pre-, post-, and follow-up- literacy raw scores were calculated for the two groups. Descriptive statistics were also calculated in terms of standard scores including primary standard scores (M= 100, SD= 15), at-risk indexes, and reading ages where available (DST-S, WRMT-R, WRAT-4, Neale) to allow the placement of the group scores in comparison to a normative sample. The ANOVAs were performed in two steps: For the pre-and post-test literacy measures (DST-S, MRP, ROPELOC) mean raw scores were analysed using repeated measures ANOVAs with Group [Cellfield, Placebo] as the between-subjects factor and Time (pre, post) as the within-subjects factor. The pre-, post- and follow-up-test literacy measures (WRMT-R, WRAT-4, Neale) were analysed with three levels of the factor Time (pre, post, follow-up).

# Psychophysiological Data Analyses (Behavioural and ERP)

ANOVAs for the behavioural and ERP data were performed in two steps: Two major ANOVAs were conducted, pre-and post-test ANOVAs, and ANOVAs including follow-updata. The sentence task was analysed separately and the lexical and phonological tasks were analysed together to investigate the potential differential impact of the Cellfield intervention on the two word-level tasks (Barnea & Breznitz, 1998; Niznikiewicz & Squires, 1996; Penolazzi et al., 2006; Ziegler et al., 1997). Means and standard errors were calculated for the pre-, post-, and follow-up-test RT data for correct responses (ms) and accuracy data (percentage), as well as overall missing responses (percentage), for the three tasks for both groups.

Mean RT and accuracy scores were entered into an ANOVA with Group [Cellfield, Placebo] as the between-subjects factor, and Time (pre, post for the first ANOVA and pre, post, follow-up for the second ANOVA) and Stimuli Type ('yes' responses, 'no' responses) as the within-subjects factors. For the lexical and phonological task the within-subjects factor Task (lexical, phonological) was added. The different stimuli types for each task are described under this chapter's 'experimental stimuli' section. Overall missing responses were analysed using ANOVA with Group [Cellfield, Placebo] as the between-subjects factor and Time (pre, post for the first ANOVA and pre, post, follow-up for the second ANOVA) as the within-subjects factor. Again, the simultaneous analysis of the lexical and phonological tasks led to another within-subjects factor of Task (lexical, phonological).

ERP waveforms were scored for peak amplitude and latency for the two stimuli types for all three tasks for both groups. Inspection of the grand group means showed distinct peaks for the P2, N4 and LPC time windows. For the lexical and phonological tasks mean peak amplitude and latency of the P2, N4, and LPC components were analysed. For the sentence task we analysed mean peak amplitude and latency of the N4 and LPC components, since previous research on sentence processing has largely focussed on these later, linguistic ERP components (for a review see Kutas et al., 2006). Although we also computed difference waveforms for the sentence task (incongruent minus congruent) these were not used for further analyses, but for demonstration purposes only. The electrode sites for the ANOVAs were F3, Fz, F4, C3, Cz, C4, CP3, CPz, CP4. Mean amplitude and latency scores were entered into an overall ANOVA contrasting the left and right hemisphere, and frontal and central-parietal sites, as dyslexic readers have been shown to have a lack of left lateralisation and sometimes more activity at frontal sites during tasks that require linguistic processing in comparisons to controls (see Chapter 5). The ANOVAs were performed in the same way as for the RT and accuracy data, adding the two electrode factors, Sagittal (frontal, central, central-parietal) and Coronal (left, mid, right). Break-down ANOVAs were conducted where appropriate by analysing sagittal and coronal sites separately.

For both the experimental behavioural and ERP data, the pre- and post-test ANOVAs included data from seven Cellfield participants and five Placebo participants and the pre-, post-, and follow-up-ANOVAs are based on five Cellfield participants and three Placebo participants. Due to the smaller number of participants at follow-up-test, we conducted the pre- and post-test ANOVAs first so that more data could be included. However, to investigate further changes after the three-week follow-on practice the pre-, post-, and follow-up-ANOVA is an additional, exploratory one. Thus the results as well as comparisons between pre- and post-data (behavioural and ERP), and pre-, post- and followup-data (behavioural and ERP) must be interpreted with caution as the small participant numbers limit statistical power. The smaller participant numbers at follow-up-test occurred because two participants from the Placebo group did not participate in the follow-up due to other school commitments and two participants from the Cellfield group did not wish to participate in the follow-up.

All repeated measures ANOVAs were conducted using STATISTICA 7.0 and Greenhouse-Geisser corrections were applied where necessary. The alpha level was set at 0.05 and Tukey post-hoc tests were used to compare individual means for significant differences where appropriate. In order to control for Type II errors, trends towards significance levels (p < 0.10) were considered. It should further be noted that the large number of dependent variables for the literacy and experimental data (ERP, reaction time and accuracy) and the statistical tests performed on these, potentially lead to a high Type I error, Hence significant results need to be treated with extreme caution, However, in order not to miss potentially important findings it was considered appropriate to risk a high Type I error rate. The findings are further limited by the small participant numbers in each group and results should be treated as pilot data. All significant and trends towards significant main effects involving the major factors of interest (Group, Time, Task, and Type) are reported. With regard to interactions, of primary interest were the changes in literacy, experimental behavioural and ERP measures over time for the Cellfield and the Placebo group. Thus, results are presented with a focus on interactions involving Group and Time that were significant, or that showed trends to significance. However interactions involving Group, Time, Task, or Type are reported when theoretically relevant. Effect sizes for all data were calculated as the ratio of the effect variance to the error variance  $(\eta p^2)$  to evaluate the magnitude and practical relevance of these effects (Kirk, 1982). An effect size of 0.2 is considered small, 0.5 medium and 0.8 and higher large (Cohen, 1988). Detailed results are presented in three main sections with the literacy data presented first, then the experimental behavioural data (reaction time, accuracy, and missing responses) followed by the experimental ERP data. Error bars represent 95% of confidence interval on the figures in the results section.

### **Chapter 9: Results**

#### **Treatment Fidelity**

Extensive screening of all raw data (literacy, psychophysiological) for statistical outliers was conducted and revealed no outliers. Participants in the Cellfield group received a mean of 13.13 intervention sessions with a mean of 10 Cellfield computer sessions for each participant and a mean of 3.13 follow-on training sessions (range two to four sessions), after the completion of the Cellfield program. The Placebo group completed a mean of 12.00 intervention sessions consisting of a mean of 9.60 Placebo game sessions (range 8 to 10 sessions) and a mean of 2.40 follow-on training sessions (range two to three sessions). The total instruction time for the Cellfield group was a mean of 780.63 mins (13.01 hours) and for the Placebo group a mean of 685.00 mins (11.42 hours).

With regard to the frequency of the Cellfield and Placebo sessions, ideally participants engaged in one session a day, for two weeks. However, participants' absences due to sickness imposed more flexible time arrangements on the conduct of the sessions. After a participant returned to school, double sessions were sometimes arranged, if appropriate for the participant's learning needs. In other cases the program was interrupted for the time of the participant's absence and then continued on a daily basis.

Whereas the Cellfield training and Placebo program are methodologically stringent and thus not vulnerable to external factors, the follow-on practice was much more variable and individually tailored to each participant. For the home practice, which occurred during the three weeks follow-on practice, parents and participants were asked to fill out a record sheet to monitor the home practice. However, only three participants returned their form, so that the intensity of the home practice during the three weeks follow-on practice cannot be satisfactory evaluated. During the time of treatment, two of the Cellfield participants and one of the Placebo participants were also engaged in other tutorial programs for their literacy difficulties at their school.

### Literacy Measures

As outlined in Chapter 8, some literacy measures were assessed only at pre- and post-test, as these measures did not provide parallel testing forms to minimise repetition effects and others were assessed at pre-, post- and follow-up-test, namely those measures which did provide two parallel testing forms to use for repeated testing. First, outcomes on measures collected at pre- and post-test will be presented, followed by tests including pre-, post- and follow-up-data.

# **Pre-and Post- Literacy Measures**

Mean raw scores and standard deviations for the Dyslexia Screenings Test- Secondary (DST-S), Motivation to Read Profile (MRP) and Review of Personal Effectiveness and Locus of Control (ROPELOC) were obtained, and pre- and post-test comparisons are shown in Table 6.

Table 7 shows mean scores and standard deviations for at-risk indexes from the DST-S for pre- and post-test comparisons. At-risk indexes are calculated by dividing the total DST-S raw score by twelve. The manual of the DST-S gives three categories, consisting of a 'mild risk' for dyslexia indexed by 0.6 to 0.8, 'strong risk' with at-risk indexes from 0.9 and higher, and an index of 1.7 and higher indicating 'very strongly atrisk'. As shown in Table 7 the average overall risk for dyslexia was 1.25 for the Cellfield group, and 0.97 for the Placebo group respectively, indicating a strong at-risk index. Inspection of the individual profiles revealed the following distribution of risk-categories for the Cellfield group at pre-test: Two participants fell into the category 'very strongly at-risk', three participants were at 'strong risk', and two participants at 'mild risk'. Within the Placebo group three participants were identified as at 'strong risk', and two as at 'mild risk'. As the DST-S was the second selection criteria for dyslexia individual categories are presented in the text. Stem and leaf profiles of the other literacy tests (Neale, WRAT-4, WRMT-R) at pre-test for both groups are presented in Appendix G. As in previous research our sample displayed multiple literacy deficits and depending on the participant the severity of difficulties varied.

# Table 6

Mean Literacy Raw Scores for Cellfield and Placebo Group at Pre- and Post-test

|                                      |                 | Pre-           | test    |                | Post-test |                  |               |       |  |  |
|--------------------------------------|-----------------|----------------|---------|----------------|-----------|------------------|---------------|-------|--|--|
|                                      | Cellfield (n=7) |                | Placebo | ( <i>n</i> =5) | Cellfiel  | d ( <i>n</i> =7) | Placebo (n=5) |       |  |  |
|                                      | RS              |                | R       | S              | R         | S                | RS            |       |  |  |
| Variable name                        | М               | SD             | М       | SD             | M         | SD               | М             | SD    |  |  |
| DST-S sum                            | 15.29           | 5.79           | 12.00   | 4.12           | 10.57     | 1.62             | 10.80         | 6.02  |  |  |
| Rapid Naming                         | 34.43           | 4.86           | 33.20   | 4.55           | 31.29     | 4.06             | 30.00         | 4.53  |  |  |
| Bead Threading                       | 7.86            | 2.54           | 7.60    | 2.19           | 9.14      | 4.64             | 8.40          | 2.30  |  |  |
| One Minute Reading                   | 27.00           | 16.91          | 38.60   | 15.37          | 33.43     | 1.39             | 43.60         | 15.32 |  |  |
| Postural Stability                   | 0.71            | 0.95           | 1.60    | 2.07           | 1.29      | 1.29             | 1.60          | 1.95  |  |  |
| Phonemic Segmentation                | 9.14            | 2.34           | 8.40    | 2.19           | 10.29     | 1.98             | 9.40          | 1.14  |  |  |
| Spoonerisms                          | 8.29            | 5.12           | 10.00   | 4.69           | 11.57     | 1.03             | 10.80         | 3.90  |  |  |
| Two Minute Spelling                  | 15.00           | 3.79           | 16.40   | 3.58           | 17.29     | 1.46             | 17.40         | 3.85  |  |  |
| Backwards Digits<br>Nonsense Passage | 3.29            | 1.25           | 4.40    | 1.82           | 3.57      | 1 <b>.79</b>     | 4.40          | 1.52  |  |  |
| Reading                              | 33.43           | 1 <b>6.9</b> 4 | 43.00   | 10.42          | 39.21     | 1.42             | 42.40         | 13.67 |  |  |
| One Minute Writing                   | 19.29           | 6.65           | 19.40   | 4.56           | 20.00     | 1.46             | 19.60         | 5.37  |  |  |
| Verbal Fluency                       | 11.43           | 4.89           | 15.60   | 2.19           | 15.57     | 1.11             | 18.00         | 3.54  |  |  |
| Semantic Fluency                     | 17.14           | 3.67           | 19.40   | 4.39           | 20.71     | 2.18             | 19.20         | 3.77  |  |  |
| Nonverbal Reasoning                  | 5.86            | 0.90           | 4.40    | 1.14           | 5.86      | 1.01             | 5.60          | 0.89  |  |  |
| MRP Reading<br>motivation            | 44.14           | 7.43           | 45.80   | 5.93           | 46.00     | 3.07             | 45.00         | 4.64  |  |  |
| ROPELOC                              |                 |                |         |                |           |                  |               |       |  |  |
| Internal Locus                       | 6.52            | 1.27           | 6.40    | 1.04           | 6.38      | 6.19             | 5.90          | 1.75  |  |  |
| External Locus                       | 2.76            | 1.18           | 3.40    | 1.38           | 2.52      | 1.23             | 4.20          | 0.69  |  |  |
| Self Confidence                      | 5.90            | 1.07           | 5.67    | 1.20           | 5.62      | 2.04             | 4.87          | 1.19  |  |  |
| Overall Effectiveness                | 4 57            | 1 46           | 4 93    | 0.95           | 4 38      | 1 27             | 4.73          | 0.64  |  |  |

RS= Raw score; DST-S= Dyslexia Screening Test-Secondary; MRP= Motivation to Read Profile; ROPELOC= Review of Personal Effectiveness and Locus of Control

At post-test the 'at-risk' index for the Cellfield group dropped to an average of 0.84 (one participant 'no risk', two participants 'mild risk', and four participants 'strong risk'). For the Placebo group the 'at-risk' index also dropped, although to a lesser extent, with an average mean of 0.87 (one participant 'very strong risk', three participants 'mild risk', and one participant 'no risk').

# Table 7

Mean DST At-Risk Indexes for Cellfield and Placebo Group at Pre- and Post-test

|                    | Pre-test |                   |        |                  | Post-test |                  |               |      |  |  |
|--------------------|----------|-------------------|--------|------------------|-----------|------------------|---------------|------|--|--|
|                    | Cellfie  | ld ( <i>n</i> =7) | Placeb | o ( <i>n</i> =5) | Cellfiel  | d ( <i>n</i> =7) | Placebo (n=5) |      |  |  |
|                    | RI       |                   | F      | u –              | R         | LI LI            | RI            |      |  |  |
| Variable name      | M        | SD                | M      | SD               | M         | SD               | M             | SD   |  |  |
| DST-S sum          | 1.25     | 0.47              | 0.97   | 0.32             | 0.84      | 0.41             | 0.87          | 0.51 |  |  |
| Rapid Naming       | 0.57     | 0.53              | 0.40   | 0.55             | 0.29      | 0.76             | 0.40          | 0.55 |  |  |
| Bead Threading     | 0.43     | 1.13              | 0.40   | 0.89             | 0.00      | 0.00             | 0.40          | 0.89 |  |  |
| One Minute         |          |                   |        |                  |           |                  |               |      |  |  |
| Reading            | 2.43     | 0.79              | 1.60   | 0.89             | 2.29      | 0.76             | 1.40          | 0.89 |  |  |
| Postural Stability | 0.00     | 0.00              | 0.00   | 0.00             | 0.14      | 0.38             | 0.00          | 0.00 |  |  |
| Phonemic           |          |                   |        |                  |           |                  |               |      |  |  |
| Segmentation       | 1.43     | 1.40              | 1.60   | 1.14             | 0.43      | 1.13             | 1.00          | 1.00 |  |  |
| Spoonerisms        | 1.71     | 1.25              | 1.40   | 1.14             | 0.71      | 1.11             | 1.20          | 1.10 |  |  |
| Two Minute         |          |                   |        |                  |           |                  |               |      |  |  |
| Spelling           | 2.29     | 0.76              | 2.00   | 0.71             | 2.00      | 1.15             | 2.00          | 0.71 |  |  |
| Backwards Digits   | 1.71     | 1.25              | 1.00   | 1.22             | 1.43      | 1.13             | 1.00          | 0.71 |  |  |
| Nonsense Passage   |          |                   |        |                  |           |                  |               |      |  |  |
| Reading            | 2.43     | 0.79              | 2.20   | 0.45             | 2.29      | 0.95             | 2.40          | 0.55 |  |  |
| One Minute         |          |                   |        |                  |           |                  |               |      |  |  |
| Writing            | 0.71     | 1.11              | 0.60   | 0.89             | 0.71      | 1.11             | 0.60          | 0.89 |  |  |
| Verbal Fluency     | 1.14     | 1.35              | 0.00   | 0.00             | 0.00      | 0.00             | 0.00          | 0.00 |  |  |
| Semantic Fluency   | 0.14     | 0.38              | 0.00   | 0.00             | 0.00      | 0.00             | 0.00          | 0.00 |  |  |
| Nonverbal          |          |                   |        |                  |           |                  |               |      |  |  |
| Reasoning          | 0.29     | 0.76              | 0.80   | 0.84             | 0.29      | 0.41             | 0.40          | 0.89 |  |  |

RI= Risk index; DST-S= Dyslexia Screening Test-Secondary

*Pre- and Post- Dyslexia Screening Test-S (DST-S): Overall Risk for Dyslexia.* For the mean overall DST-S raw score the ANOVA indicated a significant main effect of Time, F(1,10)=15.40, MSE=3.31, p<0.05,  $\eta p^2=0.61$ , which was moderated by a significant interaction between Time and Group, F(1,10)=5.44, MSE=3.31, p<0.05,  $\eta p^2=0.35$ . Tukey post-hoc tests indicated a significantly larger decrease for the DST-S score for the Cellfield group only, from pre- to post-test, whereas the Placebo group showed a non-significant (p=0.73) smaller decrease (Figure 7). Although the Placebo group had a higher DST-S score than the Cellfield group at pre-test, the difference between the two groups was not significant (p=0.86).



Figure 7. Mean DST-S raw scores for Cellfield and Placebo group at pre- and post-test.

The overall DST-S raw score gives an estimate of the overall risk of dyslexia. The test however also provides summary raw scores (see Table 6) for each of the subtests, which can also be converted into at-risk indexes (see Table 7). A series of ANOVAs on the raw scores of the subtests of the DST-S were performed to investigate further which abilities may have improved at post-test. Significant main effects of Time were achieved for the subtests one-minute reading, F(1,10)=29.83, MSE=6.39, p<0.05,  $\eta p^2=0.75$ , phonemic segmentation, F(1,10)=4.99, MSE=1.34, p<0.05,  $\eta p^2=0.33$ , spoonerisms, F(1,10)=8.08, MSE=3.01, p<0.05,  $\eta p^2=0.45$ , two-minute spelling F(1,10)=8.89, MSE=1.77, p<0.05,  $\eta p^2=0.47$ , and verbal fluency, F(1,10)=5.62, MSE=11.10, p<0.05,  $\eta p^2=0.36$ . Overall significantly improved mean test-scores in all those subtests were observed at post-compared to pre-test. The largest effect size was obtained for the subtest one-minute reading with a magnitude of  $\eta p^2=0.75$ . Effect sizes for the other subtests yielded small effects ranging between  $\eta p^2=0.33$  and 0.47. No significant interactions with Group were identified.

# Pre-and Post- Motivation to Read Profile (MRP): Overall Reading Motivation.

There were no significant effects for reading motivation (ps>0.05). The Cellfield and

Placebo groups' overall reading motivation did not increase or decrease significantly at posttest. As indicated in Table 6 both groups had a mean raw score around 45, indicating a relatively low motivation to read score, as the highest possible score is 80.

### Pre-and Post- Review of Personal Effectiveness and Locus of Control

(ROPELOC). The four scales of the ROPELOC were assessed and analysed: the internal locus of control (INT), external locus of control (EX), self confidence (SC) and overall effectiveness (OE). No significant main effects or interactions were found for INT, SC or OE (ps > 0.05). Interestingly on one measure of the ROPELOC, the EX, a significant interaction was identified between Time and Group, F(1,10) = 7.31, MSE = 0.22, p < 0.05,  $np^2 = 0.42$ . Tukey post-hoc tests revealed a trend towards a significant difference between pre- and posttest scores for the Placebo group. As can be seen in Figure 8, the Placebo group displayed higher external locus of control scores at post- compared to pre-test (p=0.08). Tukey posthoc tests also revealed that the two groups were not significantly different in their external locus at pre-test (p > 0.05). However, despite the Placebo group's increased external locus of control scores at post-test, the difference between the two groups at post-test did not reach significance (p=0.23). A higher score on the external locus of control scale indicates an individual's tendency to attribute success and failure to external causes that are not within the control of the individual him/herself. The lowest possible score is 1, the highest score is 8. The Placebo group's mean of 4.20 at post-test can be considered a moderate external locus.

# Pre-, Post-, and Follow-up- Literacy Measures

Preliminary analyses were conducted on the parallel forms of the literacy tests to investigate whether differences in outcomes varied according to the form used at pre-, post- and followup-test. The ANOVAs did not reveal any significant differences between any of the test forms used (ps> 0.05). Mean raw scores and standard deviations were obtained for the WRMT-R (word identification and word attack subtests), Neale (reading accuracy, comprehension, and rate), and WRAT-4 (spelling subtest) for both groups, and pre-, postand follow-up-test comparisons are shown in Table 8.



Figure 8. Mean External Locus raw scores for Cellfield and Placebo group at pre- and post-test.

# Table 8

Mean Literacy Raw Scores for Cellfield and Placebo Group at Pre-, Post- and Follow-uptest

|          | Pre-test |       |          |       |         | Post-test |                   |       |         | Follow-up-test |         |       |  |
|----------|----------|-------|----------|-------|---------|-----------|-------------------|-------|---------|----------------|---------|-------|--|
|          | CF (     | n=7)  | PL (n=5) |       | CF(n=7) |           | PL ( <i>n</i> =5) |       | CF(n=7) |                | PL(n=5) |       |  |
|          | RS       |       | RS       |       | RS      |           | RS                |       | RS      |                | RS      |       |  |
| Variable | М        | SD    | M        | SD    | М       | SD        | M                 | SD    | М       | SD             | М       | SD    |  |
| WRMT-R   |          |       |          |       |         |           |                   | -     |         |                |         |       |  |
| WI       | 52.14    | 12.36 | 59.60    | 11.01 | 59.86   | 15.61     | 60.60             | 13.58 | 61.71   | 15.42          | 63.00   | 13.04 |  |
| WA       | 16.00    | 6.22  | 18.80    | 7.85  | 27.00   | 9.24      | 21.20             | 8.07  | 25.86   | 8.32           | 23.60   | 7.02  |  |
| Neale    |          |       |          |       |         |           |                   |       |         |                |         |       |  |
| Acc      | 35.86    | 10.90 | 45.00    | 13.64 | 42.14   | 16.07     | 45.80             | 15.90 | 48.29   | 17.79          | 56.60   | 15.21 |  |
| Compr    | 17.29    | 5.62  | 19.20    | 7.79  | 20.71   | 6.47      | 20.20             | 7.69  | 25.60   | 10.21          | 25.29   | 7.92  |  |
| Rate     | 56.00    | 22.01 | 58.20    | 11.12 | 46.86   | 21.50     | <b>64.8</b> 0     | 14.45 | 56.57   | 11.77          | 66.60   | 19.01 |  |
| WRAT-4   |          |       |          |       |         |           |                   |       |         |                |         |       |  |
| Spelling | 24.71    | 2.98  | 25.80    | 3.03  | 26.00   | 4.86      | 26.40             | 3.85  | 25.71   | 2.69           | 26.80   | 3.11  |  |

CF= Cellfield group; PL= Placebo group; RS= Raw score; WRMT-R= Woodcock Reading Mastery Test-Revised; WI= Word Identification; WA= Word Attack; Neale= Neale Analysis of Reading Ability; Acc= Reading Accuracy; Compr= Reading Comprehension; WRAT-4= Wide Range Achievement Test Table 9 presents mean raw scores converted to a standardised measure, including reading ages (Neale) and primary standard scores (WRMT-R, WRAT-4) to allow for achievement comparisons for the sample with a normative sample. As indicated by the group means in Table 9, both groups had average standard scores of at least one standard deviation below, and reading ages of at least two years below the expected performance at pre-test on the respective tests. The largest change at post-test occurred for the Cellfield group for phonological decoding (nonword reading) as assessed by the word attack subtest of the WRMT-R, with a mean of 71.86 at pre-test and a mean of 89.29 at post-test.

## Table 9

Mean Literacy Standard Scores for Cellfield and Placebo Group at Pre-, Post- and Followup-test

|          | Pre-test            |       |       |       |       | Post              | t-test |                             | Follow-up-test        |          |       |       |
|----------|---------------------|-------|-------|-------|-------|-------------------|--------|-----------------------------|-----------------------|----------|-------|-------|
|          | - CF (n=7) PL (n=5) |       | n=5)  | CF (  | n=7)  | PL ( <i>n</i> =5) |        | $\overline{\text{CF}(n=7)}$ |                       | PL (n=5) |       |       |
|          | S                   | S*    | S     | S*    | SS*   |                   | SS*    |                             | SS*                   |          | SS*   |       |
| Variable | $\overline{M}$      | SD    | M     | SD    | M     | SD                | M      | SD                          | M                     | SD       | M     | SD    |
| WRMT-R   |                     |       |       |       |       |                   |        |                             |                       |          |       |       |
| WI       | 65.29               | 13.34 | 72.20 | 15.01 | 73.14 | 17.26             | 73.80  | 18.69                       | 75.57                 | 16.94    | 76.40 | 18.26 |
| WA       | 71.86               | 9.30  | 75.00 | 13.58 | 89.29 | 15.90             | 79.00  | 12.90                       | 86.86                 | 12.68    | 82.00 | 10.70 |
| Neale    |                     |       |       |       |       |                   |        |                             |                       |          |       |       |
| Acc      | 7.77                | 0.77  | 8.42  | 0.97  | 8.26  | 1.18              | 8.53   | 1.10                        | 8.90                  | 1.77     | 9.57  | 1.43  |
| Compr    | 8.60                | 1.17  | 9.18  | 1.89  | 9.45  | 1.59              | 9.42   | 1.75                        | 10.85                 | 2.58     | 10.75 | 2.14  |
| Rate     | 8.70                | 1.96  | 8.65  | 0.90  | 7.92  | 1.55              | 9.23   | 1.20                        | 8.57                  | 1.00     | 9.47  | 1.72  |
| WRAT-4   |                     |       |       |       |       |                   |        |                             |                       |          |       |       |
| Spelling | 79.57               | 6.68  | 81.80 | 7.01  | 81.14 | 9.96              | 82.20  | 9.63                        | <b>81</b> .7 <u>1</u> | 5.53     | 83.60 | 7.27  |

CF= Cellfield group; PL= Placebo group; SS\*= Standard Score: Primary standard scores (M= 100, SD= 15) for WRMT-R and WRAT-4, and reading ages for the Neale.

WRMT-R= Woodcock Reading Mastery Test-Revised; WI= Word Identification; WA= Word Attack; Neale= Neale Analysis of Reading Ability; Acc= Reading Accuracy; Compr= Reading Comprehension; WRAT-4= Wide Range Achievement Test-4

Pre-, Post- and Follow-up- Woodcock Reading Mastery Test-Revised (WRMT-R):

Irregular Word Reading and Nonword Reading. The ANOVA indicated a significant Time

main effect for the word identification measure, F(2,20)=6.87, MSE=18.6, p<0.05,  $\eta p^2=$ 

0.41. As confirmed by Tukey post-hoc tests, overall significant gains were made from pre- to

post-test (p < 0.05). This gain was maintained at follow-up, as indicated by a significant pre-

to follow-up difference (p < 0.05). A further significant increase from post- to follow-up-test

was not observed (p=0.26). No significant interaction involving Group and Time was found (p=0.14).

The ANOVA conducted on the word attack scores revealed a significant main effect of Time, F(2,20)=19.16, MSE=10.05, p<0.05,  $\eta p^2=0.66$ . The Time effect was moderated by a significant Time by Group interaction, F(2,20)=5.42, MSE=10.05, p<0.05,  $\eta p^2=0.35$ (see Figure 9). The Cellfield group showed a significant improvement from pre- to post-test, and this difference was maintained at follow-up, as indicated by Tukey post-hoc tests (ps<0.05). A smaller non-significant improvement was observed for the Placebo group from preto follow-up-test (p=0.31). The difference between the two groups at pre-test was not significant (p=1.00).



*Figure 9.* Mean Word Attack raw scores for Cellfield and Placebo group at pre-, post- and follow-up-test.

*Pre-, Post-, and Follow-up- Oral Reading Proficiency (Neale): Reading Accuracy, Comprehension and Rate.* The ANOVAs conducted on the reading accuracy and comprehension data indicated a significant effect of Time for both accuracy, F(2,20)=14.15, MSE=31.41, p<0.05,  $\eta p^2=0.59$ , and comprehension, F(2,20)=14.93, MSE=10.63, p<0.05,

ł

 $\eta p^2 = 0.60$ , indicating that overall the groups changed over time in their reading accuracy and comprehension. Tukey post-hoc tests revealed significant increases in overall accuracy and comprehension from pre- to follow-up-test and from post- to follow-up-test (*ps*< 0.05). Both effects can be considered moderate. For reading rate no effects reached significance (*ps*> 0.05).

*Pre-, Post-, and Follow-up- Wide Range Achievement Test (WRAT-4): Spelling.* No significant effects were found for the spelling skill measure (*ps*> 0.05).

## **Psychophysiological Measures**

Reports for the behavioural experimental data are presented first. Pre-and post data, and pre-, post- and follow-up-data for the sentence task are presented followed by pre-and post data, and pre-, post- and follow-up-data for the lexical and phonological task. The ERP experimental data are reported in the last section including findings from the pre-and post-analyses, and pre-, post- and follow-up-analyses for the sentence task, and then for the lexical and phonological task. The ANOVAs including follow-up-data were based on five Cellfield participants and three Placebo participants, thus comparisons between pre- and post-results, and pre-, post and follow-up-results need to be interpreted with particular caution.

#### **Behavioural Measures**

*Pre- and Post- Reaction Time and Accuracy Measures: Sentence Task.* For mean reaction time (RT) and accuracy data, the behavioural results presented for the sentence task were obtained using ANOVAs with the factors Group (Cellfield, Placebo), Time (pre, post) and Type (incongruent, congruent). Overall missing responses (calculated in percentages) were entered into an ANOVA, dropping the factor Type, as the interest was in investigating overall missing responses to the task.

For mean RT the ANOVA indicated a significant main effect of Time, F(1,10)=7.91, MSE=2564, p<0.05,  $\eta p^2=0.44$ . However the absence of a significant Time by Group interaction indicated that the effect did not differ between groups across time. Overall, mean RT decreased significantly from pre- (M=537.80, SE=30.46) to post-test (M=496.11, SE= 30.21). There was also a trend towards a significant main effect of Type, F(1,10)=4.06, MSE=5810, p=0.07,  $\eta p^2=0.29$ , showing that the incongruent sentence endings took longer to respond to (M=539.42, SE=32.53) than the congruent endings (M=494.49, SE=30.36). No significant effects involving group differences were obtained (ps>0.05).

Response accuracy varied significantly as a function of Type, F(1,10)= 14.74, MSE= 26.1, p < 0.05,  $\eta p^2 = 0.60$ , and the interaction between Group and Type tended towards significance, F(1,10)= 4.87, MSE= 26.1, p= 0.05,  $\eta p^2 = 0.33$ . The Cellfield group had a significantly higher accuracy for incongruent endings (M= 81.27, SE= 3.77) than congruent endings (M= 72.22, SE= 4.09). Taken together with the RT finding that incongruent sentences took longer to respond to, this suggests a speed-accuracy trade- off strategy used by the Cellfield group to respond more accurately to the incongruent sentences by slowing down reaction time. The accuracy data further revealed a trend towards a significant Time by Group interaction, F(1,10)= 4.74, MSE= 70.00, p= 0.05,  $\eta p^2= 0.32$ . Although Tukey posthoc tests did not reveal significant differences between individual means, Figure 10 suggests that response accuracy decreased from pre- (M= 79.52, SE= 4.34) to post-test (M= 73.97, SE= 3.91) (p= 0.35) for the Cellfield group and increased over time for the Placebo group ( $M_{pre}= 78.89$ , SE= 5.13;  $M_{post}= 84.00$ , SE= 4.63) (p= 0.55).

To investigate whether the two groups performed the tasks to their best possible ability, overall missing responses for both stimuli types together, were analysed. The ANOVA of missing responses revealed a trend towards significance for the interaction between Time and Group, F(1,10)=4.06, MSE=24.27, p=0.07,  $\eta p^2=0.29$ , and although Tukey post-hoc tests did not indicate any significant differences, Figure 11 suggests that the Cellfield group had more missing responses at post- (M=15.56, SE=2.65) compared to pretest (M=10.00, SE=3.49) (p=0.21), whereas no significant changes for missing responses were identified for the Placebo group (p=0.83).



*Figure 10.* Mean accuracy for Cellfield and Placebo group for the sentence task, at pre- and post-test.



*Figure 11.* Mean missing responses for Cellfield and Placebo group for the sentence task, at pre- and post-test.

*Pre-, Post- and Follow-up- Reaction Time and Accuracy Data: Sentence Task.* ANOVAs were performed in the same manner as the pre- and post-test ANOVAs, adding one level to the factor Time (pre, post, follow-up). For mean RT a trend towards significance for the main effect of Type was indicated, F(1,6)=5.12, MSE=3444, p=0.06,  $\eta p^2=0.46$ , showing that overall reaction time to congruent sentence endings (M=474.58, SE=31.00) was significantly faster than to incongruent endings (M=514.18, SE=42.33), confirming the result from the pre- and post-test ANOVA. The accuracy data showed a trend towards a significant effect of Type, F(1,6)=4.32, MSE=30.7, p=0.08,  $\eta p^2=0.42$ . Overall responses were significantly more accurate to incongruent sentence endings (M=79.02, SE=4.70) than to congruent endings (M=75.59, SE=4.92), thus showing the same results as the pre- and post-test ANOVAs. The follow-up-ANOVA for missing responses showed a significant Time by Group interaction, F(2,12)=7.88, MSE=34.02, p<0.05,  $\eta p^2=0.57$  (Figure 12), and Tukey post-hoc tests indicated that the Placebo group showed a trend towards significantly fewer missing responses at follow-up- (M=4.44, SE=4.86) compared to pre-test (M=20.00, SE=5.42) (p=0.06).



*Figure 12*. Mean missing responses for Cellfield and Placebo group for the sentence task, at pre-, post- and follow-up-test.

Post-hoc comparisons also confirmed that there were no significant pre-group differences for missing responses (p=0.71). The Cellfield group appeared to show the opposite pattern, displaying a non-significantly higher missing response rate at follow-up-(M=17.00, SE=3.77) compared to pre-test (M=8.67, SE=4.20) (p=0.28). The reader should remember that the Placebo group for this analysis consisted of only three participants.

*Pre- and Post- Reaction Time and Accuracy Data: Lexical and Phonological Task.* For mean RT and accuracy data the presented behavioural results for the lexical and phonological task were obtained using ANOVAs with the factors Group (Cellfield, Placebo), Time (pre, post), Task (lexical, phonological) and Type ('yes' responses, 'no' responses). Overall missing responses (calculated in percentages) were entered into an ANOVA, dropping the factor Type, as the interest was in investigating overall missing responses for the two tasks.

The analysis conducted on mean RT showed significant main effects of Time, F(1,10)=6.32, MSE=340504, p<0.05,  $\eta p^2=0.39$ , and Task, F(1,10)=32.70, MSE=219221, p<0.05,  $\eta p^2=0.77$ . The two-way interaction between these factors tended towards significance, F(1,10)=4.13, MSE=160624, p=0.07,  $\eta p^2=0.29$ . As confirmed by Tukey post-hoc tests, overall mean RT for the phonological task decreased significantly from pre-(M=1978.60, SE=141.81) to post-test (M=1506.48, SE=149.37) (p<0.05). Furthermore, overall the phonological task resulted in significantly longer mean RTs than the lexical task at pre- (M=1255.77, SE=109.78) and post-test (M=1120.77, SE=55.10) (p<0.05, Figure 13).

Response accuracy differed significantly as a function of Task, F(1,10)=51.32, MSE=232.5, p<0.05,  $\eta p^2=0.84$ , and Type, F(1,10)=7.54, MSE=269.4, p<0.05,  $\eta p^2=0.43$ . Overall the lexical task had a significantly higher response accuracy (M=73.61, SE=3.22) than the phonological task (M=50.99, SE=1.56) (p<0.05), indicating the higher difficulty of the phonological task. With regard to the significant main effect of Type, overall a significantly higher accuracy for 'yes' (M=66.96, SE=1.93) than 'no' responses (M=57.63, SE=3.14) (p<0.05) was achieved.



*Figure 13.* Mean reaction time for Cellfield and Placebo group together for the lexical and phonological task at pre- and post-test.

The absence of a significant Type by Task interaction indicates that accuracy for 'yes' and 'no' responses did not differ between the two tasks. A trend towards a significant three-way interaction was identified between Time, Task and Group, F(1,10)=4.32, MSE=69.9, p=0.06,  $\eta p^2=0.30$  (Figure 14). However, Tukey post-hoc tests only confirmed the significant differences from the main effect of Task, demonstrating overall higher accuracy for the lexical task than for the phonological task at pre- and post-test (ps<0.05). Inspection of the group means further suggest that the Placebo group had a higher accuracy at post- (M=56.50, SE=3.10) compared to pre-test (M=48.00, SE=3.27) (p=0.39) for the phonological task. To investigate whether the two groups performed the tasks to their best possible ability, overall missing responses, for both stimuli types together, were analysed.

A significant main effect of Task was found, F(1,10)=12.57, MSE=11.54, p<0.05,  $\eta p^2=0.56$ , indicating that overall the phonological task had a significantly higher missing response rate (M=4.81, SE=1.35) than the lexical task (M=1.29, SE=0.52) (p<0.05). This result confirms the RT and accuracy data for the phonological task, indicating a higher level of task difficulty for this task compared to the lexical task.



*Figure 14.* Mean accuracy for Cellfield and Placebo group for the lexical and phonological task at pre- and post-test.

### Pre-, Post- and Follow-up- Reaction Time and Accuracy Data: Lexical and

**Phonological Task.** ANOVAs were performed in the same manner as the pre- and post-test ANOVAs, just adding one level to the factor Time (pre, post, follow-up). For mean RT a trend towards a significant effect of Time was obtained, F(2,12)=3.82, MSE=287241, p=0.08,  $\eta p^2=0.40$ . Tukey post-hoc comparisons indicated that overall mean RT decreased significantly from pre- (M=1618.79, SE=174.13) to follow-up-test (M=1247.12, SE=105.50) (p<0.05). The absence of a significant Time by Task interaction demonstrates that the decrease in mean RT did not differ between the lexical and phonological task, a result contradictory to the finding of the pre- and post-test ANOVA. The ANOVA further revealed a significant effect of Task, F(1,6)=14.13, MSE=487911, p<0.05,  $\eta p^2=0.70$ , indicating that overall RT was significantly longer for the phonological task (M=1683.43, SE=159.79) than for the lexical task (M=1129.92, SE=101.76) (p<0.05). The main effect of Type was

a trend towards significance, F(1,6)=5.10, MSE=85448, p=0.06,  $\eta p^2=0.20$ , and the individual means indicated overall longer mean RTs for 'no' responses (M=1476.29, SE=137.34) than 'yes' responses (M=1337.06, SE=89.90), showing that the more difficult stimuli (pseudo homophones for the lexical task; nonwords for the phonological task) within each task required a longer RT.

Response accuracy differed significantly as a function of Task, F(1,6)=59.60, MSE=1213.2, p<0.05,  $\eta p^2=0.91$ , and showed a trend towards significance for the effect of Type, F(1,6)=5.12, MSE=535.9, p=0.06,  $\eta p^2=0.46$ . As reported for the pre- and post-test ANOVA, a significantly higher overall accuracy was achieved for the lexical task (M=76.54, SE=3.75) compared to the phonological task (M=52.78, SE=1.95) (p<0.05). Similarly, 'yes' responses resulted in a significantly higher accuracy (M=70.18, SE=2.21) than 'no' responses (M=59.14, SE=4.49) (p<0.05). Overall missing responses differed as a function of Task, F(1,6)=13.01, MSE=10.85, p<0.05,  $\eta p^2=0.68$ . The same significant differences as for the pre-and post-test ANOVA were indicated, showing an overall higher missing response rate for the phonological task (M=4.63, SE=1.38) compared to the lexical task (M=1.08, SE=0.54) (p<0.05).

## **ERP** Measures

In a similar manner to previous research on ERPs and linguistic processing, we focused on the later linguistic components of the ERP (N4 and LPC) for the sentence task, and included P2, N4 and LPC for the lexical and phonological tasks. For the LPC and N4 components of the sentence task, results were analysed with ANOVAs including the factors Group (Cellfield, Placebo), Time (pre, post), Type (incongruent, congruent), Sagittal sites (frontal, central, central-parietal) and Coronal sites (left, mid, right). For the P2, LPC and N4 components of the lexical and phonological task results were analysed with ANOVAs including the factors Group (Cellfield, Placebo), Time (pre, post), Task (lexical, phonological), Type ('yes' responses, 'no' responses), Sagittal sites (frontal, central, centralparietal) and Coronal sites (left, mid, right). The electrode sites used in the analyses for all ANOVAs were F3, Fz, F4, C3, Cz, C4, CP3, CPz, CP4.

### Sentence Task

*Pre-, Post- and Follow-up- Group Grand Mean Averages.* Figures 15 to 17 show group grand means for the sentence task at pre- and post-test, and Figures 18 to 20 show group grand means for pre- and follow-up-tests. The sentence task elicited a negative component around 100 ms, the N1 at frontal, central, central-parietal, and temporal-parietal sites, followed by a distinct positive peak around 200 ms, the P2. The N1 is an attention-related component and not specifically sensitive to linguistic stimuli, as it is held to reflect the perception of stimulus features in general. The N1 component was not included in the presented analyses and results. The P2 has been shown to distinguish between lexical features of linguistic stimuli, although the evidence is not conclusive. As commonly found in previous research, at Oz exclusively, polarity was inversed, showing a positive peak (P1) followed by a negative peak (N2). Positive and negative potentials at occipital sites are commonly elicited during visual paradigms and associated with the first initial visual word form analysis.

A negative deflection following the P2 component in the time window from 250 to 500 ms was identified as the N4. The last identified component was a positive component between 500 to 800 ms post stimulus, called the LPC. Both N4 and LPC have been associated with later linguistic processing, including discrimination and evaluation of linguistic material. As expected from previous research (for a review see Kutas et al., 2006), congruent endings of the sentence task elicited an N4 amplitude of much smaller magnitude than the incongruent endings (Figures 15, 16, 18, and 19). As in previous research (for a review see Pritchard et al., 1991) we also computed difference waveforms for the N4 of the sentence task for demonstration purposes only (Figures 17 and 20).



*Figure 15.* Group grand mean averages for incongruent endings in the sentence task at preand post-test.



*Figure 16.* Group grand mean averages for congruent endings in the sentence task at preand post-test.



*Figure 17*. Group grand mean averages for difference waveforms in the sentence task at preand post-test.



*Figure 18.* Group grand mean averages for incongruent endings in the sentence task at preand follow-up-test.



*Figure 19.* Group grand mean averages for congruent endings in the sentence task at preand follow-up-test.



*Figure 20*. Group grand mean averages for difference waveforms in the sentence task at preand follow-up-test.

**Pre- and Post-** N4 Amplitude. As expected N4 amplitude differed significantly as a function of Type, F(1,10)=15.88, MSE=78.96, p<0.05,  $\eta p^2=0.61$ , indicating overall significantly larger N4 amplitudes for incongruent sentence endings (M=-8.38, SE=0.91) than congruent endings (M=-4.93, SE=0.69) (p<0.05). A trend towards a significant interaction between Time, Coronal and Group was found, F(2,20)=2.85, MSE=2.52, p=0.08,  $\eta p^2=0.22$ . Tukey post-hoc tests did not indicate any significant pre- to post-test differences, however, the Cellfield group had significantly larger N4 amplitudes at post-test at left (M=-6.41, SE=1.00) and mid sites (M=-6.23, SE=1.12) compared to right sites (M=-4.89, SE=0.70) (p<0.05, Figure 21). At pre-test this effect of coronal was not present showing N4 amplitudes of similar magnitude for the Cellfield group across left, mid, and right sites.



*Figure 21*. Mean N4 amplitude for Cellfield and Placebo group, at left, mid, and right sites, at pre- and post-test.

For the Placebo group Tukey post-hoc tests indicated the opposite pattern: At posttest N4 amplitude was significantly smaller at left sites (M= -6.43, SE= 1.19) compared to mid (M= -8.01, SE= 1.32) and right sites (M= -7.80, SE= 0.83) (ps< 0.05). However the Placebo group showed a trend towards significantly larger N4 amplitudes at midline (M= - 8.14, SE= 1.25) compared to left sites (M= -6.71, SE= 1.24) (p= 0.07) at pre-test. No differences between the two groups at pre- and post-test were significant (ps> 0.05).

*Pre-and Post- N4 Latency.* The ANOVA did not produce any significant or trends towards significant main effects or interactions involving Time or Group.

*Pre-, Post- and Follow-up- N4 Amplitude.* The ANOVA revealed a significant main effect of Type, F(1,6)=13.08, MSE=77.28, p<0.05,  $\eta p^2=0.69$ . As for the pre- and post-ANOVA the incongruent endings elicited significantly larger N4 amplitudes (M=-8.05, SE=1.24) than the congruent endings (M=-4.89, SE=0.75) (p<0.05). The main effect of Time tended towards significance, F(2,12)=4.10, MSE=13.08, p=0.06,  $\eta p^2=0.41$  and Tukey post-hoc comparisons indicated a trend towards overall significantly decreased N4 amplitudes from pre- (M=-7.01, SE=0.96) to follow-up-test (M=-5.77, SE=1.05) (p=0.09). The ANOVA also indicated a trend towards a significant interaction between Group, Sagittal and Coronal, F(4,24)=2.73, MSE=5.28, p=0.08,  $\eta p^2=0.31$ . Tukey post-hoc tests revealed that only the Cellfield group had significantly smaller N4 amplitudes at the right central-parietal site (M=-4.00, SE=1.23) compared to the right frontal (M=-6.67, SE=1.01) and also the left central-parietal site (M=-6.32, SE=1.47) (ps<0.05). No other significant distributional differences or differences between the two groups were indicated.

*Pre-, Post- and Follow-up- N4 Latency.* The ANOVA revealed a significant main effect of Time, F(2,12)=4.36, MSE=6455, p<0.05,  $\eta p^2=0.42$ , and Tukey post-hoc tests confirmed overall significantly longer N4 latencies at follow-up- (M=361.45, SE=19.97) compared to post-test (M=334.54, SE=17.77) (p<0.05), and non-significantly shorter N4 latencies at post- (M=334.54, SE=17.77) compared to pre-test (M=357.07, SE=22.66) (p=0.10). No effects involving Group reached significance.

**Pre- and Post- LPC Amplitude.** The ANOVA indicated a trend towards a significant three-way interaction between Time, Coronal and Group, F(2,20)=3.96, MSE=2.72, p=0.05,  $\eta p^2=0.28$ . Although Tukey post-hoc tests did not indicate significant comparisons, the group means suggest that LPC amplitude decreased from pre- (M=5.87, SE=0.67) to post-test (M=4.97, SE=0.47) (p=0.30) for the Cellfield group at right

electrode sites selectively. The opposite effect occurred for the Placebo group, showing a smaller LPC amplitude at post- (M= 5.63, SE= 0.82) compared to pre-test (M= 6.87, SE= 0.96) (p= 0.21) at left electrode sites selectively (Figure 22). The two groups' LPC amplitudes at pre-test at the left and right sites did not differ significantly (ps> 0.05), suggesting bilateral activation on this measure for both groups at pre-test.



*Figure 22*. Mean LPC amplitude for Cellfield and Placebo group at left, mid, and right sites, at pre- and post-test.

With regard to Group effects the ANOVA showed a significant interaction between Group and Sagittal, F(2,20)=7.71, MSE=5.59, p<0.05,  $\eta p^2=0.44$ , and Tukey post-hoc tests indicated significant LPC amplitude differences only for the Placebo group, with significantly smaller LPC amplitudes at frontal sites (M=4.72, SE=0.73) compared to central- (M=6.63, SE=0.71) and central-parietal sites (M=7.29, SE=0.67) (ps<0.05). No significant distributional differences within the Cellfield group were evident.

**Pre- and Post- LPC Latency.** For LPC latency the ANOVA did not indicate theoretically relevant significant or tending towards significance main effects or interactions involving Time or Group (ps> 0.05). However, the analysis revealed a trend towards a

significant interaction involving Time, Sagittal and Coronal, F(4,40)=2.67, MSE=2804, p=0.06,  $\eta p^2=0.21$ . Tukey post-hoc tests did not indicate significant comparisons, and the only theoretically relevant time effect occurred at the mid central site, showing overall decreased LPC latency at post- (M=651.60, SE=12.64) compared to pre-test (M=697.36, SE=16.95) (p=0.32, Figure 23).



*Figure 23*. Mean LPC latency for Cellfield and Placebo group together at frontal, central, and central-parietal sites, over left, mid, and right sites, at pre- and post-test.

**Pre-, Post- and Follow-up- LPC Amplitude.** For LPC amplitude a trend towards significance for the main effect of Time was observed, F(2,12)=4.79, MSE=15.19, p=0.06,  $\eta p^2=0.44$ , which was further qualified by a significant interaction between Time and Type, F(2,12)=5.04, MSE=10.40, p<0.05,  $\eta p^2=0.46$ . As shown in Figure 24 and confirmed by Tukey post-hoc tests, overall LPC amplitude decreased significantly from pre- (M=6.07, SE=0.91) to follow-up-test (M=3.53, SE=0.52) (p<0.05) and tended towards a significant decrease from post- (M=5.32, SE=0.50) to follow-up-test (M=3.53, SE=0.52) (p=0.05) for the congruent endings only. In addition, the incongruent and congruent endings had significantly different LPC amplitudes only at follow-up-test, with incongruent endings

eliciting larger LPC amplitudes (M= 5.68, SE= 0.61) than congruent endings (M= 3.53, SE= 0.52) (p < 0.05).



*Figure 24*. Mean LPC amplitude for Cellfield and Placebo group together for incongruent and congruent endings, at pre-, post- and follow-up-test.

The ANOVA further indicated a significant interaction between Time, Coronal and Group, F(4,24)=4.90, MSE=1.87, p<0.05,  $\eta p^2=0.45$ . Tukey post-hoc comparisons confirmed significant differences between the two groups: The Cellfield group showed significantly decreased LPC amplitudes from pre- (M=5.98, SE=0.96) to post-test (M=4.58, SE=0.34) and from pre- (M=5.98, SE=0.96) to follow-up-test (M=3.74, SE=0.25) (p<0.05) at right electrode sites and similarly significantly decreased LPC amplitude from pre- (M=5.97, SE=0.78) to follow-up-test (M=4.38, SE=0.70) (p<0.05) at midline electrode sites (Figure 25). In addition at follow-up only, the difference between LPC amplitude at left sites and right sites was significant, showing significantly larger LPC amplitudes at left (M=5.24, SE=3.74) compared to right sites (M=3.74, SE=0.25) (p<0.05) for the Cellfield group. Tukey post-hoc test also revealed that left and right sites did not differ at pre-test for the Cellfield and Placebo group (p<0.05).

In contrast, the Placebo group showed a significant decrease in LPC amplitude from pre- (M= 6.61, SE= 1.31) to follow-up-test (M= 4.53, SE= 0.88) (p< 0.05) and nonsignificantly from pre- (M= 6.61, SE= 1.31) to post-test (M= 5.02, SE= 0.97) (p= 0.11) at left electrode sites, however the difference between left and right sites did not reach significance at post- or follow-up-test (ps> 0.05), showing bilaterally LPC amplitudes of similar magnitude for the Placebo group. At midline sites, the Placebo group showed the same effect as the Cellfield group with significantly decreased LPC amplitudes at follow-up- (M= 5.03, SE= 0.90) compared to pre-test (M= 6.79, SE= 1.00) (p< 0.05). These different distributional related changes in LPC amplitude for the two groups will be discussed in Chapter 10.



*Figure 25*. Mean LPC amplitude for Cellfield and Placebo group, at left, mid, and right sites, at pre-, post- and follow-up-test.

**Pre-, Post- and Follow-up- LPC Latency.** The ANOVA showed a trend towards significance for the interaction between Time and Group, F(2,12)=3.13, MSE=14198, p=0.08,  $\eta p^2=0.34$ , and although Tukey post-hoc tests did not demonstrate significant effects, the group means suggest that the Placebo group had shorter LPC latencies at post- (M=

643.34, SE= 24.92) compared to pre-test (M= 709.52, SE= 16.31) (p= 0.11, Figure 26). No significant LPC latency variations were identified for the Cellfield group.



*Figure 26.* Mean LPC latency for Cellfield and Placebo group, at pre-, post- and follow-up-test.

# Lexical and Phonological Task

*Pre-, Post- and Follow-up- Group Grand Mean Averages.* In the sentence task, four peaks were identified for the lexical and phonological task, N1, P2, N4 and LPC at preand post-test (Figures 27 to 30), and at follow-up-test (Figures 31 to 34). For a detailed description of the components the reader is referred to the beginning of this section in this chapter.



*Figure 27*. Group grand mean averages for pseudo homophones in the phonological task at pre- and post-test.



*Figure 28.* Group grand mean averages for nonwords in the phonological task at pre- and post-test.






*Figure 30.* Group grand mean averages for pseudo homophones in the lexical task at preand post-test.





*Figure 31*. Group grand mean averages for the pseudo homophones in the phonological task at pre- and follow-up-test.



*Figure 32*. Group grand mean averages for the nonwords in the phonological task at preand follow-up-test.







*Figure 34.* Group grand mean averages for the pseudo homophones in the lexical task at pre- and follow-up-test.

*Pre- and Post- P2 Amplitude*. All results presented for P2, N4, and LPC were obtained with ANOVAs including the factors Group (Cellfield, Placebo), Time (pre, post), Task (lexical, phonological), Type ('yes' responses, 'no' responses), Sagittal sites (frontal, central, central-parietal) and Coronal sites (left, mid, right). The electrode sites for the analyses were F3, Fz, F4, C3, Cz, C4, CP3, CPz, CP4.

The ANOVA indicated a trend towards significance for the main effect of Group, F(1,10) = 4.81, MSE = 1191.01, p = 0.05,  $\eta p^2 = 0.32$ , and the group means showed significantly larger P2 amplitudes for the Cellfield group (M=10.49, SE=1.54) than the Placebo group (M=5.26, SE=1.82) (p<0.05). P2 amplitude differed as a function of Time as indicated by a significant main effect of Time, F(1,10)=9.24, MSE= 82.20, p<0.05,  $\eta p^{2}=$ 0.48. The main effect of Time was further qualified by three interactions, none of which included Group. The interaction between Time and Type reached significance, F(1,10)=5.39, MSE= 36.91, p < 0.05,  $\eta p^2 = 0.35$ , the interaction between Time and Sagittal, F(2,20) =3.49, MSE= 5.88, p=0.08,  $\eta p^2=0.26$ , tended towards significance, as did the interaction of Time, Type, Sagittal, and Coronal, F(4,40) = 2.74, MSE = 2.50, p = 0.08,  $\eta p^2 = 0.21$ . The fourway interaction will be considered further. Tukey post-hoc tests indicated that overall P2 amplitudes decreased significantly for 'yes' responses from pre- (Ms > 8.39, SE = 1.04) to post-test (Ms < 7.37, SE = 1.59) (p < 0.05) across electrode sites, whereas P2 amplitude for the 'no' responses selectively decreased significantly from pre- (Ms > 8.51, SE = 1.54) to posttest (Ms < 7.39, SE = 1.56) (p < 0.05) at mid and right frontal sites. As the overall distributional differences for the 'yes' and 'no' response types are not of theoretical relevance comparisons involving sagittal and coronal sites will not be described.

**Pre- and Post- P2 Latency.** The ANOVA indicated a trend towards a significant interaction between Time and Type, F(1,10)=4.24, MSE=701, p=0.07,  $\eta p^2=0.29$ , which was further qualified by a significant three-way interaction involving Time, Type and Group, F(1,10)=6.03, MSE=701, p<0.05,  $\eta p^2=0.37$ , and a trend towards a significant interaction of Time, Task, and Type, F(1,10)=4.57, MSE=2279, p=0.06,  $\eta p^2=0.31$ . Moreover, the five-way interaction involving Time, Task, Type, Sagittal and Group, F(2,20)=2.92, MSE=735, p=0.09,  $\eta p^2=0.23$ , tended towards significance. A break-down

ANOVA, dropping the Coronal factor and analysing the three sagittal sites (frontal, central, central-parietal) separately, was performed to investigate this interaction further. None of the three break-down ANOVAs revealed theoretically relevant significant comparisons with Tukey post-hoc tests (ps> 0.05).

*Pre-, Post- and Follow-up- P2 Amplitude.* All follow-up-test ANOVAs for P2, N4, and LPC component were performed in the same manner as for the pre-post analyses, adding one level of Time (pre, post, follow-up) to the analyses.P2 amplitude differed as a function of Time, as indicated by a trend towards a significant main effect, F(2,12)=3.40, MSE=103.12, p=0.07,  $\eta p^2=0.40$ . There was also a trend towards significance for the interaction between Time and Group, F(2,12)=3.69, MSE=103.12, p=0.08,  $\eta p^2=0.48$ . Tukey post-hoc tests confirmed significantly decreased P2 amplitude for the Cellfield group at follow-up-test (M=5.39, SE=0.97) compared to pre-test (M=9.08, SE=1.68) (p<0.05, Figure 35).



Figure 35. Mean P2 amplitude for Cellfield and Placebo group, at pre-, post- and follow-up-

test.

ť

136

The ANOVA also indicated a trend towards a significant three-way interaction between Type, Coronal and Group, F(2,12)=3.49, MSE=3.93, p=0.08,  $\eta p^2=0.34$ . The only distributional difference in P2 amplitude for the Cellfield group indicated by Tukey post-hoc tests was a trend towards significantly smaller P2 amplitudes at left sites for the 'yes' responses (M=7.13, SE=1.58) compared to midline sites (M=8.21, SE=1.63) (p=0.08), and no significant differences for the right sites. More significant differences were identified for the Placebo group with larger P2 amplitudes at left and mid sites for 'yes'(Ms>5.43, SE=2.10) and 'no' responses (Ms>6.57, SE=2.31) compared to 'yes' and 'no' responses at right sites (Ms<5.06, SE=1.76) (ps<0.05).

*Pre-, Post- and Follow-up- P2 Latency.* The ANOVA revealed no significant main effects, or significant or tending towards significance interactions involving Time or Group. The four-way interaction between Task, Type, Coronal and Group tended towards significance, F(2,12)=4.48, *MSE*= 387, p=0.05,  $\eta p^2=0.43$ , however Tukey post-hoc tests did not indicate any trends towards significance or significant comparisons (*ps*> 0.05).

*Pre- and Post- N4 Amplitude.* N4 amplitude differed as a function of Time, Sagittal, Coronal and Group as indicated by a trend towards a significant interaction between these factors, F(4,40)=2.78, MSE=3.25, p=0.08,  $\eta p^2=0.22$  (Figures 36a, b, c).Tukey post-hoc tests indicated that overall the Placebo group showed an increased N4 amplitude at post- (Ms>-10.34, SE=2.27) compared to pre-test (Ms<-10.17, SE=2.59) at selective electrode sites, including midline central-parietal sites (p=0.10), right central (p<0.05), and right central-parietal sites (p=0.10). In contrast, although not significantly, the Cellfield group showed a diminished N4 amplitude at post- (M=-3.74, SE=1.92) compared to pre-test (M=-5.27, SE=1.47) (p=0.38) at the right central-parietal site. The smaller N4 amplitude at the right central-parietal site for the Cellfield group, was not accompanied by an increased N4 amplitude at the left central-parietal site, however the N4 amplitude remained of similar magnitude, with a slight non-significant increase in amplitude at this particular site at post-test as shown by planned comparisons ( $M_{pre}=-5.68$ , SE=1.87;  $M_{post}=-5.95$ , SE=2.26). Moreover, whereas N4 amplitude at the left central-parietal site did not differ significantly from the N4 amplitude at the right central-parietal site at pre-test for the Cellfield group (p=1.00) or for the Placebo group (p=1.00), Tukey post-hoc tests indicated that at post-test only the Cellfield group had a significantly larger N4 amplitude at the left central-parietal site (M=-5.95, SE=2.26) than at the right central-parietal site (M=-3.74, SE=1.92) (p<0.05). This suggests bilateral processing of the Cellfield and Placebo group at this particular site at pre-test, but less right and more left lateralised processing at post-test for the Cellfield group.

In addition, the ANOVA revealed two interactions involving Time. The first interaction between Time and Type, F(1,10)=4.90, MSE=56.09, p=0.05,  $\eta p^2=0.33$ , and the second interaction between Time, Task, Sagittal, and Coronal, F(4,40)=3.36, MSE=4.07, p=0.05,  $\eta p^2=0.25$ , tended towards significance. However, for both interactions Tukey post-hoc tests did not show any theoretically relevant significant or tending towards significant effects.



*Figure 36a*. Mean N4 amplitude for Cellfield and Placebo group at left frontal, left central, and left central-parietal sites, at pre- and post-test.



*Figure 36b*. Mean N4 amplitude for Cellfield and Placebo group at mid frontal, mid central, and mid central-parietal sites, at pre- and post-test.



*Figure 36c.* Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre- and post-test.

With regard to Group effects, the ANOVA indicated a significant interaction of Task, Type and Group, F(1,10)=5.58, MSE=27.04, p<0.05,  $\eta p^2=0.36$ , which was qualified by a trend towards a significant four-way interaction between Task, Type, Group and Sagittal F(2,20)=3.90, MSE=12.70, p=0.06,  $\eta p^2=0.28$ . Tukey post-hoc tests revealed significant differences only for the Cellfield group, showing larger N4 amplitudes at frontal sites for the pseudo homophones ('no' responses) of the lexical task (M=-8.79, SE=1.65) and the pseudo homophones ('yes' responses) of the phonological task (M=-9.29, SE=1.58) than at central-parietal sites (for the lexical task: M=-5.19, SE=1.84; for the phonological task: M=-5.97, SE=2.04) (ps<0.05). No differences between the two groups were significant (ps>0.05).

**Pre- and Post-** N4 Latency. The ANOVA showed a trend towards significance for the main effect of Task, F(1,10)=3.75, MSE=26727, p=0.08,  $\eta p^2=0.27$ , which was moderated by a significant interaction between Time and Task, F(1,10)=8.36, MSE=6789, p<0.05,  $\eta p^2=0.46$  (Figure 37). Tukey post-hoc tests indicated overall significantly longer N4 latencies for the phonological (M=355.83, SE=13.88) compared to the lexical task (M=317.54, SE=12.76) (p<0.05) at post-test, and although Tukey post-hoc tests did not reveal any significant comparisons from pre- to post-test, the task means suggest that for the lexical task N4 latency overall decreased from pre- (M=339.98, SE=11.54) to post-test (M=317.54, SE=12.76) (p=0.11), but not for the phonological task.

With regard to Group effects, the ANOVA revealed a trend towards significance for the interaction of Type, Sagittal, Coronal and Group, F(4,40)=3.00, MSE=531, p=0.06,  $\eta p^2=0.23$ . Tukey post-hoc tests confirmed a few significant distributional activation patterns for 'yes' and 'no' responses for the two groups. The Cellfield group had significantly longer N4 latencies at the left frontal site for 'yes' responses (M=375.50, SE=11.60) compared to the left central site (M=344.57, SE=19.08) and also in comparison to the 'no' responses at the left frontal site (M=351.11, SE=15.93) (p<0.05). The same pattern was observed for the mid frontal site, showing significantly longer N4 latencies (M=378.75, SE=15.34) than the mid central-parietal site (M=350.68, SE=12.69) (p<0.05).



*Figure 37*. Mean N4 latency for Cellfield and Placebo group together for the lexical and phonological task, at pre- and post-test.

For the Placebo group a similar pattern was identified, with significantly longer N4 latencies for 'yes' responses at the left frontal site (M= 335.95, SE= 13.72) and the mid central site (M= 343.20, SE= 16.49) compared to the left central site (M= 303.30, SE= 22.58) (ps< 0.05). In addition, the 'no' responses had a significantly longer N4 latency at the mid frontal site (M= 342.30, SE= 17.36) compared to the mid central-parietal site (M= 308.50, SE= 14.28) (p< 0.05). No significant or trend toward significant differences between the two groups were indicated. Overall the distributional latency differences suggest a longer frontal engagement for both groups.

**Pre-, Post- and Follow-up-** N4 Amplitude. The interaction between Time and Group tended towards significance, F(2,12)=3.82, MSE=51.97, p=0.08,  $\eta p^2=0.39$ , and was further qualified in a trend towards a significant four-way interaction between Time, Sagittal, Coronal, and Group, F(8,48)=2.46, MSE=2.80, p=0.09,  $\eta p^2=0.29$ . Tukey posthoc tests revealed a significant increase in N4 amplitude for the Cellfield group from pre-(M=-5.35, SE=2.73) to follow-up-test (M=-7.70, SE=2.05) (p<0.05) at the left central-

parietal site exclusively (Figure 38a). The reader is reminded that the pre-post ANOVA reported previously, showed a non-significant increase at this particular site for the Cellfield group. No other effects reached or tended towards significance for this group.

Within the Placebo group trends towards significant (ps < 0.07) and significant comparisons (ps < 0.05) were mainly indicated for the midline electrode sites, with decreased N4 amplitudes at the mid frontal site from pre-(M= -11.00, SE= 2.85) to followup-test (M= -8.11, SE= 3.22) and post- (M= -12.04, SE= 2.22) to follow-up-test (M= -8.11, SE= 3.22), at the mid central site from pre- (M= -12.51, SE= 3.91) to follow-up-test (M= -9.65, SE= 3.20), and at the mid central-parietal site from post- (M= -14.00, SE= 4.06) to follow-up-test (M= -10.14, SE= 2.87) (Figure 38b). Moreover, the Placebo group showed significantly larger N4 amplitude at the right central-parietal site at post-test (M= -8.75, SE= 0.77) compared to the left central-parietal site (M= -7.95, SE= 1.94) (p< 0.05), thus showing the opposite pattern to the Cellfield group (Figure 38c). The N4 amplitude was not significantly different at these two left and right sites for the Placebo group at pre-test (p> 0.05). Tukey post-hoc tests also confirmed that the two groups' N4 amplitudes at pre-test did not differ significantly from each other (p> 0.05).

**Pre-, Post- and Follow-up- N4 Latency.** The only effect observed was a trend towards significance for the main effect of Task, F(1,6)=4.97, MSE=30231, p=0.07,  $\eta p^2=0.45$ , and no interactions involving Time or Group reached significance or tended towards significance. Overall the phonological task tended to have a significantly longer N4 latency (M=350.16, SE=15.58) than the lexical task (M=322.94, SE=13.28).



*Figure 38a.* Mean N4 amplitude for Cellfield and Placebo group at left frontal, left central, and left central-parietal sites, at pre-, post-, and follow-up-test.



*Figure 38b.* Mean N4 amplitude for Cellfield and Placebo group at mid frontal, mid central, and mid central-parietal sites, at pre-, post-, and follow-up-test.



*Figure 38c.* Mean N4 amplitude for Cellfield and Placebo group at right frontal, right central, and right central-parietal sites, at pre-, post-, and follow-up-test.

**Pre- and Post- LPC Amplitude.** The ANOVA revealed a trend towards a significant interaction between Time, Task and Group, F(1,10)=3.99, MSE=56.02, p=0.07,  $\eta p^2=0.29$ , and a significant interaction between Time and Type was found, F(1,10)=16.58, MSE=21.30, p<0.05,  $\eta p^2=0.62$ . Both interactions were qualified by a trend towards a significant higher-order five-way interaction between Time, Task, Type, Sagittal and Group, F(2,20)=2.76, MSE=6.25, p=0.09,  $\eta p^2=0.22$ .

To investigate this interaction further, break-down ANOVAs were performed for frontal, central and central-parietal sites separately, averaged over coronal sites. The first break-down ANOVA at frontal sites revealed a significant main effect of Type, F(1,10)=5.60, MSE=6.06, p<0.05,  $\eta p^2=0.36$ , showing overall significantly larger LPC amplitudes for 'no' responses (M=3.69, SE=0.94) than 'yes' responses (M=2.48, SE=0.84) (p<0.05). The interaction between Time, Task and Group reached significance, F(1,10)=5.00, MSE=5.71, p<0.05,  $\eta p^2=0.33$ , and although Tukey post-hoc tests did not indicate significant comparisons, the group means suggest that at the frontal sites the Cellfield group had a larger LPC amplitude for the lexical task (M=4.74, SE=1.33) compared to the phonological task (M=2.61, SE=1.20) (p=0.35) at post-test only (Figure 39). The graph further suggests that for the Cellfield group the LPC amplitude decreased from pre- (M=4.33, SE=1.04) to post-test (M=2.61, SE=1.20) (p=0.55) for the phonological task at frontal sites. No significant amplitude differences appeared to be evident for the Placebo group (ps>0.05).



*Figure 39.* Mean LPC amplitude for Cellfield and Placebo group for the lexical and phonological task, at frontal sites, at pre- and post-test.

The second break-down ANOVA at central sites indicated a significant Time by Type interaction F(1,10)=17.78, MSE=2.38, p<0.05,  $\eta p^2=0.64$ , which was further qualified by a significant interaction between Time, Type and Group, F(1,10)=5.34, MSE=2.38, p<0.05,  $\eta p^2=0.35$ . As shown in Figure 40 and confirmed by Tukey post-hoc tests the Placebo group showed significantly decreased LPC amplitudes from pre-(M=5.52, SE=1.20) to post-test (M=2.85, SE=1.46) (p<0.05) for 'yes' responses at central sites. In addition, the 'no' responses resulted in significantly larger LPC amplitudes (M=7.19, SE=2.07) than the 'yes' responses (M=2.85, SE=1.46) (p<0.05) at post-test for the Placebo

group. No significant differences in LPC amplitude were identified for the Cellfield group at these sites (ps> 0.05).



*Figure 40.* Mean LPC amplitude for Cellfield and Placebo group for the lexical 'yes'- and 'no' responses, at central sites, at pre- and post-test.

A trend towards a significant interaction of Time, Task and Group was also found, F(1,10)=4.33, MSE=7.81, p=0.06,  $\eta p^2=0.30$ , and Tukey post-hoc tests demonstrated a trend towards significance for the Cellfield group. Similarly to frontal sites, at post-test only LPC amplitude was larger in response to the lexical task (M=9.16, SE=1.70) than the phonological task (M=5.35, SE=1.23) (p=0.06, Figure 41). In addition, whereas at frontal sites LPC amplitude appeared to have decreased for the phonological task from pre- to posttest for the Cellfield group, at central sites LPC amplitude was of similar magnitude for the phonological task at pre- (M=6.60, SE=1.08) and post-test (M=5.35, SE=1.23). No significant differences in LPC amplitude were observed for the Placebo group (ps>0.05).



*Figure 41*. Mean LPC amplitude for Cellfield and Placebo group for the lexical and phonological task, at central sites, at pre- and post-test.

Finally, the last break-down ANOVA at central-parietal sites showed a significant interaction between Time and Type, F(1,10)=19.04, MSE=3.20, p<0.05,  $\eta p^2=0.66$ , and Tukey post-hoc tests confirmed that overall LPC amplitudes increased significantly at central-parietal sites for 'no' responses from pre- (M=5.98, SE=1.10) to post-test (M=8.31, SE=1.37) (p<0.05, Figure 42). Additionally at post-test the LPC amplitude for 'no' responses was significantly larger (M=8.31, SE=1.37) than for 'yes' responses (M=6.22, SE=1.27) (p<0.05), whereas at pre-test, the opposite pattern was observed: Although not significantly the 'yes' responses had a larger LPC amplitude at pre-test (M=7.11, SE=0.77) than the 'no' responses (M=5.98, SE=1.10) (p=0.15).



*Figure 42*. Mean LPC amplitude for Cellfield and Placebo group together for the lexical and phonological task, at central-parietal sites, at pre- and post-test.

*Pre- and Post- LPC Latency.* The ANOVA indicated a significant interaction between Time, Coronal and Group, F(2,20)=4.93, MSE=3284, p<0.05,  $\eta p^2=0.33$ . As shown in Figure 43 and confirmed by Tukey post-hoc tests the Cellfield group showed a trend towards significantly longer LPC latencies at left electrode sites (M=656.10, SE=14.04) compared to mid electrode sites (M=626.11, SE=17.16) (p=0.09) and nonsignificantly also compared to right sites (M=631.45, SE=13.40) (p=0.26) at post-test only. The opposite seemed to be evident for the Placebo group and although Tukey post-hoc tests did not indicate significant differences, LPC latency was shorter at left electrode sites (M=619.32, SE=16.61) than right electrode sites (M=648.93, SE=15.86) (p=0.24) at post-test only.

The ANOVA further indicated a significant interaction between Task and Type, F(1,10)= 5.34, *MSE*= 32155, p < 0.05,  $\eta p^2 = 0.35$ , and Tukey post-hoc tests confirmed a trend towards a significant difference between the real words of the lexical task ('yes' responses) and pseudo homophones of the phonological task ('yes' responses), with the pseudo homophones showing an overall longer LPC latency (M=665.71, SE=12.40) than the real words (M=616.52, SE=17.01) (p=0.06).



*Figure 43*. Mean LPC latency for Cellfield and Placebo group at left, mid, and right sites, at pre- and post-test.

**Pre-, Post- and Follow-up- LPC Amplitude.** The ANOVA indicated a significant five-way interaction between Time, Task, Type, Sagittal and Group, F(4,24)=4.77, *MSE*= 5.20, p < 0.05,  $\eta p^2 = 0.44$ . As for the pre- and post-results, break-down ANOVAs were performed at each sagittal site (frontal, central, central-parietal) to investigate this interaction further.

The first break-down ANOVA at frontal sites indicated a trend towards significance for the main effect of Type, F(1,6)=4.54, MSE=6.90, p=0.08,  $\eta p^2=0.43$ , showing overall significantly larger LPC amplitudes for the 'no' responses (M=3.20, SE=1.17) than the 'yes' responses (M=2.02, SE=0.82) (p<0.05). LPC amplitude further varied as a function of Time and Group as indicated by a trend towards a significant interaction between these factors, F(2,12)=3.99, MSE=2.65, p=0.07,  $\eta p^2=0.40$ . Although Tukey post-hoc tests did not indicate significant comparisons, Figure 44 suggests that the Placebo group had LPC amplitudes of similar magnitude at pre- (M= 2.32, SE= 1.55) and post-test (M= 2.33, SE= 1.79) (p> 0.05), but increased amplitudes at follow-up-test (M= 3.89, SE= 1.40).



*Figure 44*. Mean LPC amplitude for Cellfield and Placebo group at pre-, post- and followup-test.

The second break-down ANOVA at central sites did not reveal any significant effects or trends towards significance. The third break-down ANOVA at central-parietal sites produced a trend towards significance for the interaction between Time and Type, F(2,12)=3.43, MSE= 7.44, p= 0.08,  $\eta p^2=$  0.36, however no significant comparisons were identified with Tukey post-hoc tests (ps> 0.05). Whereas the pre- and post- break- down ANOVA revealed overall significantly larger LPC amplitudes at post- compared to pre-test for 'no' responses, inclusion of follow-up-data only showed an overall non-significant increase in LPC amplitude from pre- (M= 4.97, SE= 1.49) to post-test (M= 7.54, SE= 2.00) (p= 0.21) for the 'no' responses.

In summary, no significant LPC amplitude variations for the Cellfield group were indicated by the break-down ANOVAs, and the Placebo group showed only a nonsignificant increase of LPC amplitude at frontal electrode sites. **Pre-, Post- and Follow-up- LPC Latency.** The ANOVA revealed a trend towards a significant main effect of Task, F(1,6)=3.99, MSE=32694, p=0.06,  $\eta p^2=0.47$ , which was qualified by a trend towards a significant two-way interaction of Task and Type, F(1,6)=5.70, MSE=28161, p=0.05,  $\eta p^2=0.49$ . As for the pre- and post-test ANOVA, Tukey post-hoc tests confirmed significantly longer LPC latencies for the pseudo homophones ('yes' responses) of the phonological task (M=686.19, SE=14.52) than the real words ('yes' responses) of the lexical task (M=628.67, SE=18.82) (p<0.05).

#### **Chapter 10: Discussion**

The aim of the current study was to provide an evaluation of the Cellfield intervention for the treatment of dyslexia, using behavioural (reading and related skills, reaction time, and accuracy measures) and neural indicators (ERPs) as intervention outcome measures. First, the outcomes on the literacy measures will be discussed, followed by a discussion of the findings of the ERP experiments, and lastly an integration of the findings for the literacy and neural outcomes will be provided concluding with a summary of the limitations of the study and recommendations for future intervention research.

It should be noted that generally the participant numbers were small (7 Cellfield, and 5 Placebo participants) due to the given time frame of the project. With regard to the experimental data (behavioural and ERP data) it should further be noted: Although comparisons between pre- and post-data and pre-, post- and follow-up-data are made the interpretations need to be taken with caution since the sample size for the follow-up was particularly small (5 Cellfield, and 3 Placebo participants).

#### Literacy Outcomes

Literacy outcomes are of most direct relevance in estimating the efficacy of the Cellfield intervention. Subsequently four major streams of evidence will be discussed: (1) The impact of the Cellfield intervention on phonological skills, (2) The lack of gains in higher-order skills such as text reading accuracy and comprehension following the Cellfield intervention, (3) The absence of any gains in reading rate and spelling following the Cellfield and followon practice program, and (4) Motivational and perceived self-effectiveness aspects.

#### The Impact of the Cellfield Intervention on Phonological Skills

Considerable gains in phonological skills were achieved post intervention by the Cellfield group only, as assessed by the word attack subtest from the WRMT-R. Although the effect size was rather small ( $\eta p^2 = 0.35$ ), the Cellfield group achieved a mean standard score of 89.29 at post-test, and thus was no longer one or more than one standard deviation below average performance. This finding replicates that of the previous evaluation study of the

Cellfield intervention by Prideaux et al. (2005) who reported significant gains in the word attack measure following the Cellfield intervention. The gain in word attack skills in the present study was maintained at follow-up, showing that the Cellfield participants did retain their improved phonological skills. However, a further improvement was not observed. As the Cellfield intervention contains a large amount of phonology-based exercises this finding was as expected. The finding of strengthened phonological decoding skills is also in line with current evidence from other intervention studies frequently reporting gains in phonological skills following various intervention programs (e.g., Foorman et al., 1998, 2003; Torgesen et al., 1997a, 1997b). In contrast, the Placebo group showed only a non-significant small improvement in phonological decoding skills at post- and follow-up-test, and the group mean standard score remained one standard deviation below the mean (M= 82.00).

ļ

Another significant interaction between Time and Group  $(\eta p^2 = 0.35)$  was achieved on the DST-S, which consists of a variety of subtests to track major problem areas in children with dyslexia. The Cellfield group showed a significant decrease in their overall risk for dyslexia from pre- to post-test, whereas the Placebo group's at-risk index decrease was smaller and not significant. The mean group at-risk index for the Cellfield group decreased from 1.25 (at-risk) to 0.84 (mild risk). A further break-down analysis of the subtests from the DST-S revealed only time effects, showing that both groups improved in various subtests from pre- to post-test. However, a closer investigation of the group means showed that the Cellfield group had a larger improvement on the subtests phonemic segmentation (a test of phonological separation skill) and spoonerisms (a test of phonological manipulation skill) compared to the Placebo group. Although the difference was not statistically significant, it shows the differential impact of the Cellfield intervention on aspects of phonological processing. As the DST-S does not provide a parallel test form, gains due to repeated testing cannot be completely ruled out; however, as the Placebo group did not significantly decrease their overall at-risk index, this seems unlikely. Finally it should be noted that it cannot be determined which aspects of the Cellfield intervention may have caused the observed effects, as the program integrates various exercises to target multiple deficits of dyslexia (Prideaux et al., 2005; Shaywitz et al., 2008).

## The Lack of Gains in Higher-order Skills such as Text Reading Accuracy and Comprehension following the Cellfield Intervention

The absence of gains in text reading accuracy and comprehension as assessed by the Neale immediately following the Cellfield intervention and for the Cellfield group only, suggests that transfer to higher-order literacy skills did not occur. This finding is in contrast to that of Prideaux et al. (2005) who reported significant gains in Neale reading accuracy and comprehension with medium effect sizes following the Cellfield intervention. Prideaux et al. attributed the large gains in reading accuracy and comprehension following the Cellfield intervention to the integrative nature of the Cellfield program, an effect we could not replicate as improvements for the Cellfield group were mainly observed for phonological skills. The differences in sample selection and age range between this study and the Prideaux et al. study may account for these discrepancies. In addition no control/placebo group was implemented in the Prideaux et al. study. Also the sample of the current study was very small and findings cannot be generalised.

Interestingly the students' oral reading proficiency as assessed by the Neale showed a medium Time effect for reading accuracy ( $\eta p^2 = 0.56$ ) and reading comprehension ( $\eta p^2 = 0.60$ ), which only emerged at follow-up, indicating substantial improvements for both groups from pre- to follow-up-test, and post- to follow-up-test. For reading accuracy the overall gains translated into reading ages for the Cellfield group (Reading ages;  $M_{pre} = 7.77$ ,  $M_{follow-up} = 8.90$ ) showed a gain of 1.13 years and for the Placebo group (Reading ages:  $M_{pre} = 8.42$ ,  $M_{follow-up} = 9.57$ ) a gain of 1.15 years. For reading comprehension, the gain for the Cellfield group was 2.25 years (Reading ages:  $M_{pre} = 8.60$ ,  $M_{follow-up} = 10.85$ ) and for the Placebo group 1.57 years (Reading ages:  $M_{pre} = 9.18$ ,  $M_{follow-up} = 10.75$ ). These effects can be considered of practical significance as previous researchers state that gains of three months are considerable (e.g., Le Fevre, Moore, & Wilkinson, 2003). Although the Neale provides two parallel testing forms, the same test form as at pre-test was used at the third testing time, thus not excluding the possibility of a carry-over effect. However the medium effect size and the fact that the time lapse between the pre- and follow-up-testing was at least six weeks for each participant, suggest that gains are not only attributable to repeated testing and that the provided follow-on practice proved beneficial overall.

As outlined in Chapter 8 the follow-on practice was more individually tailored and thus methodologically less stringent than the Cellfield/Placebo programs. Thus, it is not possible to satisfactorily evaluate the elements of the practice that may have produced gains. However, it can be speculated that the repeated reading of graded reading material (DIBELS) and books followed by comprehension questions about what has been read in the follow-on practice would have beneficial impacts to some extent on reading comprehension and accuracy, matching the skills assessed by the Neale. Previous research has also indicated that for older reading-disabled children, combined methods of intervention including explicit phonological exercises but also strategy-based techniques to assist word recognition can result in larger gains (Lovett, Lacerenza, & Borden, 2000b; Swanson & Hoskyn, 1998). The techniques used during the follow-on practice included both elements of strategy-based training (e.g., developing questions about a text being read, answering questions, teaching of explicit rules such as the silent 'e' rule) and to a smaller extent phonological exercises (e.g., phoneme and syllable identification in words).

In addition, the Cellfield group did not show significantly larger gains for reading accuracy and comprehension at follow-up compared to the Placebo group. Thus, the claim that Cellfield results in superior gains due to its integrative approach was not confirmed in our sample. If the Cellfield intervention had led the way for improvements in higher-order skills like text reading accuracy and comprehension, then a cumulative effect on these measures would have been expected and the Cellfield group would have shown larger improvements than the Placebo group in comprehension and accuracy. However, the group means indicate that whereas the Cellfield group had a slightly higher gain for comprehension than the Placebo group, the opposite held true for the accuracy gain. One possible explanation for the emerging gain in comprehension and accuracy at follow-up-test is that the combined and individually tailored follow-on practice may be more effective in boosting higher-level skills such as text reading accuracy and comprehension, especially in

older children, whereas the Cellfield intervention's strength lies within the boost of basic phonological and orthographic-phonological mapping skills. Our finding that the Placebo group did not improve their phonological skills substantially following the follow-on practice supports this interpretation. Moreover, it may indicate that a combination of a basic phonological and visual-phonological training such as Cellfield with higher-level order instructions, which add strategy teaching and semantics to the training may maximise the training impact for older reading-disabled children. This conclusion has also been suggested by Shaywitz et al. (2008) who report that in some older dyslexic children explicit phonics and visual-auditory temporal training may not be sufficient, and additional strategy-based interventions may be needed to achieve a larger improvement in reading skills.

# The Absence of any Gains in Reading Rate and Spelling following the Cellfield and Follow-on Practice Program

The finding that neither the Cellfield intervention nor the follow-on practice resulted in any gains in either reading fluency or spelling is not surprising. As reported consistently in the intervention research literature those domains are the hardest and take the longest to remedy (e.g., Lyon & Moats, 1997; Oakland et al., 1998; Torgesen et al., 2001). With regard to the spelling outcomes, moreover, the Cellfield intervention does not include specific spelling training; thus a generalisation to spelling ability may be too much to expect. However, the evaluation study by Prideaux et al. (2005) did report small but significant gains in spelling skills. The follow-on practice in the current study did integrate spelling practice, but again the generalisation from practiced words to new words in a standardised spelling test is rarely seen (for a review see Wanzek et al., 2006).

In terms of reading fluency, the Cellfield Company reports that it is common for treated children to drop in reading rate immediately after the intervention, as they start to decode words instead of skipping or guessing them (Prideaux et al., 2005). We did not obtain that result. However, inspection of the means indicates a slight non-significant decrease in reading rate at post- compared to pre-test for the Cellfield group only. The follow-on training program, however, did include fluency practice through repeated reading, and failed to improve reading rate, and thus is consistent with a large amount of previous research failing to improve reading fluency especially in older children (for a review see Shaywitz et al., 2008).

In summary, these findings are preliminary, and given the number of participants in the groups, tentative, but it appears that the Cellfield intervention had a stimulating impact on basic phonological skills, whereas the utilised follow-on practice strengthened higherlevel skills such as text reading accuracy and comprehension in the present sample. We suggest that this indicates the more beneficial impact of both intervention approaches (phonological and orthographic-phonological training as in the Cellfield intervention, and strategy-based reading instruction with an emphasis on text comprehension as in the followon practice) in treating reading problems comprehensively. The larger gains in phonological skills for the Cellfield group only, and the overall gains for both groups in text reading accuracy and comprehension, support this conclusion. It should be noted, however, that overall, at the conclusion of the study, both groups were still performing at below average levels on most of the literacy measures, with the word attack measure for the Cellfield group being one exception. In addition, effect sizes were of small to medium magnitude. These findings are consistent with the smaller gains commonly reported in the literature for older reading-disabled children compared to younger children at primary school level (e.g., Lyon, 1995; Shaywitz et al., 2008; Swanson & Hoskyn, 1998).

#### Motivational and Perceived Self-Effectiveness Aspects

The ROPELOC and MRP were administered at pre- and post-test to gain an insight into the participants' motivation to read and their perceived self-effectiveness. Previous research has indicated that a child's reading motivation can be influenced and increased through reading training in general (Wigfield et al., 2004).

With regard to the reading motivation measure, both groups showed a relatively low reading motivation, thus confirming previous research that has indicated relationships between learning difficulties and motivational aspects including motivation to read, externalising problems, and coping styles (Butkowsky & Willows, 1980; Gambrell et al., 1996; Hinshaw, 1992; Lepola, Vaurus, & Maeki, 2000; Poskiparta et al., 2003; Wigfield & Guthrie, 1997). In contrast to our expectation, reading motivation did not change at post-test for the Cellfield group and gains in the phonological decoding measure were observed without an increased motivation to read. Wigfield et al. (2004) reported increased motivation to read following a twelve-week reading instruction program in normal-reading children in Grade 3. The absence of a motivational improvement in the Cellfield group may be due to (1) the assessment of reading motivation at post-test being too early to observe changes in reading motivation and (2) the Cellfield intervention not directly aiming at increasing reading motivation. With regard to the first aspect, it may be speculated that at a later point in time, when more successful reading experience has been accumulated, reading motivation may increase. However this would need to be addressed systematically by assessing students' motivation to read over several years. This latter aspect is indirectly supported by a study by Worthy, Patterson, Salas, Prater, and Turner (2002). These authors reported increased reading motivation for their reading-disabled sample with an individually tailored reading program including specific motivational elements to improve reading motivation (e.g., own book choices, motivating and encouraging tutoring style).

The only significant result obtained was on the external locus of control scale of the ROPELOC, which measures an individual's tendency to attribute success and failure to external causes such as luck. The Placebo group showed a significant increase from pre- to post-test, whereas the Cellfield group showed a slight but non-significant decrease in their external locus of control score. Previous research has indicated that children with learning difficulties attribute success and failure more to external causes such as luck (e.g., Beitchman & Young, 1997) and an increase in external locus of control for the Placebo group may be associated with the Placebo sessions they completed. The Placebo program was a game with various levels of difficulty and one could lose and win these levels. The need for the Placebo group to attribute any failure or success on the game may have been relatively higher than for the Cellfield group, as the Cellfield intervention is designed to produce a higher score or a score just below that from the previous session for positive reinforcement.

#### **ERP** and Behavioural Outcomes

The discussion of the physiological findings will focus on the time changes observed for the Cellfield group. Results for the sentence task will be discussed first, followed by the lexical and phonological tasks.

#### Sentence Processing: Sentence Task

The behavioural findings, as expected, indicated overall significantly longer mean RTs for the incongruent than congruent endings at all testing times. In contrast to our expectation, the Cellfield group did not show a longer RT at post- and follow-up-test compared to pretest. Indeed both groups showed significantly decreased RTs from pre- to post-test irrespective of congruent/incongruent endings suggesting an effect of repeated testing. With regard to time changes for the Cellfield group, the Cellfield group had lower response accuracy and more missing responses at post- compared to pre-test, although these differences were not significant. This finding is in line with our hypothesis that the Cellfield group would start applying phonological decoding skills following the Cellfield intervention to perform the task. The reader is reminded that the response time period was limited for the sentence task. Subsequently, a decoding strategy is more time-consuming than a visual strategy and it can be speculated that this led to more missing responses. A sentence task, which does not limit the time to respond but allows each individual to respond in his or her own time, may have revealed a different result for accuracy/missing responses for the Cellfield group. The Placebo group showed significantly fewer missing responses at followup- compared to pre-test, a finding we currently cannot explain. We can only speculate that it is a repeated testing effect.

The ERP findings were consistent with previous findings on N4 in semantic sentence tasks. Overall incongruent endings produced much larger N4 amplitudes than congruent endings at all three testing times, indicated by significant main effects for Type (for a review see Kutas et al., 2006). This suggests that overall our dyslexic sample was able to detect the semantic incongruity in the sentence task, which is in contrast to our hypothesis ('linguistic specificity hypothesis') and to a finding from a study by Brandeis et al. (1994) who reported N4 amplitude of similar magnitude towards congruent and incongruent endings for German dyslexic compared to control readers (see also Robichon et al., 2002). However, as we did not test a control group we cannot directly compare our findings to those of this study.

With regard to time changes in N4 amplitude, no pre- to post differences reached significance. However, the Cellfield group showed significantly larger N4 amplitudes at left and midline sites compared to right sites at post-test only. Inclusion of follow-up- data, however, revealed overall decreased N4 amplitudes from pre- to follow-up-test. The overall decrease of N4 amplitude from pre- to follow-up-test replicates previous research findings showing that N4 amplitude for incongruent endings decreases with repetition (for a review see Kutas et al., 2006). The three testing times may have caused a repetition priming effect. However, in contrast to normal adults, the dyslexic sample in the present study showed decreased amplitudes to both incongruent and congruent endings.

The larger N4 amplitude for the Cellfield group at post-test at left compared to right sites suggests a more left lateralised processing strategy. In normal adults N4 is often maximal over the right central-parietal site in particular in response to incongruent endings, so we may have expected larger N4 amplitudes for the Cellfield group in the right compared to the left hemisphere. However, a developmental study on a congruent/incongruent sentence task by Holcomb et al. (1992) indicated that younger normal reading adolescents (7 to12 years) had a left focus of N4 compared to an older group (15 to 26 years), who had a right focus. Our sample ranged in age from 12 to 14 years, so it is likely that they fell into the younger group. Thus, the finding of larger N4 amplitudes at left compared to right hemispheric sites, as for the Cellfield group at post-test, suggests an age appropriate distribution of N4. Thus, the larger left N4 amplitude for the Cellfield group may be associated with an increased effort to integrate words into a sentence context, an interpretation proposed by Neville et al. (1993) who found larger N4 amplitudes for language impaired children compared to controls in a congruent/incongruent sentence task (for adult dyslexic sample see also Robichon et al., 2002).

The second component investigated, the LPC, showed a significant decrease in amplitude at right sites from pre- to post- and pre- to follow-up for the Cellfield group only.

Furthermore, the difference in LPC amplitude between left and right sites reached significance at follow-up, showing smaller LPC amplitudes at right compared to left sites. No significant pre-test differences were identified for left or right electrode sites. The Placebo group showed the opposite pattern, with significantly decreasing LPC amplitudes in the left from pre- to follow-up. In addition, the difference between left and right sites did not reach significance at pre-, post- or follow-up, indicating an overall bilateral activation for the Placebo group across testing times.

The decrease of LPC amplitude at right sites for the Cellfield group is in line with our 'normalisation' hypothesis and although the right decrease was not accompanied by a significant increase at left sites, the findings suggest the beginning of a more specialised linguistic processing (e.g., Aylward et al., 2003; Temple et al., 2003). ERP studies that have investigated LPC activity have offered various interpretations. For example, Gunter et al. (1997) stated that the P6 or LPC "elicited in sentence material may reflect a more general language-related reanalysis process in which the outcome of both early syntactic and semantic analyses are jointly re-evaluated" (p. 673). In line with this interpretation of LPC activity, the decreased LPC activity at right sites for the Cellfield group may reflect a transition phase before an efficient shift to left hemisphere processing can be successfully implemented, which will then facilitate the integration of syntactic and semantic analyses. It can be suggested that at a later point in time more left hemisphere processing would be observed. However, further studies are needed to support this interpretation. Ackerman et al. (1994) argued that LPC amplitude reflects further processing and Hillyard, Krausz, and Picton (1974) earlier proposed that LPC activity in general reflects the increased effort involved in processing stimuli that are difficult to discriminate. As a consequence, dyslexic readers may be less efficient and less automatised when engaging in these elaborative discrimination processes, in particular in a sentence task like the one used in our study.

Studies investigating LPC in a sentence task in dyslexia are scarce. One such study that has been conducted is by Robichon et al. (2002), who compared dyslexic and control readers in a congruent/incongruent sentence task. In line with our result, both congruent and incongruent endings elicited a P600 (LPC) following the N4. Moreover, they found larger P600 amplitudes following congruent endings for the dyslexic readers than controls. The authors suggested that the enlarged P600 reflects a parsing problem in dyslexics in that they have difficulty in completing the parsing process in a given sentence. No spatial differences were evident between the controls and dyslexic readers, thus making further comparisons between this study and our study difficult.

Taken together, previous research findings suggest that the LPC component is associated with elaborative and complex linguistic processing and that dyslexic readers differ in LPC amplitude from controls. Our study finding demonstrates that LPC amplitude can be altered following an intervention. A more lateralised activation pattern (larger engagement of the left hemisphere and decreased engagement of the right hemisphere) can possibly lead to activation of specific resources required for complex linguistic processing such as sentence comprehension

### Single-Word Processing: Lexical and Phonological Tasks

The behavioural data overall confirmed the importance of phonological processing difficulties in dyslexia (Snow et al., 1998; Snowling, 2000) as response accuracy for the phonological task was significantly lower and reaction times slower than for the lexical task for both groups at all three testing times. This finding is in line with a study finding by Breznitz (2003) who reported lower accuracy and longer reaction times for the dyslexic sample tested on orthographic and phonological visual tasks, with the phonological task showing the most pronounced differences between dyslexic and control readers. Breznitz suggested that successful performance on the phonological task depends on accurate phonological representations to sound out the pseudo homophones, and deficient phonological representations in dyslexic readers will decrease task performance.

The stimuli types were also discriminated by RT and accuracy data, showing overall lower accuracy and longer reaction times for 'no' responses than 'yes' responses. This finding is expected as the 'no' responses in the tasks were the more difficult stimuli (pseudo homophones and nonwords) and consistent with the general finding in controls that real words ('yes' stimuli in the lexical task in the current study) are responded to faster than nonwords (e.g., Miller-Shaul & Breznitz, 2004). This finding suggests some degree of linguistic familiarity in the dyslexic sample. Given that the present sample was at high school level, this was as expected.

With regard to time changes for the two groups, although we expected the Cellfield group to have longer RTs at post- and follow-up- than pre-test the findings suggest otherwise: Both groups showed significantly decreased RT from pre- to post-test for the phonological task and overall decreased RT for both tasks when follow-up-data was included. As we have argued for the sentence task, the RT decrease could reflect a repeated testing effect and increased familiarity with the tasks. As expected task performance did not change for the Cellfield group indicating the application of a decoding strategy. As suggested by Breznitz (2003) inefficient phonological representations can decrease task performance for dyslexic readers, thus we can speculate that the newly acquired phonological decoding skills are not yet successful enough to improve task performance.

The ERP results for the single-word tasks indicated different patterns for the P2 and N4 components for the two groups. Although the LPC component indicated some group and time differences most of the findings were only suggestive, non-significant effects and will thus not be discussed here. The finding for the P2 for the lexical and phonological tasks was significantly decreased P2 amplitude for both groups from pre- to post-test for the 'yes' responses across electrode sites and at selective sites for the 'no' responses. However, inclusion of follow-up-data revealed that only the Cellfield group showed significantly decreased P2 amplitude across electrodes sites from pre- to follow-up-test. This effect was found irrespective of task and stimuli types, suggesting an overall more automatised initial processing of the linguistic stimuli for the Cellfield group, which only emerged after the three-week follow-on practice. P2 amplitude has been previously associated with item decoding and retrieval in dyslexic and control samples. For instance, in a study by Stelmack et al. (1988), dyslexic readers showed larger P2 amplitudes than controls in a visual memory recognition task. However, two aspects are important to note: (1) In the current study the pre-and post-test results showed P2 amplitude decreases for both the Cellfield and Placebo groups and (2) overall the Cellfield group had significantly larger P2 amplitudes than the Placebo group.

The major finding for N4 showed significant differences for the Cellfield and Placebo group in N4 amplitudes. The Placebo group showed a significant increase in N4 amplitude at the right-central parietal site from pre- to post-test, whereas the Cellfield group did not show any significant pre- to post-test differences, but had a significantly larger N4 amplitude at the left central-parietal site than at the right central-parietal site at post-test only. When follow-up-data was included, only the Cellfield group showed a significant time difference: N4 amplitude increased significantly from pre- to follow-up-test at the left central-parietal site selectively. Again, the Placebo group did not show increased N4 amplitudes at this particular site. While the possibility of a type 1 error always exists, it can be speculated that the Cellfield group started engaging left language functions following the intervention. This finding is consistent with previous imaging research that has investigated neural changes following interventions for dyslexia and found increased left activity for dyslexic readers post-intervention (e.g., Bakker, & Vinke, 1985; Bakker et al., 1990; Simos et al., 2002; Temple et al., 2003). In contrast, the Placebo group in the present study showed larger N4 amplitudes at right hemispheric sites, suggesting compensatory processes (e.g., Shavwitz et al., 2002, 2003). The findings further indicated that at pre-test, as expected, neither group showed significant differences between left and right electrode sites, thus confirming results of imaging and ERP studies on dyslexia and controls (e.g., Aylward et al., 2003).

Pritchard et al. (1991) provided a detailed classification scheme of ERP negativities including N2a, b and c, and N4a, b and c. The stimuli used in the present study most likely represent N4c activity as they represent an abstract classification task ('yes' or 'no' it does/does not spell/sound like a real word). N4 activity, according to Pritchard et al. is generally thought to reflect intentional classification of task relevant stimuli. Thus the increased N4 amplitude for the Cellfield group at post-test may indicate increased efforts to discriminate the linguistic stimuli in general.

With regard to the lateralisation effect, it can be speculated that the increased N4 amplitude for the Cellfield group at the left central-parietal site reflects increased activation strength in the left hemisphere associated with language processing in normal adults.

Similarly Miles and Stelmack (1994) have associated the observed left-larger-than-right asymmetry of N4 in controls with hemispheric specialisation for skilled reading, and its lack in dyslexic readers with deficiencies in hemispheric specialisation during a visual memory task. The authors also pointed out that left hemisphere processing is supposed to result from automatic and efficient language processing as a result of development and increasing exposure to printed material. Smaller amplitudes in the left hemisphere for dyslexics may indicate deficient left language systems (see also Licht et al., 1992; Preston & Guthrie, 1974) and this is consistent with the findings from imaging studies (for a review Shaywitz et al., 2008). In an earlier study Stelmack et al. (1988) also found smaller and less lateralised N4 amplitudes for dyslexics compared to controls and offered the explanation that dyslexics are less engaged in semantic evaluation and memory search, whereas controls have a larger and more efficiently organised lexicon to access.

A more specific interpretation of the lack of left lateralisation in dyslexic readers was proposed by Penolazzi et al. (2006), who compared dyslexic readers and controls on a variety of phonological, semantic and orthographic tasks. These authors also found a lack of left activation, as indicated by smaller N4 amplitudes for dyslexics compared to controls in response to the phonological task. They suggested that this finding is an index of deficient phonological processing, reflecting difficulties with phoneme-grapheme conversions. This interpretation leads to the speculation that increased left-lateralised N4 amplitudes for the Cellfield group indicates the beginning of more efficient phonological representations and the actual application of phonological decoding strategies when encountering word stimuli.

To the best of our knowledge, there is no study which has investigated N4 both before and after an intervention program. Thus, although all reported studies indirectly support our conclusions, caution is called for in these interpretations. Clearly our conclusions would have been strengthened if a normal reading control group had been investigated. However, this was not possible in the given time-frame for the project.

Another aspect with regard to the proposed 'increased linguistic specificity hypothesis', will be highlighted here: The lack of lateralisation at pre-test for both groups and the larger N4 amplitude at the left central-parietal site for the Cellfield group at postand follow-up was observed irrespective of tasks and stimuli types. Studies that have investigated visual word recognition in normal adults have commonly reported that N4 amplitude is larger for pseudowords and real words compared to nonwords, which show no or little N4 activity (Kutas et al., 2006). In line with our hypothesis, N4 amplitude did not differ between these stimuli types at pre-test for either group. A similar result to ours was obtained in a study by Lovrich et al. (1996) who investigated auditory classification of phonological and semantic words in dyslexic readers and controls, and found a more prominent and broader distribution of N4 for dyslexic readers than controls for a phonological classification task regardless of stimuli type (rhyming and non-rhyming stimuli). The authors suggested that dyslexic readers might find it more demanding to classify words according to their phonological features than semantic features. In addition, the ability to detect phonological relevant cues may be less automatic in dyslexic readers. This suggests that our dyslexic sample was less able to discriminate among the linguistic features of the word stimuli.

In contrast to our hypothesis, the Cellfield group did not show larger N4 amplitudes in response to real words and pseudo homophones than nonwords at post-or follow-up- test, but rather overall increased left N4 amplitude regardless of stimuli types and tasks. It can be suggested that the increased responsiveness is not yet specific enough to discriminate among subtle linguistic distinctions of the word stimuli and that the overall increased N4 amplitudes reflect the effort to make sense of the real words, pseudo homophones, and nonwords by applying phonological strategies.

#### Integration of the Literacy, ERP and Behavioural Outcomes

The ERP findings of decreased LPC amplitudes at right sites for the sentence task, and increased N4 amplitudes at left sites for the lexical and phonological tasks for the Cellfield group at post- and follow-up-test, suggest plasticity of neural functions (Shaywitz et al., 2004). Whereas both components have been associated with language processing in previous research (Gunter et al., 1997; Kutas et al., 2006; Ziegler et al., 1997), the question arises as to why specific time changes for the Cellfield group were evident in LPC for the sentence task, but in N4 for the lexical and phonological tasks? A study by Lovrich et al. (1996) is of direct relevance for understanding these findings. They found delayed and larger LPC amplitudes for their dyslexic sample during both rhyme and semantic classification tasks and variations in the N4 time window between dyslexic and control readers only for the phonological rhyme task. Lovrich et al. inferred that LPC activity might be more related to complex processing that involves some kind of semantic processing. This supports our finding showing that LPC in the semantic sentence task was more sensitive to change for the Cellfield group, whereas the earlier component of N4 revealed more changes for the Cellfield group for the word-level tasks, which require only a minimum of semantic processing. However, it should be noted that the study by Lovrich et al. involved tasks in the auditory modality, thus our findings for the visual modality are not directly comparable.

Similarly Helenius et al. (1999a) have reported different ERP and MEG patterns for single-word versus sentence reading tasks for their dyslexic sample, whereas ERP and MEG patterns of the normal controls did not differ between tasks. Subsequently it can be proposed that linguistic processing on a single-word level involves different skills from processing on a sentence level (Aylward et al., 2003; Helenius et al., 1999a). The word-level tasks require mainly word recognition and involve lexical and phonological skills whereas the sentence task involves word recognition, memory skills, and reading comprehension. These differences in task demands could be associated with the observed neural changes in N4 and LPC.

We noted earlier that the N4 amplitude for word-level tasks has been held to reflect phonological processing (Penolazzi et al., 2006) and the LPC amplitude to reflect continuous processing and re-evaluation in a sentence context (Gunter et al., 1997). Thus, the increased N4 amplitude at the left central-parietal site for the Cellfield group following intervention may relate to the Cellfield group's increased phonological skills as assessed by the word attack subtest from the WRMT-R at post-and follow-up-test. The increased N4 amplitude at the left central parietal site for the lexical and phonological tasks may have facilitated the significant improvement of phonological skills observed for the Cellfield group. It should be noted, however, that the 'chicken-and-egg' problem cannot be solved
here (Mathes & Denton, 2002) and that the improved phonological skills may have caused the brain activity to change or vice versa. Nevertheless, imaging studies that have investigated neural changes following interventions for dyslexia have demonstrated positive correlations between strengthened left brain activity and reading skill in dyslexic readers (e.g., Temple et al., 2003).

The absence of improved task performance on the experimental lexical and phonological tasks for the Cellfield group at post-or follow-up-test was expected. It could result from the fact that the word attack subtest from the WRMT-R allows more time to respond to the test items than the experimental tasks, so that the accuracy data did not differentiate the two groups at post- or follow-up-test. Similarly, in an imaging study by Shaywitz et al. (2004), RT and accuracy data did not differentiate a treated dyslexic group and an untreated dyslexic group, whereas imaging data indicated considerable differences between the groups. Other ERP research on dyslexia has also sometimes failed to show strong relations between neural and behavioural findings, in that differences between dyslexic and control readers were evident in ERPs but not in behavioural data (e.g., Landi & Perfetti, 2007; Shaywitz et al., 2004). The dissociation of ERPs and RT has been claimed to reflect the fact that ERPs and RT may measure somewhat different aspects of processing. ERPs are more specifically held to reflect cognitive process only, whereas RT is a combination of cognitive processes related to decision-making and the actual response (Landi & Perfetti, 2007). Thus ERP data may be more sensitive to changes in cognitive processes than the RT data. Also, our study did not reveal any significant latency differences for the two groups for the investigated ERP components and latencies of ERPs are usually associated with RT.

With regard to the sentence task, we have speculated that the larger N4 amplitude at left and mid sites compared to right sites for the Cellfield group reflects the beginning of reorganisation of brain activity. In previous research, N4 has also been associated with phonological processing. Thus, the non-significant decrease in task performance for the Cellfield group at post- and follow-up- compared to pre-test could be associated with the use of a phonological decoding strategy during the sentence task. In addition, the significant decrease in LPC amplitude at right hemispheric sites could also be associated with the decreased task performance of the Cellfield group at post- and follow-up-test since right processing is generally associated with compensatory processes in dyslexics. A decreased right focused activity may worsen task performance, while the shift to more left language based processing is in development (for a review see Shaywitz et al., 2008).

The decreased LPC amplitude at right sites for the Cellfield group at post- and follow-up-test could also relate to the absence of gains in Neale accuracy and comprehension immediately following the Cellfield intervention. As argued earlier, the LPC amplitude decrease at right hemisphere sites reflects a transition phase from bilateral/right focussed processing to more left language-specialised processing (Bakker et al., 1990), with the latter facilitating task performance on the sentence task and reading comprehension and accuracy as assessed by the Neale. The absence of any significant improvements in reading accuracy and comprehension as assessed by the Neale immediately following the Cellfield intervention for the Cellfield group would support this interpretation in the following ways: Firstly, full left lateralisation is not yet successfully accomplished. Thus, improvements in higher-level literacy skills may not be expected. Secondly, a decrease of bilateral or right focussed processing, which is usually associated with compensatory mechanisms in dyslexia, may additionally impact on the absence of gains in reading comprehension and accuracy. These interpretations are indirectly supported by an imaging study by Shaywitz et al. (2004), who reported decreased right activity and increased left activity in their dyslexic sample following a basically phonological intervention. Shaywitz et al. stated that compensatory processes in the right hemisphere are no longer needed as left activity functions take over. For our sample this suggests that the testing conducted immediately after the two-week Cellfield intervention and three weeks later after the follow-on practice may have been too early to establish a more left-focussed processing style.

However, as both groups improved their reading comprehension and accuracy significantly at follow-up, which we primarily attributed to the follow-on practice, this interpretation can be challenged. Moreover, the Cellfield group did not reveal left specific increases in LPC amplitude at follow-up- compared to pre- and post-test. This questions the association between LPC amplitude and reading comprehension and accuracy. We do not have an explanation for this finding. At this stage the improvements in reading accuracy and comprehension at follow-up-test as indicated by the Neale were not consistently reflected in the neural correlates. Further studies are needed, which include several EEG recordings while an intervention is in progress to gain a deeper understanding of indicated neural changes and the point in time these start to emerge.

# Limitations

Overall, it should be noted that several limitations might have impacted on the results. The study sample was very small, calling for caution about generalisation of the study's findings to other dyslexic samples. It cannot be determined which aspects of the Cellfield intervention or the follow-on practice may have produced the observed outcomes on literacy and neural measures. The integrative nature of the Cellfield program does not allow the isolation of aspects of the intervention which are more or less beneficial in overcoming difficulties in dyslexia (Prideaux et al., 2005), and the follow-on practice was likewise individually tailored and involved various literacy exercises.

The study was not conducted as a double-blind trial. Due to limited resources it was not possible to have two researchers for the conduction of the trial. The researcher conducted the initial screening, all pre-, post- and follow-up-assessments (literacy and ERP experiments) and the intervention and follow-on practice with the participants and thus researcher effects cannot be ruled out entirely (e.g., observer-expectancy effect, Rosenthal effect). With regard to the ERP part of the study it should be noted that we did not test a control, non-dyslexic sample due to time constraints, and results would have been clearly strengthened by including a non-dyslexic control group. All comparisons of results with findings from ERP studies using control and dyslexic readers are therefore indirect. The ERP data did indicate some specific time changes for the Cellfield group only, but it is important to consider that in general the differences in ERPs between the groups did not reach significance, and observed changes were generally within the groups only. In addition, whereas ideally the Placebo group's activation pattern should not have changed at post-test, the findings indicated some changes for the Placebo group, a finding consistent with previous imaging studies which have sometimes indicated changes in activation pattern during the second scanning for a normal-reading control group (e.g. Aylward et al., 2003; for a review see Noble & McCandliss, 2005).

## Future Research

The last section of this chapter will be dedicated to recommendations for future research indicated by the current study. We will highlight the contribution of neuroscience to intervention research and stress the importance of integrating various individual differences of the dyslexic population to maximise intervention outcomes.

## The Contribution of Neuroscience to the Field of Dyslexia

The boom of imaging studies in dyslexia over the past decade has contributed immensely to our understanding of dyslexia and has already shown impressively that the brain can be reorganised following interventions. Intervention studies, which use neural indicators as outcome measures following an intervention for dyslexia, are almost exclusively imaging studies and have indicated a more left lateralised activation pattern of the brain. Our preliminary ERP findings give some support to these imaging findings. However, future studies are needed to test this hypothesis and we recommend the integration of imaging and EEG data to maximise spatial and temporal resolution (Gruenling et al., 2004). Our findings are promising in suggesting that ERPs may be a useful and cost-effective method of evaluating intervention outcomes and we hope this research will stimulate more intervention research integrating ERPs as outcome measures in dyslexia. In addition, Goswami (2004) highlights the potential of ERP indicators for the assessment of learning difficulties by stating that "cognitive developmental neuroscience has established a number of neural 'markers' that can be used to assess development, for example of the language system (e.g. N400)...and that certain patterns may turn out to be indicative of certain developmental disorders" (p. 12).

Taken together, ERP and imaging markers may become useful indicators for the assessment of dyslexia and other learning difficulties and for the measurement of change

following interventions. Early assessment including literacy and neural indicators would allow at-risk readers to be identified and facilitate early intervention or prevention of reading difficulties (Shaywitz & Shaywitz, 2005). The crucial and creative step now is to build a bridge between these neurophysiological findings and educational practice. The more the research knowledge is shared with educational bodies such as educational politicians, schools, teachers and parents, the larger the impact on educational practice allowing the most recent and state-of-the-art assessment, prevention, and intervention for the individual affected by dyslexia. As Goswami (2004) makes clear, "educational and cognitive psychologists need to take the initiative, and think 'outside the box' about how current neuroscience techniques can help to answer outstanding educational questions" (p.12).

### Which Intervention and for Whom?

Intervention research in dyslexia is commonly presented with a dilemma: Research points to a multidimensional deficit in dyslexia calling for multidimensional approaches to treat various difficulties associated with dyslexia (Alexander & Slinger-Constant, 2004; Pennington, 2006; Wolf et al., 2000). However, multidimensional intervention approaches often do not allow the isolation of specific components of the intervention, so it cannot be determined which aspect of the training is beneficial for which aspects of reading and spelling. When isolated training programs are evaluated, we are often presented with the problem that only a few aspects of the reading and spelling problems improve, leaving the dyslexic reader still struggling. This dilemma makes it difficult to recommend one intervention program over another.

As mentioned earlier, the intervention evaluated in the current study, the Cellfield intervention, treats multiple deficits associated with dyslexia and therefore integrates various basic function and literacy exercises into the program. In a similar manner the follow-on practice involved integrative and individually tailored literacy exercises. However, we did not find improvements in higher-order literacy skills following the Cellfield intervention and gains were mainly observed for the phonological domain. The additional value of the Cellfield intervention and its superiority in improving phonological skills compared to other phonological programs needs to be subjected to further critical investigation. In contrast, the follow-on practice improved higher-order literacy skills significantly, but did not significantly strengthen phonological skills for the Placebo group or produce a further increase in phonological skills for the Cellfield group. Spelling and reading rate were not improved by the combination of the programs, highlighting the need for more specific and longer intervention techniques directly targeting spelling and reading fluency. It should also be noted that at the conclusion of this study both groups were still performing below average on most of the literacy tests, indicating the need for overall more intense and ongoing support. In conclusion, we hope that future studies will investigate the Cellfield intervention with a larger dyslexic sample to allow comparisons with our preliminary findings.

It has become increasingly clear that the dyslexic population is a diverse population. Dyslexic profiles vary in severity of overt difficulties as well as in which aspects of literacy are affected most (for a review see Snowling, 2000). We therefore cannot expect to find a "cure" for dyslexia that would be beneficial for all dyslexic children and adults. As Shaywitz et al. (2008) point out, "several types of intervention programs are effective. Evidence is not yet available that would allow the selection of one specific program over others or to support the choice of an individual program that would be specifically beneficial to particular groups of dyslexic readers" (p. 463). This clearly highlights the need for future intervention research to study individual disability profiles and determine predictors of response to interventions. As outlined in Chapter 6, various cognitive and reading-related skills have been found to be predictors of successful reading development (for a review see Bowey, 2005) and of responses to interventions (for reviews see Snowling, 2000; Torgesen, 2000).

Motivational and emotional factors that are more prevalent among children with learning difficulties, such as low self-esteem, low motivation for learning and reading, and externalising and internalising problems (e.g., Beitchman & Young, 1997; Casey et al., 1992), have often been neglected in intervention research as potential influencing variables on intervention outcomes. Thus, the present study was designed to gain insight into some of these motivational aspects (reading motivation and locus of control) of the dyslexic sample investigated. As expected on the basis of previous research, reading motivation was relatively low at pre-test (Morgan & Fuchs, 2007; Lepola et al., 2000; Poskiparta et al., 2003), which may partly explain the rather small literacy gains at post- and follow-up-test. However, we argued that the testing of reading motivation, which was conducted immediately after completion of the intervention (two weeks), might have occurred too early to produce large increases in reading motivation. A study by Worthy et al. (2002) highlights the possibility of improving reading motivation in dyslexic children. These authors reported significant improvements after one to two semesters of literacy tutoring for reading skills and reading motivation in a dyslexic sample. The program specifically involved motivational techniques, such as allowing the students to choose their own books, asking students to give feedback about the tutoring, and having tutors who were strongly and positively committed to the tutoring of the students. Morgan and Fuchs (2007) emphasise this finding by asking, "are poor readers doubly disadvantaged in that they soon begin to lag behind their peers in both skill and will? If so, then their poor reading skills and low reading motivation may begin to influence each other" (p. 166). It would thus be fruitful for future intervention studies to integrate these motivational aspects into intervention programs and assess motivational aspects before and after interventions to shed more light on the possible contribution of motivational factors in maximising training outcomes.

In summary, findings on individual differences in dyslexia are of great value for strengthening intervention research in that these variables can be systematically assessed before and after interventions. Subsequent intervention efforts will then provide a more comprehensive picture of the individual disability profile and can assist more effectively in overcoming the learning difficulty. We would like to conclude this chapter with one question, that we would recommend future intervention research take into consideration: What conditions of funding, procedure, intervention techniques and support are necessary to ensure that all children receive the kinds of reading instruction and interventions they require to become fluent, proficient readers?

#### References

- Achenbach, T. (2001). The child behavioural check list. Achenbach system of empirically based assessment aseba. Victoria: ACER.
- Ackerman, P., Dykman, R., & Oglesby, D. (1994). Visual event-related potentials of dyslexic children to rhyming and nonrhyming stimuli. *Journal of Clinical and Experimental Neuropsychology 16*, 138-154.
- Adlergrinberg, D., & Stark, L. (1978). Eye-movements, scanpaths, and dyslexia. *American* Journal of Optometry and Physiological Optics, 55, 557-570.
- Aghababian, V., & Nazir, T. A. (2000). Developing normal reading skills: Aspects of the visual processes underlying word recognition. *Journal of Experimental Child Psychology*, 76, 123-150.
- Agnew, J., Dorn, C., & Eden, G. (2004). Effect of intensive training on auditory processing and reading skills. *Brain and Language*, 88.
- Al Otaiba, S., & Fuchs, D. (2006). Who are the young children for whom best practices in reading are ineffective? An experimental and longitudinal study. *Journal of Learning Disabilities*, 39, 414-431.
- Alexander, A. W., Andersen, H. G., Heilman, P. C., Voeller, K. K. S., & Torgesen, J. K. (1991). Phonological awareness training and remediation of analytic decoding deficits in a group of severe dyslexics. *Annals of Dyslexia*, 41, 193-206.
- Alexander, A. W., & Slinger-Constant, A. M. (2004). Current status of treatments for dyslexia: Critical review. *Journal of Child Neurology*, 19, 744-758.
- Alexander, J. R. M., & Martin, F. (2000). Norming tests of basic reading skills. Australian Journal of Psychology, 52, 139-148.
- Allen, M., Badecker, W., & Osterhout, L. (2003). Morphological analysis in sentence processing: An ERP study. Language and Cognitive Processes, 18, 405-430.
- Anderson, J. E., & Holcomb, P. J. (1995). Auditory and visual semantic priming using different stimulus onset asynchronies: An event-related brain potential study. *Psychophysiology*, 32, 177-190.
- Au, A., & Lovegrove, B. (2006). Examining the validity of IQ-discrepancy definition in dyslexia: What do temporal processes tell us? *Australian Journal of Psychology*, 58, 61-61.
- Aylward, E. H., Richards, T. L., Berninger, V. W., Nagy, W. E., Field, K. M., Grimme, A. C., et al. (2003). Instructional treatment associated with changes in brain activation in children with dyslexia. *Neurology*, *61*, 212-219.
- Baddeley, A. D., Ellis, N. C., Miles, T. R., & Lewis, V. J. (1982). Developmental and acquired dyslexia: A comparison. *Cognition*, 11, 185-199.

- Badian, N. A. (1997). Dyslexia and the double deficit hypothesis. Annals of Dyslexia, 47, 69-87.
- Baker, L., Fernandez-Fein, S., Scher, D., & Williams, H. (1998). Home experiences related to the development of word recognition. In J. L. Metsala & L. C. Ehri (Eds.), *Word recognition in beginning literacy* (pp. 263-287). Mahwah, NJ: Erlbaum.
- Bakker, D. J. (2006). Treatment of developmental dyslexia: A review. *Pediatric Rehabilitation*, *9*, 3-13.
- Bakker, D. J., Bouma, A., & Gardien, C. J. (1990). Hemisphere-specific treatment of dyslexia subtypes: A field experiment. *Journal of Learning Disabilities*, 23, 433-438.
- Bakker, D. J., Moerland, R., & Goekoop-Hoefkens, M. (1981). Effects of hemispherespecific stimulation on the reading performance of dyslexic boys: A pilot study. *Journal* of Clinical Neuropsychology, 3, 155-159.
- Bakker, D. J., & Vinke, J. (1985). Effects of hemisphere-specific stimulation on brain activity and reading in dyslexics. *Journal of Clinical and Experimental Neuropsychology*, 7, 505-525.
- Barnea, A., & Breznitz, Z. (1998). Phonological and orthographic processing of hebrew words: Electrophysiological aspects. *Journal of Genetic Psychology*, 159, 492-504.
- Barnea, A., Lamm, O., Epstein, R., & Pratt, H. (1994). Brain potentials from dyslexic children recorded during short-term memory tasks. *International Journal of Neuroscience*, 74, 227-237.
- Beck, I. L. (2005). Making sense of phonics. The hows and whys: Guilford Press.
- Beitchman, J. H., & Young, A. R. (1997). Learning disorders with a special emphasis on reading disorders: A review of the past 10 years. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 1020-1032.
- Bentin, S., Mouchetant-Rostaing, Y., Giard, M. H., Echallier, J. F., & Pernier, J. (1999). ERP manifestations of processing printed words at different psycholinguistic levels: Time course and scalp distribution. *Journal of Cognitive Neuroscience*, 11, 235-260.
- Besner, D., Twilley, L., McCann, R. S., & Seergobin, K. (1990). On the association between connectionism and data are a few words necessary. *Psychological Review*, *97*, 432-446.
- Besson, M., Kutas, M., & Van Petten, C. (1992). An event-related potential (ERP) analysis of semantic congruity and repetition effects in sentences. *Journal of Cognitive Neuroscience*, 4, 132-149.
- Biederman, J., Faraone, S., Milberger, S., Curtis, S., Chen, L., Marrs, A., et al. (1996).
  Predictors of persistence and remission of adhd into adolescence: Results from a fouryear prospective follow-up study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 343-351.

- Bischof, J., Gratzka, V., Strehlow, U., Haffner, J., Parzer, P., & Resch, F. (2002). Reliability, trainability, and stability of auditory discrimination performance in two computer supported assessment and training tasks. *Zeitschrift Fur Kinder-Und Jugendpsychiatrie* Und Psychotherapie, 30, 261-270.
- Black, J. L., Collins, D. W. K., Deroach, J. N., & Zubrick, S. (1984). A detailed study of sequential saccadic eye-movements for normal-reading and poor-reading children. *Perceptual and Motor Skills*, 59, 423-434.

. . ...

l

ì

ł

- Boddy, J. (1986). Event-related potentials in chronometric analysis of primed word recognition with different stimulus onset asynchronies. *Psychophysiology*, 23, 232-245.
- Bowers, P. G., & Swanson, L. B. (1991). Naming speed deficits in reading-disability: Multiple measures of a singular process. *Journal of Experimental Child Psychology*, 51, 195-219.
- Bowey, J. A. (2005). Predicting individual differences in learning to read. In M. J. Snowling & C. Hulme (Eds.), *The science of reading: A handbook* (pp. 155-173). UK, Oxford: Blackwell Publishing.
- Bradley, L., & Bryant, P. E. (1978). Difficulties in auditory organization as a possible cause of reading backwardness. *Nature*, 271, 746-747.
- Brandeis, D., Vitacco, D., & Steinhausen, H. (1994). Mapping brain electric micro-states in dyslexic children during reading. *Acta Paedopsychiatry*, *56*, 239-247.
- Brannan, J. R., Solan, H. A., Ficarra, A. P., & Ong, E. (1998). Effect of luminance on visual evoked potential amplitudes in normal and disabled readers. *Optometry and Vision Science*, 75, 279-283.
- Breitmeyer, B. G., & Ganz, L. (1976). Implications of sustained and transient channels for theories of visual-pattern masking, saccadic suppression, and information-processing. *Psychological Review*, 83, 1-36.
- Breznitz, Z. (2003). Speed of phonological and orthographic processing as factors in dyslexia: Electrophysiological evidence. *Genetic Social and General Psychology Monographs*, 129, 183-206.
- Breznitz, Z., & Meyler, A. (2003). Speed of lower-level auditory and visual processing as a basic factor in dyslexia: Electrophysiological evidence. *Brain and Language*, 85, 166-184.
- Breznitz, Z., & Misra, M. (2003). Speed of processing of the visual-orthographic and auditory-phonological systems in adult dyslexics: The contribution of "Asynchrony" to word recognition deficits. *Brain and Language*, 85, 486-502.
- Bruck, M. (1992). Persistence of dyslexics phonological awareness deficits. *Developmental Psychology*, 28, 874-886.

- Brunswick, N., McCrory, E., Price, C. J., Frith, C. D., & Frith, U. (1999). Explicit and implicit processing of words and pseudowords by adult developmental dyslexics: A search for wernicke's wortschatz? *Brain*, 122, 1901-1917.
- Brunswick, N., & Rippon, G. (1994). Auditory event-related potentials, dichotic-listening performance and handedness as indexes of lateralization in dyslexic and normal readers. *International Journal of Psychophysiology*, 18, 265-275.
- Burr, D. C., Morrone, M. C., & Ross, J. (1994). Selective suppression of the magnocellular visual pathway during saccadic eye-movements. *Nature*, 371, 511-513.
- Butkowsky, I. S., & Willows, D. M. (1980). Cognitive-motivational characteristics of children varying in reading-ability: Evidence for learned helplessness in poor readers. *Journal of Educational Psychology*, 72, 408-422.
- Cao, F., Bitan, T., Chou, T. L., Burman, D. D., & Booth, J. R. (2006). Deficient orthographic and phonological representations in children with dyslexia revealed by brain activation patterns. *Journal of Child Psychology and Psychiatry*, 47, 1041-1050.
- Caplygin, D. (2001). *Cellfield intervention for dyslexia*. Australia: Cellfield Company Limited.
- Casey, R., Levy, S. E., Brown, K., & Brooks-Gunn, J. (1992). Impaired emotional health in children with mild reading disability. *Journal of Developmental and Behavioral Pediatrics*, 13, 256-260.
- Castles, A., & Coltheart, M. (1993). Varieties of developmental dyslexia. *Cognition, 47*, 149-180.
- Chase, C., & Jenner, A. R. (1993). Magnocellular visual deficits affect temporal processing of dyslexics. *Annals of the New York Academy of Sciences*, 682, 326-329.
- Chiappe, P., Stringer, R., Siegel, L. S., & Stanovich, K. E. (2002). Why the timing deficit hypothesis does not explain reading disability in adults. *Reading and Writing*, *15*, 73-107.
- Chwilla, D. J., Brown, C. M., & Hagoort, P. (1995). The N400 as a function of the level of processing. *Psychophysiology*, *32*, 274-285.
- Clemente, C. D. (1985). Grays's anatomy. Philadelphia, USA: Lea & Febiger.
- Clifton, C., Speer, S., & Abney, S. P. (1991). Parsing arguments: Phrase structure and argument structure as determinants of initial parsing decisions. *Journal of Memory and Language*, *30*, 251-271.
- Clisby, C., Fowler, M. S., Hebb, G. S., Walters, J., Southcott, P., & Stein, J. F. (2000).Outcome of treatment of visual problems in children with reading difficulties.*Professional Association of Teachers in Special Education Bulletin*, 9-14.
- Cognitive Concepts. (1998). *Earobics auditory development and phonics reading program*. Evanston IL: Cognitive Concepts.

- Cohen, J. (1988). Statistical power analysis for the behavioral sciences. Hillsdale, NJ: Erlbaum
- Cohen, L., Dehaene, S., Naccache, L., Lehericy, S., Dehaene-Lambertz, G., Henaff, M. A., et al. (2000). The visual word form area: Spatial and temporal characterization of an initial stage of reading in normal subjects and posterior split-brain patients. *Brain*, 123, 291-307.
- Cohen-Mimran, R., & Sapir, S. (2007). Auditory temporal processing deficits in children with reading disabilities. *Dyslexia*, 13, 175-192.
- Coles, M. G. H., Smid, H. G. O. M., Scheffers, M. K., & Otten, L. J. (1995). Mental chronometry and the study of human information processing. In M.S. Rugg & M. G. H. Coles (Eds.), *Electrophysiology of mind. Event-related brain potentials and cognition* (pp. 86-131). Oxford: University Press.
- Coltheart, M. (1978). Toward a psychology of reading. *Quarterly Journal of Experimental Psychology*, *30*, 389-391.
- Coltheart, M. (1981). MRC psycholinguistic database. Retrieved 01/02, 2008, from http://www.psy.uwa.edu.au/mrcdatabase/uwa\_mrc.htm
- Coltheart, M. (2005). Modeling reading: The dual-route approach. In M. J. Snowling & C. Hulme (Eds.), *The science of reading: A handbook* (pp. 6-24). UK, Oxford: Blackwell Publishing.
- Coltheart, M., Curtis, B., Atkins, P., & Haller, M. (1993). Models of reading aloud: Dualroute and parallel-distributed-processing approaches. *Psychological Review*, 100, 589-608.
- Coltheart, M., & Leahy, J. (1996). Assessment of lexical and nonlexical reading abilities in children: Some normative data. *Australian Journal of Psychology*, 48, 136-140.
- Coltheart, M., Patterson, K., & Marshall, J. C. (1980). *Reading phonological recoding and deep dyslexia*. London: Routledge & Kegan Paul.
- Coltheart, M., Rastle, K., Perry, C., Langdon, R., & Ziegler, J. (2001). DRC: A dual route cascaded model of visual word recognition and reading aloud. *Psychological Review*, 108, 204-256.
- Coltheart, V., & Leahy, J. (1992). Childrens and adults reading of nonwords: Effects of regularity and consistency. *Journal of Experimental Psychology-Learning Memory and Cognition, 18*, 718-729.
- Compton, D. L., DeFries, J. C., & Olson, R. K. (2001). Are RAN- and phonological awareness-deficits additive in children with reading disabilities? *Dyslexia*, 7, 125-149.
- Corina, D. P., Richards, T. L., Serafini, S., Richards, A. L., Steury, K., Abbott, R. D., et al. (2001). FMRI auditory language differences between dyslexic and able reading children. *Neuroreport*, 12, 1195-1201.

- Cornelissen, P., Richardson, A., Mason, A., Fowler, S., & Stein, J. (1995). Contrast sensitivity and coherent motion detection measured at photopic luminance levels in dyslexics and controls. *Vision Research*, 35, 1483-1494.
- Coulson, S., King, J. W., & Kutas, M. (1998). Expect the unexpected: Event-related brain response to morphosyntactic violations. *Language and Cognitive Processes*, 13, 21-58.
- Courchesne, E. (1978). Neurophysiological correlates of cognitive-development: Changes in long-latency event-related potentials from childhood to adulthood. *Electroencephalography and Clinical Neurophysiology, 45*, 468-482.
- Crainthoreson, C., & Dale, P. S. (1992). Do early talkers become early readers: Llinguistic precocity, preschool language, and emergent literacy. *Developmental Psychology*, 28, 421-429.
- Critchley, M. (1970). Definitions of dyslexia. Journal of Learning Disabilities, 3, 281-281.
- de Lemos, M. M. (1989a). *Standard progressive matrices: Australian manual*. Melbourne: ACER.
- de Lemos, M. M. (1989b). The Australian re-standardisation of the standard progressive matrices. *Psychological Test Bulletin, 2*, 17-24.
- Deeney, T., Wolf, M., & O'Rourke, A. G. (2001). "I like to take my own sweet time": Case study of a child with naming-speed deficits and reading disabilities. *Journal of Special Education*, 35, 145-155.
- Dehaene, S. (1995). Electrophysiological evidence for category-specific word-processing in the normal human brain. *Neuroreport, 6*, 2153-2157.
- Dehaene, S., Le Clec'H, G., Poline, J. B., Le Bihan, D., & Cohen, L. (2002). The visual word form area: A prelexical representation of visual words in the fusiform gyrus. *Neuroreport*, 13, 321-325.
- Demonet, J. F., Taylor, M. J., & Chaix, Y. (2004). Developmental dyslexia. *Lancet*, 363, 1451-1460.
- Denckla, M. B., & Rudel, R. G. (1976). Rapid automatized naming (RAN): Dyslexia differentiated from other learning-disabilities. *Neuropsychologia*, 14, 471-479.
- Denton, C. A., Fletcher, J. M., Anthony, J. L., & Francis, D. J. (2006). An evaluation of intensive intervention for students with persistent reading difficulties. *Journal of Learning Disabilities*, 39, 447-466.
- Deweirdt, W. (1988). Speech-perception and frequency discrimination in good and poor readers. *Applied Psycholinguistics*, *9*, 163-183.
- Dibels. (2000). Dynamic indicators of basic early literacy skills. Retrieved 10/04, 2008, from https://dibels.uoregon.edu/measures/materials.php
- Dore, W., & Rutherford, R. (2001). Closing the gap. Retrieved 12/02, 2008, from http://www.bdainternationalconference.org/2001/presentations/sat\_s1\_a\_3.htm

- Dryer, R., Beale, I. L., & Lambert, A. J. (1999). The balance model of dyslexia and remedial training: An evaluative study. *Journal of Learning Disabilities*, *32*, 174-186.
- DSM-IV. (1994). *Diagnostic and statistical manual of mental disorders: DSM-IV*: American Psychiatric Association.
- Dunn, B. R., Dunn, D. A., Languis, M., & Andrews, D. (1998). The relation of ERP components to complex memory processing. *Brain and Cognition*, 36, 355-376.
- Dunn, L. M., & Dunn, L. M. (1981). *Peabody picture vocabulary test- Revised*. Minnesota: American Guidance Service.
- Dunning, D. B., Mason, J. M., & Stewart, J. P. (1994). Reading to preschoolers: A response to Scarborough and Dobrich (1994) and recommendations for future-research. *Developmental Review*, 14, 324-339.
- Eckert, M. A., & Leonard, C. M. (2000). Structural imaging in dyslexia: The planum temporale. *Mental Retardation and Developmental Disabilities Research Reviews*, *6*, 198-206.
- Eckert, M. A., Lombardino, L. J., & Leonard, C. M. (2001). Planar asymmetry tips the phonological playground and environment raises the bar. *Child Development*, 72, 988-1002.
- Ehri, L. C. (2002). Phases of acquisition in learning to read words and implications for teaching. *British Journal of Educational Psychology: Monograph Series*, 1, 7-28.
- Ehri, L. C. (2005). Development of sight word reading: Phases and findings. In M. J.Snowling & C. Hulme (Eds.), *The science of reading: A handbook* (pp. 135-154). UK, Oxford: Blackwell Publishing.
- Ellis, N. (1989). Reading development, dyslexia and phonological skills. *Irish Journal of Psychology*, *10*, 551-567.
- Everatt, J., Weeks, S., & Brooks, P. (2008). Profiles of strengths and weaknesses in dyslexia and other learning difficulties. *Dyslexia*, 14, 16-41.
- Fabiani, M., Gratton, G., & Coles, M. G. H. (2000). Event-related brain potentials: Methods, theory, and applications. In J. Cacioppo, T. L. G. Tassinary & G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 53-84). USA: Cambridge University Press.
- Farmer, M. E., & Klein, R. (1993). Auditory and visual temporal processing in dyslexic and normal readers. *Annals of the New York Academy of Sciences, 682*, 339-341.
- Farmer, M. E., & Klein, R. M. (1995). The evidence for a temporal processing deficit linked to dyslexia: A review. *Psychonomic Bulletin & Review*, 2, 460-493.
- Fawcett, A. J., & Nicolson, R. I. (1994). Naming speed in children with dyslexia. Journal of Learning Disabilities, 27, 641-646.
- Fawcett, A. J., & Nicolson, R. I. (2004). *The dyslexia screening test- Secondary (DST-S)*. UK, London: Harcourt Assessment.

- Fawcett, A. J., Nicolson, R. I., & Dean, P. (1996). Impaired performance of children with dyslexia on a range of cerebellar tasks. *Annals of Dyslexia*, *46*, 259-283.
- Fisher, S. E., & DeFries, J. C. (2002). Developmental dyslexia: Genetic dissection of a complex cognitive trait. *Nature Reviews Neuroscience*, 3, 767-780.
- Fletcher, J. M., Denton, C., & Francis, D. J. (2005). Validity of alternative approaches for the identification of learning disabilities: Operationalizing unexpected underachievement. *Journal of Learning Disabilities*, 38, 545-552.
- Flynn, J. M., & Rahbar, M. H. (1994). Prevalence of reading failure in boys compared with girls. *Psychology in the Schools, 31*, 66-71.
- Fonseca, L. C., Tedrus, G., & Gilbert, M. A. R. (2006). Event related potentials during the visual discrimination of words and pseudowords by children. *Arquivos De Neuro-Psiquiatria, 64*, 553-558.
- Foorman, B., Breier, J., & Fletcher, J. (2003). Interventions aimed at improving reading success: An evidence-based approach. *Developmental Neuropsychology*, 24, 613-639.
- Foorman, B. R., Francis, D. J., Fletcher, J. M., Schatschneider, C., & Mehta, P. (1998). The role of instruction in learning to read: Preventing reading failure in at-risk children. *Journal of Educational Psychology*, 90, 37-55.
- Friederici, A. D., Gunter, T. C., Hahne, A., & Mauth, K. (2004). The relative timing of syntactic and semantic processes in sentence comprehension. *Neuroreport*, 15, 165-169.
- Friederici, A. D., Opitz, B., & von Cramon, D. Y. (2000). Segregating semantic and syntactic aspects of processing in the human brain: An fMRI investigation of different word types. *Cerebral Cortex*, 10, 698-705.
- Friederici, A. D., Steinhauer, K., & Frisch, S. (1999). Lexical integration: Sequential effects of syntactic and semantic information. *Memory & Cognition*, 27, 438-453.
- Friedman, D., Boltri, J., Vaughan, H., & Erlenmeyerkimling, L. (1985). Effects of age and sex on the endogenous brain potential components during 2 continuous performancetasks. *Psychophysiology*, 22, 440-452.
- Frith, U. (1985). Beneath the surface of developmental dyslexia. In K. E. Patterson, J. C. Marshall & M. Coltheart (Eds.), *Surface dyslexia: Neuropsychological and cognitive studies of phonological reading* (pp. 301-330). London: Erlbaum.
- Frith, U. (1997). Brain, mind and behaviour in dyslexia. In C. Hulme & M. J. Snowling (Eds.), *Dyslexia: Biology, cognition and intervention* (pp. 1-19). London: Whurr.
- Fulbright, R. K., Jenner, A. R., Mencl, W. E., Pugh, K. R., Shaywitz, B. A., Shaywitz, S. E., et al. (1999). The cerebellum's role in reading: A functional MR imaging study. *American Journal of Neuroradiology*, 20, 1925-1930.
- Galaburda, A., & Livingstone, M. (1993). Evidence for a magnocellular defect in developmental dyslexia. Annals of the New York Academy of Sciences, 682, 70-82.

- Galaburda, A. M., & Kemper, T. L. (1979). Cytoarchitectonic abnormalities in developmental dyslexia: Case-study. *Annals of Neurology*, 6, 94-100.
- Galaburda, A. M., Sherman, G. F., Rosen, G. D., Aboitiz, F., & Geschwind, N. (1985). Developmental dyslexia: 4 consecutive patients with cortical anomalies. *Annals of Neurology*, 18, 222-233.
- Gambrell, L. B., Palmer, B. M., Codling, R. M., & Mazzoni, S. A. (1996). Assessing motivation to read. *Reading Teacher*, 49, 518-533.
- Ganis, G., Kutas, M., & Sereno, M. I. (1996). The search for "common sense": An electrophysiological study of the comprehension of words and pictures in reading. *Journal of Cognitive Neuroscience*, 8, 89-106.
- Garnsey, S. M., Tanenhaus, M. K., & Chapman, R. M. (1989). Evoked-potentials and the study of sentence comprehension. *Journal of Psycholinguistic Research*, 18, 51-60.
- Georgiewa, P., Rzanny, R., Gaser, C., Gerhard, U. J., Vieweg, U., Freesmeyer, D., et al. (2002). Phonological processing in dyslexic children: A study combining functional imaging and event related potentials. *Neuroscience Letters*, 318, 5-8.
- Georgiewa, P., Rzanny, R., Hopf, J. M., Knab, R., Glauche, V., Kaiser, W. A., et al. (1999). FMRI during word processing in dyslexic and normal reading children. *Neuroreport*, 10, 3459-3465.
- Gernsbacher, M. (1994). Handbook of psycholinguistics. New York: Academic Press.
- Geschwind, N. (1970). Organization of language and brain. Science, 170, 940.
- Glushko, R. J. (1979). Organization and activation of orthographic knowledge in reading aloud. *Journal of Experimental Psychology-Human Perception and Performance*, 5, 674-691.
- Goswami, U. (2004). Neuroscience and education. British Journal of Educational Psychology, 74, 1-14.
- Goswami, U., & Bryant, P. (1990). *Phonological skills and learning to read*. Hillsdale, NJ: Erlbaum.
- Grace, G. M., & Spreen, O. (1994). Hemisphere-specific stimulation of l-and p-types: A replication study and a critical appraisal. In R. Licht & G. Spyer (Eds.), *The balance model of dyslexia* (pp. 133-181). Assen, The Netherlands: Van Gorcum.
- Grande, C. G. (1988). Delinquency: The learning-disabled students reaction to academic school failure. *Adolescence*, 23, 209-219.
- Greatrex, J. C., & Drasdo, N. (1995). The magnocellular deficit hypothesis in dyslexia: A review of reported evidence. *Ophthalmic and Physiological Optics*, *15*, 501-506.
- Greenham, S., Stelmack, R., & van der Vlugt, H. (2003). Learning disability subtypes and the role of attention during the naming of pictures and words: An event-related potential analysis. *Developmental Neuropsychology 23*, 339-358.

- Griffiths, Y. M., & Snowling, M. J. (2002). Predictors of exception word and nonword reading in dyslexic children: The severity hypothesis. *Journal of Educational Psychology*, 94, 34-43.
- Grigorenko, E. L. (2001). Developmental dyslexia: An update on genes, brains, and environments. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 42, 91-125.
- Grunling, C., Ligges, M., Huonker, R., Klingert, M., Mentzel, H. J., Rzanny, R., et al. (2004). Dyslexia: The possible benefit of multimodal integration of fMRI- and EEGdata. *Journal of Neural Transmission*, 111, 951-969.
- Gunter, T. C., Stowe, L. A., & Mulder, G. (1997). When syntax meets semantics. *Psychophysiology*, *34*, 660-676.
- Habib, M. (2000). The neurological basis of developmental dyslexia: An overview and working hypothesis. *Brain, 123*, 2373-2399.
- Habib, M., & Robichon, F. (1996). Parietal lobe morphology predicts phonological skills in developmental dyslexia. *Brain and Cognition*, 32, 139-142.
- Hackley, S. A., Woldorff, M., & Hillyard, S. A. (1990). Cross-modal selective attention effects on retinal, myogenic, brain-stem, and cerebral evoked-potentials. *Psychophysiology*, 27, 195-208.
- Hagoort, P. (2003). Interplay between syntax and semantics during sentence comprehension:
   ERP effects of combining syntactic and semantic violations. *Journal of Cognitive Neuroscience*, 15, 883-899.
- Hagoort, P., Brown, C., & Groothusen, J. (1993). The syntactic positive shift (sps) as an ERP measure of syntactic processing. *Language and Cognitive Processes*, 8, 439-483.
- Hairston, W. D., Burdette, J. H., Flowers, D. L., Wood, F. B., & Wallace, M. T. (2005).
   Altered temporal profile of visual-auditory multisensory interactions in dyslexia.
   Experimental Brain Research, 166, 474-480.
- Hallahan, D. P., Llyod, J.W., Kauffman, J.M., Weiss, M., & Martinez, E.A. (2005). Learning disabilities: Foundations, characteristics, and effective teaching. Boston: Allyn & Bacon.
- Hammill, D. D., Mather, N., Allen, E. A., & Roberts, R. (2002). Using semantics, grammar, phonology, and rapid naming tasks to predict word identification. *Journal of Learning Disabilities*, 35, 121-136.
- Hansen, P. C., Stein, J. F., Orde, S. R., Winter, J. L., & Talcott, J. B. (2001). Are dyslexics' visual deficits limited to measures of dorsal stream function? *Neuroreport*, 12, 1527-1530.
- Harbin, T. J., Marsh, G. R., & Harvey, M. T. (1984). Differences in the late components of the event-related potential due to age and to semantic and non-semantic tasks. *Electroencephalography and Clinical Neurophysiology*, 59, 489-496.

- Harm, M. W., McCandliss, B. D., & Seidenberg, M. S. (2003). Modeling the successes and failures of interventions for disabled readers. *Scientific Studies of Reading*, 7, 155-182.
- Harm, M. W., & Seidenberg, M. S. (1999). Phonology, reading acquisition, and dyslexia: Insights from connectionist models. *Psychological Review*, 106, 491-528.
- Hart, B., & Risley, T. R. (1992). American parenting of language-learning children: Persisting differences in family-child interactions observed in natural home environments. *Developmental Psychology*, 28, 1096-1105
- Hatcher, P. J., Hulme, C., & Ellis, A. W. (1994). Ameliorating early reading failure by integrating the teaching of reading and phonological skills: The phonological linkage hypothesis. *Child Development*, 65, 41-57.
- Hauk, O., Davis, M. H., Ford, M., Pulvermuller, F., & Marslen-Wilson, W. D. (2006). The time course of visual word recognition as revealed by linear regression analysis of ERP data. *Neuroimage*, 30, 1383-1400.
- Helenius, P., Salmelin, R., Service, E., & Connolly, J. F. (1999a). Semantic cortical activation in dyslexic readers. *Journal of Cognitive Neuroscience*, 11, 535-550.
- Helenius, P., Tarkiainen, A., Cornelissen, P., Hansen, P. C., & Salmelin, R. (1999b).
  Dissociation of normal feature analysis and deficient processing of letter-strings in dyslexic adults. *Cerebral Cortex*, 9, 476-483.
- Hemstim. (2008). Hss computer software. Retrieved 02/02, 2008, from www.pits-online.nl
- Hinojosa, J. A., Martin-Loeches, M., & Rubia, F. J. (2001). Event-related potentials and semantics: An overview and an integrative proposal. *Brain and Language*, 78, 128-139.
- Hinshaw, S. P. (1992). Externalizing behavior problems and academic underachievement in childhood and adolescence: Causal relationships and underlying mechanisms. *Psychological Bulletin*, 111, 127-155.
- Hinshelwood, J. (1907). Four cases of congenital word-blindness occuring in the same family. *The British Medical Journal*, *2*, 1303.
- Holcomb, P. J., Ackerman, P. T., & Dykman, R. A. (1985). Cognitive event-related brain potentials in children with attention and reading deficits. *Psychophysiology*, 22, 656-667.
- Holcomb, P. J., Ackerman, P. T., & Dykman, R. A. (1986). Auditory event-related potentials in attention and reading disabled boys. *International Journal of Psychophysiology*, *3*, 263-273.
- Holcomb, P. J., Coffey, S. A., & Neville, H. J. (1992). Visual and auditory sentence processing: A developmental analysis using event-related brain potentials. *Developmental Neuropsychology*, 8, 203-241.
- Holcomb, P. J., & McPherson, W. B. (1994). Event-related brain potentials reflect semantic priming in an object decision task. *Brain and Cognition*, 24, 259-276.

- Horwitz, B., Rumsey, J. M., & Donohue, B. C. (1998). Functional connectivity of the angular gyrus in normal reading and dyslexia. *Proceedings of the National Academy of Sciences of the United States of America*, 95, 8939-8944.
- Hutzler, F., Kronbichler, M., Jacobs, A. M., & Wimmer, H. (2006). Perhaps correlational but not causal: No effect of dyslexic readers' magnocellular system on their eye movements during reading. *Neuropsychologia*, 44, 637-648.
- Hynd, G. W. (1992). Neurological aspects of dyslexia: Comment on the balance model. *Journal of Learning Disabilities*, 25, 110.
- Hynd, G. W., & Semrudclikeman, M. (1989). Dyslexia and brain morphology. *Psychological Bulletin*, 106, 447-482.
- Irlen, H., & Lass, M. J. (1989). Improving reading problems due to symptoms of scotopic sensitivity syndrome using irlen lenses and overlays. *Education*, 109, 413-417.
- Ivens, S. M., Koslin, S. H., & Zeno, B. L., et al. (1995). The educator's word frequency guide. USA: Touchstone Applied Science Associates.
- Jasper, A. W. (1958). The consumer interview approach in solving our egg industry problems. *Poultry Science*, *37*, 1215-1216.
- Jeffries, S., & Everatt, J. (2004). Working memory: Its role in dyslexia and other specific learning difficulties. *Dyslexia*, 10, 196-214.
- Johnson, R. (1989). Developmental evidence for modality-dependent P300 generators: A normative study. *Psychophysiology*, *26*, 651-667.
- Jones, F. L., & McMillan, J. (2001). Scoring occupational categories for social research: A review of current practice, with Australian examples. Work Employment and Society, 15, 539-563.
- Jorm, A. F. (1983). Specific reading retardation and working memory: A review. *British Journal of Psychology*, 74, 311-342.
- Jorm, A. F., & Share, D. L. (1983). Phonological recoding and reading acquisition. *Applied Psycholinguistics*, *4*, 103-147.
- Kame'enui, E., Simmons, D., Good, R., & Harn, B. (2000). The use of fluency-based measures in early identification and evaluation of intervention efficacy in schools.
   Parkton: MD: York.
- Kappers, E. J. (1997). Outpatient treatment of dyslexia through stimulation of the cerebral hemispheres. *Journal of Learning Disabilities, 30,* 100-125.
- Kaufmann, W. E., & Galaburda, A. M. (1989). Cerebrocortical microdysgenesis in neurologically normal subjects: A histopathologic study. *Neurology*, 39, 238-244.
- Kavale, K. A. (2005). Identifying specific learning disability: Is responsiveness to intervention the answer? *Journal of Learning Disabilities, 38*, 553-562.

- King, W. M., Giess, S. A., & Lombardino, L. J. (2007). Subtyping of children with developmental dyslexia via bootstrap aggregated clustering and the gap statistic: Comparison with the double-deficit hypothesis. *International Journal of Language & Communication Disorders*, 42, 77-95.
- Kinsbourne, M., Rufo, D. T., Gamzu, E., Palmer, R. L., & Berliner, A. K. (1991). Neuropsychological deficits in adults with dyslexia. *Developmental Medicine and Child Neurology*, 33, 763-775.
- Kirk, R. E. (1982). *Experimental design: Procedures for the behavioral sciences*. Belmont, CA: Brooks/Cole.
- Klingberg, T., Hedehus, M., Temple, E., Salz, T., Gabrieli, J. D. E., Moseley, M. E., et al. (2000). Microstructure of temporo-parietal white matter as a basis for reading ability: Evidence from diffusion tensor magnetic resonance imaging. *Neuron*, 25, 493-500.
- Kok, A. (2001). On the utility of P3 amplitude as a measure of processing capacity. *Psychophysiology*, *38*, 557-577.
- Kounios, J., & Holcomb, P. J. (1994). Concreteness effects in semantic processing: ERP evidence supporting dual-coding theory. *Journal of Experimental Psychology-Learning Memory and Cognition, 20*, 804-823.
- Kramer, A. F., Strayer, D. L., & Buckley, J. (1991). Task versus component consistency in the development of automatic processing: A psychophysiological assessment. *Psychophysiology*, 28, 425-437.
- Kronbichler, M., Hutzler, F., & Wimmer, H. (2002). Dyslexia: Verbal impairments in the absence of magnocellular impairments. *Neuroreport*, *13*, 617-620.
- Kucera, H., & Francis, W. N. (1967). *Computational analysis of present-day american english*. Providence: Brown University Press.
- Kulkarni, M., Kalantre, S., Upadhye, S., Karande, S., & Ahuja, S. (2001). Approach to learning disability. *Indian Journal of Pediatrics*, 68, 539-546.
- Kuperberg, G. R. (2007). Neural mechanisms of language comprehension: Challenges to syntax. *Brain Research*, 1164, 142-142.
- Kuperberg, G. R., Sitnikova, T., Caplan, D., & Holcomb, P. J. (2003). Electrophysiological distinctions in processing conceptual relationships within simple sentences. *Cognitive Brain Research*, 17, 117-129.
- Kutas, M. (1987). Event-related brain potentials (ERPs) elicited during rapid serial visual presentation of congruous and incongruous sentences. *Electroencephalography and Clinical Neurophysiology, Supplement 40*, 406-411.
- Kutas, M., & Hillyard, S. A. (1980). Reading senseless sentences: Brain potentials reflect semantic incongruity. *Science*, 207, 203-205.
- Kutas, M., & Hillyard, S. A. (1983). Event-related brain potentials to grammatical errors and semantic anomalies. *Memory & Cognition*, 11, 539-550.

- Kutas, M., & Hillyard, S. A. (1984). Brain potentials during reading reflect word expectancy and semantic association. *Nature*, 307, 161-163.
- Kutas, M., & Hillyard, S. A. (1989). An electrophysiological probe of incidental semantic association. *Journal of Cognitive Neuroscience*, 1, 38-49.
- Kutas, M., Lindamood, T. E., & Hillyard, S. A. (1984). Word expectancy and eventrelated brain potentials during sentence processing. In S. Kornblum & J. Requin (Eds.), *Preparatory states and processes* (pp. 217-237). Hillsdale, NJ: Erlbaum.
- Kutas, M., Van Petten, C., & Kluender, R. (2006). Psycholinguistics electrified ii: 1995-2005. In M. Traxler & M. A. Gernsbacher (Eds.), *Handbook of psycholinguistics* (pp. 659-724). New York: Elsevier.
- Kutas, M., & Van Petten, C. K. (1994). Psycholinguistics electrified: Event-related brain potential investigations. In M. Gernsbacher (Ed.), *Handbook of psycholinguistics* (pp. 83-56). New York: Academic Press.
- Landi, N., & Perfetti, C. A. (2007). An electrophysiological investigation of semantic and phonological processing in skilled and less-skilled comprehenders. *Brain and Language*, 102, 30-45.
- Langenberg, D. N. (2000). US national reading panel: Teaching children to read: Reports of the subgroups [Electronic Version], 1-449. Retrieved 09/10 from http://www.nichd.nih.gov/publications/nrp/smallbook.cfm.
- Larson, K. A. (1988). A research review and alternative hypothesis explaining the link between learning-disability and delinquency. *Journal of Learning Disabilities*, *21*, 357.
- Le Fevre, D. M., Moore, D. W., & Wilkinson, I. A. G. (2003). Tape-assisted reciprocal teaching: Cognitive bootstrapping for poor decoders. *British Journal of Educational Psychology*, 73, 37-58.
- Lehmkuhle, S., Garzia, R. P., Turner, L., Hash, T., & Baro, J. A. (1993). A defective visual pathway in children with reading-disability. *New England Journal of Medicine, 328*, 989-996.
- Lepola, J., Salonen, P., & Vauras, M. (2000). The development of motivational orientations as a function of divergent reading careers from pre-school to the second grade. *Learning and Instruction*, *10*, 153-177.
- Lewis, C., Hitch, G. J., & Walker, P. (1994). The prevalence of specific arithmetic difficulties and specific reading difficulties in 9-year-old to 10-year-old boys and girls. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 35*, 283-292.
- Licht, R., Bakker, D. J., Kok, A., & Bouma, A. (1988). The development of lateral eventrelated potentials (ERPs) related to word naming: A 4 year longitudinal-study. *Neuropsychologia*, 26, 327-340.

- Licht, R., Bakker, D. J., Kok, A., & Bouma, A. (1992). Grade-related changes in eventrelated potentials (ERPs) in primary-school children: Differences between 2 reading tasks. *Journal of Clinical and Experimental Neuropsychology*, 14, 193-210.
- Livingstone, M. S., Rosen, G. D., Drislane, F. W., & Galaburda, A. M. (1991). Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proceedings of the National Academy of Sciences of the United States of America*, 88, 7943-7947.
- Lorusso, M. L., Facoetti, A., Paganoni, P., Pezzani, M., & Molteni, M. (2006). Effects of visual hemisphere-specific stimulation versus reading-focused training in dyslexic children. *Neuropsychological Rehabilitation*, 16, 194-212.
- Lovegrove, W. (1993). Weakness in the transient visual-system: A causal factor in dyslexia. Annals of the New York Academy of Sciences, 682, 57-69.
- Lovegrove, W., Martin, F., & Slaghuis, W. (1986). A theoretical and experimental case for a visual deficit in specific reading-disability. *Cognitive Neuropsychology*, *3*, 225-267.
- Lovegrove, W. J., Garzia, R.P., & Nicholson, S.B. (1990). Experimental evidence for a transient system deficit in specific reading disability. *Journal of the American Optometric Association*, *61*, 137-146.
- Lovett, M. W., Lacerenza, L., & Borden, S. L. (2000b). Putting struggling readers on the phast track: A program to integrate phonological and strategy-based remedial reading instruction and maximize outcomes. *Journal of Learning Disabilities, 33*, 458-476.
- Lovett, M. W., & Steinbach, K. A. (1997). The effectiveness of remedial programs for reading disabled children of different ages: Does the benefit decrease for older children? *Learning Disability Quarterly, 20*, 189-210.
- Lovett, M. W., Steinbach, K. A., & Frijters, J. C. (2000a). Remediating the core deficits of developmental reading disability: A double-deficit perspective. *Journal of Learning Disabilities*, 33, 334-358.
- Lovrich, D., Cheng, J., & Velting, D. (1996). Late cognitive brain potentials, phonological and semantic classification of spoken words, and reading ability in children. *Journal of Clinical and Experimental Neuropsychology*, 18, 161-177.
- Lovrich, D., Cheng, J. C., & Velting, D. M. (2003). ERP correlates of form and rhyme letter tasks in impaired reading children: A critical evaluation. *Child Neuropsychology*, 9, 159-174.
- Lovrich, D., Kazmerski, V., Cheng, J. C., & Geisler, M. W. (1994). A developmental study of visual ERP distributions during spatial and phonetic processing. *Electroencephalography and Clinical Neurophysiology*, *90*, 103-113.
- Luck, S. J., Hillyard, S.A. (1994). Electrophysiological correlates of feature analysis during visual search. *Psychophysiology*, *31*, 291-308.

- Lundberg, I., Frost, J., & Petersen, O. P. (1988). Effects of an extensive program for stimulating phonological awareness in preschool-children. *Reading Research Quarterly*, 23, 263-284.
- Lupker, S. J. (2005). Visual word recognition: Theories and findings. In M. J. Snowling & C.Hulme (Eds.), *The science of reading: A handbook*. Oxford, UK: Blackwell Publishing.
- Lyon, G. R. (1995). Toward a definition of dyslexia. Annals of Dyslexia, 45, 3-27.
- Lyon, G. R., & Moats, L. C. (1997). Critical conceptual and methodological considerations in reading intervention research. *Journal of Learning Disabilities*, *30*, 578-588.
- Lyon, G. R., Shaywitz, S. E., & Shaywitz, B. A. (2003). A definition of dyslexia. Annals of Dyslexia, 53, 1-14.
- MacDonald, M. C., Pearlmutter, N. J., & Seidenberg, M. S. (1994). Lexical nature of syntactic ambiguity resolution. *Psychological Review*, 101, 676-703.
- MacPhee, K. (1998). Spell, read, phonological auditory training. Charlottetown, PEI: Learning Systems.
- Manis, F. R., Seidenberg, M. S., & Doi, L. M. (1999a). See dick ran: Rapid naming and the longitudinal prediction of reading subskills in first and second graders. *Scientific Studies* of Reading, 3, 129-157.
- Marsh, G., Friedman, M., Welch, V., & Desberg, P. (1981). A cognitive-developmental theory of reading acquisition. In G. MacKinnon & T. Waller (Eds.), *Reading research:* Advances in theory and practice (pp. 199-221). New York: Academic.
- Martin, F., Mackenzie, B., Lovegrove, W., & McNicol, D. (1993). Irlen lenses in the treatment of specific reading-disability: An evaluation of outcomes and processes. *Australian Journal of Psychology*, 45, 141-150.

Martin, F., & Pratt, C. (2001). The nonword reading test. Melbourne, Victoria: ACER.

- Martin-Loeches, M., Hinojosa, J. A., Gomez-Jarabo, G., & Rubia, F. J. (1999). The recognition potential: An ERP index of lexical access. *Brain and Language*, 70, 364-384.
- Marzola, E., Shepherd, M. (2005). Assessment of reading difficulties. In J. R. Birsh (Ed.), *Multisensory teaching of basic language skills* (pp. 171-185). Baltimore: MD: Brookes.
- Maskel, S., & Felton, R. (2001). Analysis of achievement at the hill leanring centre: 1990-1994. In C. McIntyre & J. Pickering (Eds.), *Clinical studies of multisensory structured language education for students with dyslexia and related disorders* (pp. 121-137).
  Dallas, TX: International Multisensory Structured Language Education Council.
- Mathes, P. G., & Denton, C. A. (2002). The prevention and identification of reading disability. *Seminars in Pediatric Neurology*, *9*, 185-191.
- Maurer, U., Brem, S., Bucher, K., Kranz, F., Benz, R., Steinhausen, H. C., et al. (2007). Impaired tuning of a fast occipito-temporal response for print in dyslexic children learning to read. *Brain*, 130, 3200-3210.

- May, J. G., Lovegrove, W. J., Martin, F., & Nelson, P. (1991). Pattern-elicited visual evoked-potentials in good and poor readers. *Clinical Vision Sciences*, *6*, 131-136.
- May, J. G., Williams, M. C., & Dunlap, W. P. (1988). Temporal-order judgements in good and poor readers. *Neuropsychologia*, *26*, 917-924.
- McAnally, K. I., & Stein, J. F. (1996). Auditory temporal coding in dyslexia. *Proceedings of* the Royal Society of London Series B-Biological Sciences, 263, 961-965.
- McCallum, W. C., Farmer, S. F., & Pocock, P. V. (1984). The effects of physical and semantic incongruities on auditory event-related potentials. *Electroencephalography* and Clinical Neurophysiology, 59, 477-488.
- McCandliss, B. D., Cohen, L., & Dehaene, S. (2003). The visual word form area: Expertise for reading in the fusiform gyrus. *Trends in Cognitive Sciences*, *7*, 293-299.
- McCandliss, B. D., & Noble, K. G. (2003). The development of reading impairment: A cognitive neuroscience model. *Mental Retardation and Developmental Disabilities Research Reviews*, *9*, 196-204.
- McCandliss, B. D., Posner, M. I., & Givon, T. (1997). Brain plasticity in learning visual words. *Cognitive Psychology*, *33*, 88-110.
- McClelland, J. L., & Plaut, D. C. (1993). Computational approaches to cognition top-down approaches. *Current Opinion in Neurobiology*, *3*, 209-216.
- McClelland, J. L., St John, M., & Taraban, R. (1989). Sentence comprehension: A parallel distributed processing approach. *Language and Cognitive Processes*, *4*, 287-335.
- McDonough, B. E., Warren, C. A., & Don, N. S. (1992). Event-related potentials in a guessing task the gleam in the eye effect. *International Journal of Neuroscience*, 65, 209-219.
- McPhillips, M. (2003). A commentary on an article published in the February 2003 edition of 'dyslexia', 'evaluation of an exercise-based treatment for children with reading difficulties' (Reynolds, Nicolson, & Hambly). *Dyslexia, 9*, 161-163.
- Merzenich, M. M., Jenkins, W. M., Johnston, P., Schreiner, C., Miller, S. L., & Tallal, P. (1996). Temporal processing deficits of language-learning impaired children ameliorated by training. *Science*, 271, 77-81.
- Meyer, M. S. (2000). The ability-achievement discrepancy: Does it contribute to an understanding of learning disabilities? *Educational Psychology Review*, *12*, 315-337.
- Miles, E., & Miles, T. R. (1990). Specific difficulties in reading and spelling. In G. M.Rajinder & P. Coxhead (Eds.), *Intervention with children* (pp. 199-214). Florence, KY, US: Taylor & Francis/Routledge.
- Miles, J., & Stelmack, R. M. (1994). Learning-disability subtypes and the effects of auditory and visual priming on visual event-related potentials to words. *Journal of Clinical and Experimental Neuropsychology*, *16*, 43-64.

- Miller-Shaul, S., & Breznitz, Z. (2004). Electrocortical measures during a lexical decision task: A comparison between elementary school-aged normal and dyslexic readers and adult normal and dyslexic readers. *Journal of Genetic Psychology*, *165*, 399-424.
- Mitchell, D. C., & Holmes, V. M. (1985). The role of specific information about the verb in parsing sentences with local structural ambiguity. *Journal of Memory and Language*, 24, 542-559.
- Morais, J. (1991). Phonological awareness: A bridge between language & literacy. In D. Sawyer & B. Fox (Eds.), *Phonological awareness in reading: The evolution of current perspectives* (pp. 31-71). New York: Springer-Verlag.
- Morgan, P. L., & Fuchs, D. (2007). Is there a bidirectional relationship between children's reading skills and reading motivation? *Exceptional Children*, 73, 165-183.
- Morgan, W. P. (1896). A case of congenital word-blindness. *The British Medical Journal*, 2, 1378-1379.
- Morris, R. D., Stuebing, K. K., Fletcher, J. M., Shaywitz, S. E., Lyon, G. R., Shankweiler, D.
  P., et al. (1998). Subtypes of reading disability: Variability around a phonological core. *Journal of Educational Psychology*, 90, 347-373.
- Morton, J. (1979). Facilitation in word recognition: Experiments causing change in the logogen model. In P. A. Kolers, M. E. Wrolstad & H. Bouma (Eds.), *Processing of visible language* (pp. 259-268). New York NY: Plenum Press.
- Mullis, R. J., Holcomb, P. J., Diner, B. C., & Dykman, R. A. (1985). The effects of aging on the p3 component of the visual event-related potential. *Electroencephalography and Clinical Neurophysiology*, 62, 141-149.
- Mummery, C. J., Patterson, K., Hodges, J. R., & Price, C. J. (1998). Functional neuroanatomy of the semantic system: Divisible by what? *Journal of Cognitive Neuroscience*, 10, 766-777.
- Munte, T. F. (1993). Event-related potentials in the analysis of language. Zeitschrift Fur Elektroenzephalographie Elektromyographie Und Verwandte Gebiete, 24, 34-40.
- Munte, T. F., Heinze, H. J., Matzke, M., Wieringa, B. M., & Johannes, S. (1998). Brain potentials and syntactic violations revisited: No evidence for specificity of the syntactic positive shift. *Neuropsychologia*, 36, 217-226.
- Neale, M. D. (1999). Neale analysis of reading ability. Melbourne, Victoria: ACER.
- Nelson, H. E. (1991). National adult reading test- Second edition. Berkshire: NFER- Nelson.
- Neuhaus, G. F., & Swank, P. R. (2002). Understanding the relations between ran letter subtest components and word reading in first-grade students. *Journal of Learning Disabilities*, 35, 158-174.
- Neville, H., Nicol, J. L., Barss, A., Forster, K. I., & Garrett, M. F. (1991). Syntactically based sentence processing classes: Evidence from event-related brain potentials. *Journal of Cognitive Neuroscience*, 3, 151-165.

- Neville, H. J., Coffey, S. A., Holcomb, P. J., & Tallal, P. (1993). The neurobiology of sensory and language processing in language-impaired children. *Journal of Cognitive Neuroscience*, 5, 235-253.
- Neville, H. J., Kutas, M., Chesney, G., & Schmidt, A. L. (1986). Event-related brain potentials during initial encoding and recognition memory of congruous and incongruous words. *Journal of Memory and Language*, 25, 75-92.
- Nicolson, R. I., & Fawcett, A. J. (1990). Automaticity: A new framework for dyslexia research. *Cognition*, 35, 159-182.
- Nicolson, R. I., & Fawcett, A. J. (1994). Reaction-times and dyslexia. *Quarterly Journal of Experimental Psychology*, 47, 29-48.
- Nicolson, R. I., Fawcett, A. J., Berry, E. L., Jenkins, I. H., Dean, P., & Brooks, D. J. (1999). Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. *Lancet*, 353, 1662-1667.
- Nicolson, R. I., Fawcett, A. J., & Dean, P. (2001). Developmental dyslexia: The cerebellar deficit hypothesis. *Trends in Neurosciences*, 24, 508-511.
- Nigam, A., Hoffman, J. E., & Simons, R. F. (1992). N400 to semantically anomalous pictures and words. *Journal of Cognitive Neuroscience*, *4*, 15-22.
- Niznikiewicz, M., & Squires, N. K. (1996). Phonological processing and the role of strategy in silent reading: Behavioral and electrophysiological evidence. *Brain and Language*, *52*, 342-364.
- Noble, K. G., & McCandliss, B. D. (2005). Reading development and impairment: Behavioral, social, and neurobiological factors. *Journal of Developmental and Behavioural Pediatrics 26*, 370-378.
- Nobre, A. C., & McCarthy, G. (1994). Language-related ERPs: Scalp distributions and modulation by word type and semantic priming. *Journal of Cognitive Neuroscience*, *6*, 233-255.
- Oakland, T., Black, J. L., Stanford, G., Nussbaum, N. L., & Balise, R. R. (1998). An evaluation of the dyslexia training program: A multisensory method for promoting reading in students with reading disabilities. *Journal of Learning Disabilities*, 31, 140-147.
- Olson, R., & Byrne, B. (2005). Genetic and environmental influences on reading and language ability and disability. In H. Catts & A. Kamhi (Eds.), *The connections between language and reading disability* (pp. 173-200). Hillsdale, NJ: Erlbaum.
- Olson, R. K., Kliegl, R., & Davidson, B. J. (1983). Dyslexic and normal readers' eyemovements. *Journal of Experimental Psychology*, 9, 816-825.
- Osterhout, L., Holcomb, P. J., & Swinney, D. A. (1994). Brain potentials elicited by gardenpath sentences: Evidence of the application of verb information during parsing. *Journal* of Experimental Psychology-Learning Memory and Cognition, 20, 786-803.

- Osterhout, L., McKinnon, R., Bersick, M., & Corey, V. (1996). On the language specificity of the brain response to syntactic anomalies: Is the syntactic positive shift a member of the P300 family? *Journal of Cognitive Neuroscience*, *8*, 507-526.
- Osterhout, L., & Nicol, J. (1999). On the distinctiveness, independence, and time course of the brain responses to syntactic and semantic anomalies. *Language and Cognitive Processes*, 14, 283-317.
- Pae, H. K., Wise, J. C., Cirino, P. T., Sevcik, R. A., Lovett, M. W., Wolf, M., et al. (2005). The Woodcock Rading Mastery test: Impact of normative changes. *Assessment*, 12, 347-357.
- Paulesu, E., Demonet, J. F., Fazio, F., Mc Crory, E., Chanoine, V., Brunswick, N., et al. (2001). Cultural diversity and biological unity in dyslexia. *Neuroimage*, 13, 584-584.
- Paulesu, E., Frith, U., Snowling, M., Gallagher, A., Morton, J., Frackowiak, R. S. J., et al. (1996). Is developmental dyslexia a disconnection syndrome? Evidence from PET scanning. *Brain*, 119, 143-157.
- Peer, L. (2003). Commentary on Reynolds et al. (2003): Directions for future research. *Dyslexia*, 9, 123-124.
- Pennington, B. F. (2006). From single to multiple deficit models of developmental disorders. *Cognition, 101*, 385-413.
- Pennington, B. F., Cardoso-Martins, C., Green, P. A., & Lefly, D. L. (2001). Comparing the phonological and double deficit hypotheses for developmental dyslexia. *Reading and Writing*, 14, 707-755.
- Pennington, B. F., & Gilger, J. W. (1996). How is dyslexia transmitted? In C. H. Chase, G. D. Rosen & G. F. Sherman (Eds.), *Developmental dyslexia. Neural, cognitive, and genetic mechanisms* (pp. 41-61). Baltimore, MD: York Press.
- Pennington, B. F., Groisser, D., & Welsh, M. C. (1993). Contrasting cognitive deficits in attention-deficit hyperactivity disorder versus reading-disability. *Developmental Psychology*, 29, 511-523.
- Pennington, B. F., Vanorden, G. C., Smith, S. D., Green, P. A., & Haith, M. M. (1990). Phonological processing skills and deficits in adult dyslexics. *Child Development*, 61, 1753-1778.
- Penolazzi, B., Spironelli, C., Vio, C., & Angrilli, A. (2006). Altered hemispheric asymmetry during word processing in dyslexic children: An event-related potential study. *Neuroreport*, 17, 429-433.
- Phillips, J. A., Noppeney, U., Humphreys, G. W., & Price, C. J. (2002). Can segregation within the semantic system account for category-specific deficits? *Brain*, 125, 2067-2080.

- Picton, T. W., Bentin, S., Berg, P., Donchin, E., Hillyard, S. A., Johnson, R., et al. (2000). Guidelines for using human event-related potentials to study cognition: Recording standards and publication criteria. *Psychophysiology*, 37, 127-152.
- Plante, E., Van Petten, C., & Senkfor, A. J. (2000). Electrophysiological dissociation between verbal and nonverbal semantic processing in learning disabled adults. *Neuropsychologia*, 38, 1669-1684.
- Plaut, D. C. (2005). Connectionist approaches to reading. In M. J. Snowling & C. Hulme (Eds.), *The science of reading: A handbook* (pp. 24-39). UK, Oxford: Blackwell Publishing.
- Pokorni, J. L., Worthington, C. K., & Jamison, P. J. (2004). Phonological awareness intervention: Comparison of Fast Forword, Earobics, and LIPS. *Journal of Educational Research*, 97, 147-157.
- Poldrack, R. A., Wagner, A. D., Prull, M. W., Desmond, J. E., Glover, G. H., & Gabrieli, J.
  D. E. (1999). Functional specialization for semantic and phonological processing in the left inferior prefrontal cortex. *Neuroimage*, 10, 15-35.
- Poskiparta, E., Niemi, P., Lepola, J., Ahtola, A., & Laine, P. (2003). Motivational-emotional vulnerability and difficulties in learning to read and spell. *British Journal of Educational Psychology*, 73, 187-206.
- Posner, M. I., Abdullaev, Y., McCandliss, B. D., & Sereno, S. C. (1999). Neuroanatomy, circuitry and plasticity of word reading. *Neuroreport*, 10, Cover 3.
- Pratt, C., Kemp, N., & Martin, F. (1996). Sentence context and word recognition in children with average reading ability and with a specific reading disability. *Australian Journal of Psychology*, 48, 155-159.
- Preston, M. S., & Guthrie, J. T. (1974). Visual evoked-responses (VERs) in normal and disabled readers. *Psychophysiology*, 11, 452-457.
- Price, C. J., Wise, R. J. S., & Frackowiak, R. S. J. (1996). Demonstrating the implicit processing of visually presented words and pseudowords. *Cerebral Cortex*, 6, 62-70.
- Prideaux, L. A., Marsh, K. A., & Caplygin, D. (2005). Efficacy of the Cellfield Intervention for reading difficulties: An integrated computer-based approach targeting deficits associated with dyslexia. *Australian Journal of Learning Disabilities*, 10, 51-62.
- Pritchard, W. S., Shappell, S. A., & Brandt, M. E. (1991). Psychophysiology of N200/N400:
  A review and classification scheme. In Jennings, Ackles & M. G. H. Coles (Eds.),
  Advances in psychophysiology (pp. 43-106). Lodnon: Jessica Kingsley Ltd.
- Ptok, M., Berendes, K., Gottal, S., Grabherr, B., Schneeberg, J., & Wittler, M. (2007). Developmental dyslexia. The role of phonological processing for the development of literacy. *Hals-, Nasen-, Ohren-Heilkunde, 55*, 737-746.

- Pugh, K. R., Shaywitz, B. A., Shaywitz, S. E., Constable, R. T., Skudlarski, P., Fulbright, R. K., et al. (1996). Cerebral organization of component processes in reading. *Brain*, 119, 1221-1238.
- Rack, J. (2003). The who, what, why and how of intervention programmes: Comments on the DDAT evaluation. *Dyslexia*, *9*, 137-139.
- Rack, J. P. (1985). Orthographic and phonetic coding in developmental dyslexia. British Journal of Psychology, 76, 325-340.

ł

1

- Rack, J. P., Snowling, M. J., Hulme, C., & Gibbs, S. (2007). No evidence that an exercisebased treatment programme (DDAT) has specific benefits for children with reading difficulties. *Dyslexia*, 13, 97-104.
- Rack, J. P., Snowling, M. J., & Olson, R. K. (1992). The nonword reading deficit in developmental dyslexia: A review. *Reading Research Quarterly*, 27, 28-53.
- Radeau, M., Besson, M., Fonteneau, E., & Castro, S. L. (1998). Semantic, repetition and rime priming between spoken words: Behavioral and electrophysiological evidence. *Biological Psychology*, 48, 183-204.
- Rae, C., Lee, M. A., Dixon, R. M., Blamire, A. M., Thompson, C. H., Styles, P., et al. (1998). Metabolic abnormalities in developmental dyslexia detected by h-1 magnetic resonance spectroscopy. *Lancet*, 351, 1849-1852.
- Ramus, F. (2001). Dyslexia: Talk of two theories. Nature, 412, 393-395.
- Ramus, F. (2003). Developmental dyslexia: Specific phonological deficit or general sensorimotor dysfunction? *Current Opinion in Neurobiology*, 13, 212-218.
- Rastle, K., Harrington, J., & Coltheart, M. (2002). 58,534 nonwords: The ARC nonword database. *Quarterly Journal of Experimental Psychology*, *3*, 1339-1362.
- Raven, J. C. (1938). Standard progressive matrices. Victoria: ACER.
- Rayner, K., Garrod, S., & Perfetti, C. A. (1992). Discourse influences during parsing are delayed. *Cognition*, 45, 109-139.
- Rayner, K., Juhasz, B. J., & Pollatsek, A. (2005). Eye movements during reading. In M. J.Snowling & C. Hulme (Eds.), *The science of reading: A handbook*. UK, Oxford:Blackwell Publishing.
- Repovs, G., & Baddeley, A. (2006). The multi-component model of working memory: Explorations in experimental cognitive psychology. *Neuroscience*, 139, 5-21.
- Reynolds, D., Nicolson, R.I., & Hambly, H. (2003). Evaluation of an exercise-based treatment for children with reading difficulties. *Dyslexia*, *9*, 48-71.
- Reynolds, D., & Nicolson, R. I. (2007). Follow-up of an exercise-based treatment for children with reading difficulties. *Dyslexia*, 13, 78-96.

- Richards, G. E., Ellis, L. A., Neill, J. T. (2002). The ROPELOC: Review of personal effectiveness and locus of control: A comprehensive instrument for reviewing life effectiveness. *Paper presented at Self-Concept Research: Driving International Research Agendas, 6-8 August, Sydney.*
- Richards, I. L., Moores, E., Witton, C., Reddy, P. A., Rippon, G., Rochelle, K. S. H., et al. (2003). Science, sophistry and 'commercial sensitivity': Comments on 'Evaluation of an exercise-based treatment for children with reading difficulties', by Reynolds, Nicolson and Hambly. *Dyslexia*, 9, 146-150.
- Richards, T. L., Berninger, V. W., Aylward, E. H., Richards, A. L., Thomson, J. B., Nagy,
  W. E., et al. (2002). Reproducibility of proton mr spectroscopic imaging (PEPSI):
  Comparison of dyslexic and normal-reading children and effects of treatment on brain lactate levels during language tasks. *American Journal of Neuroradiology, 23*, 1678-1685.
- Riva, D., & Giorgi, C. (2000). The cerebellum contributes to higher functions during development: Evidence from a series of children surgically treated for posterior fossa tumours. *Brain*, 123, 1055-1061.
- Robichon, F., Besson, M., & Habib, M. (2002). An electrophysiological study of dyslexic and control adults in a sentence reading task. *Biological Psychology*, *59*, 29-53.
- Romani, A., Conte, S., Callieco, R., Bergamaschi, R., Versino, M., Lanzi, G., et al. (2001). Visual evoked potential abnormalities in dyslexic children. *Functional Neurology*, 16, 219-229.
- Rumsey, J. M., Andreason, P., Zametkin, A. J., Aquino, T., King, A. C., Hamburger, S. D., et al. (1992). Failure to activate the left temporoparietal cortex in dyslexia: An o-15 positron emission tomographic study. *Archives of Neurology*, 49, 527-534.
- Rumsey, J. M., Nace, K., Donohue, B., Wise, D., Maisog, J. M., & Andreason, P. (1997). A positron emission tomographic study of impaired word recognition and phonological processing in dyslexic men. *Archives of Neurology*, 54, 562-573.
- Russeler, J., Johannes, S., Kowalczuk, J., Wieringa, B. M., & Munte, T. F. (2003). Developmental dyslexics show altered allocation of attention in visual classification tasks. *Acta Neurolology Scandinavia*, 107, 22-30.
- Sabisch, B., Hahne, A., Glass, E., von Suchodoletz, W., & Friederici, A. D. (2006). Auditory language comprehension in children with developmental dyslexia: Evidence from event-related brain potentials. *Journal of Cognitive Neuroscience*, 18, 1676-1695.
- Santos, A., Joly-Pottuz, B., Moreno, S., Habib, M., & Besson, M. (2007). Behavioural and event-related potentials evidence for pitch discrimination deficits in dyslexic children: Improvement after intensive phonic intervention. *Neuropsychologia*, 45, 1080-1090.
- Scarborough, H. S. (1990). Very early language deficits in dyslexic children. *Child Development*, 61, 1728-1743.

- Schatschneider, C., Carlson, C. D., Francis, D. J., Foorman, B. R., & Fletcher, J. M. (2002). Relationship of rapid automatized naming and phonological awareness in early reading development: Implications for the double-deficit hypothesis. *Journal of Learning Disabilities*, 35, 245-256.
- Schatschneider, C., & Torgesen, J. K. (2004). Using our current understanding of dyslexia to support early identification and intervention. *Journal of Child Neurology*, *19*, 759-765.
- Schneider, W., Roth, E., & Ennemoser, M. (2000). Training phonological skills and letter knowledge in children at risk for dyslexia: A comparison of three kindergarten intervention programs. *Journal of Educational Psychology*, 92, 284-295.
- Schulte-Korne, G., Bartling, J., Deimel, W., & Remschmidt, H. (2004a). Spatial-frequencyand contrast-dependent visible persistence and reading disorder: No evidence for a basic perceptual deficit. *Journal of Neural Transmission*, *111*, 941-950.
- Schulte-Korne, G., Deimel, W., Bartling, J., & Remschmidt, H. (2004b). Neurophysiological correlates of word recognition in dyslexia. *Journal of Neural Transmission*, 111, 971-984.
- Schulte-Korne, G., Deimel, W., & Remschmidt, H. (1997). The importance of phonological decoding and orthographical knowledge for spelling ability in adults. *Zeitschrift Fur Klinische Psychologie-Forschung Und Praxis, 26*, 210-217.
- Schulte-Korne, G., Deimel, W., & Remschmidt, H. (2001). On the diagnosis of dyslexia. Zeitschrift Fur Kinder Und Jugendpsychiatrie Und Psychotherapie, 29, 113-116.
- Schwartz, M. F., Saffran, E. M., & Marin, O. S. M. (1980). The word order problem in agrammatism 1. Comprehension. *Brain and Language*, 10, 249-262.
- Scientific Learning Cooperation. (1996). *Fastforword*. Berkley, CA: Scientific Learning Cooperation.
- Seidenberg, M. S. (2005). Connectionist models of word reading. *Current Directions in Psychological Science*, 14, 238-242.
- Seidenberg, M. S., & McClelland, J. L. (1989). A distributed, developmental model of word recognition and naming. *Psychological Review*, 96, 523-568.
- Seki, A., Koeda, T., Sugihara, S., Kamba, M., Hirata, Y., Ogawa, T., et al. (2001). A functional magnetic resonance imaging study during sentence reading in Japanese dyslexic children. *Brain & Development*, 23, 312-316.
- Shallice, T., Warrington, F. K., & McCarthy, R. (1983). Reading without semantics. *Quarterly Journal of Experimental Psychology*, 35, 111-138.
- Shappell, S. A., Pritchard, W. S., Brandt, M. E., & Barratt, E. S. (1986). The specificity of N400 to semantic mismatches. *Psychophysiology*, 23, 460-460.
- Share, D. L. (1995). Phonological recoding and self-teaching: Sine qua non of reading acquisition. *Cognition*, 55, 151-218.

- Share, D. L. (1999). Phonological recoding and orthographic learning: A direct test of the self-teaching hypothesis. *Journal of Experimental Child Psychology*, 72, 95-129.
- Share, D. L., Jorm, A. F., MacLean, R., & Matthews, R. (2002). Temporal processing and reading disability. *Reading and Writing*, 15, 151-178.
- Shastry, B. S. (2007). Developmental dyslexia: An update. *Journal of Human Genetics*, 52, 104-109.
- Shaywitz, B. A., Shaywitz, S. E., Blachman, B. A., Pugh, K. R., Fulbright, R. K., Skudlarski,
  P., et al. (2004). Development of left occipitotemporal systems for skilled reading in
  children after a phonologically-based intervention. *Biological Psychiatry*, 55, 926-933.
- Shaywitz, B. A., Shaywitz, S. E., Pugh, K. R., Mencl, W. E., Fulbright, R. K., Skudlarski, P., et al. (2002). Disruption of posterior brain systems for reading in children with developmental dyslexia. *Biological Psychiatry*, *52*, 101-110.
- Shaywitz, S. E., Morris, R., and Shaywitz, B.A. (2008). The education of dyslexic children from childhood to young adulthood. *Annual Review of Psychology*, 59, 451-475
- Shaywitz, S. E., Escobar, M. D., Shaywitz, B. A., Fletcher, J. M., & Makuch, R. (1992). Evidence that dyslexia may represent the lower tail of a normal distribution of reading ability. *New England Journal of Medicine*, 326, 145-150.
- Shaywitz, S. E., Fletcher, J. M., & Shaywitz, B. A. (1994). Issues in the definition and classification of attention-deficit disorder. *Topics in Language Disorders*, 14, 1-25.
- Shaywitz, S. E., & Shaywitz, B. A. (2003). Dyslexia (specific reading disability). *Pediatrics in Review*, 24, 147-153.
- Shaywitz, S. E., & Shaywitz, B. A. (2005). Dyslexia (specific reading disability). *Biological Psychiatry.*, 57, 1301-1309.
- Shaywitz, S. E., Shaywitz, B. A., Fletcher, J. M., & Escobar, M. D. (1990). Prevalence of reading disability in boys and girls: Results of the Connecticut longitudinal study. *Journal of the American Medical Association*, 264, 998-1002.
- Shaywitz, S. E., Shaywitz, B. A., Fulbright, R. K., Skudlarski, P., Mencl, W. E., Constable,
  R. T., et al. (2003). Neural systems for compensation and persistence: Young adult outcome of childhood reading disability. *Biological Psychiatry*, 54, 25-33.
- Shaywitz, S. E., Shaywitz, B. A., Pugh, K. R., Fulbright, R. K., Constable, R. T., Mencl, W. E., et al. (1998). Functional disruption in the organization of the brain for reading in dyslexia. *Proceedings of the National Academy of Sciences of the United States of America*, 95, 2636-2641.
- Shibasaki, H., & Miyazaki, M. (1992). Event-related potential studies in infants and children. Journal of Clinical Neurophysiology, 9, 408-418.
- Simon, G. G., Bernard, C., Largy, P., Lalonde, R., & Rebai, M. (2004). Chronometry of visual word recognition during passive and lexical decision tasks: An ERP investigation. *International Journal of Neuroscience*, 114, 1401-1432.

- Simos, P. G., Breier, J. I., Fletcher, J. M., Foorman, B. R., Bergman, E., Fishbeck, K., et al. (2000). Brain activation profiles in dyslexic children during non-word reading: A magnetic source imaging study. *Neuroscience Letters*, 290, 61-65.
- Simos, P. G., Fletcher, J. M., Bergman, E., Breier, J. I., Foorman, B. R., Castillo, E. M., et al. (2002). Dyslexia-specific brain activation profile becomes normal following successful remedial training. *Neurology*, 58, 1203-1213.
- Snow, C., Burns, S., & Griffin, P. (1998). Preventing reading difficulties in young children.Washington DC: National Academic Press.
- Snowling, M., Stackhouse, J., & Rack, J. (1986). Phonological dyslexia and dysgraphia: A developmental analysis. *Cognitive Neuropsychology*, 3, 309-339.
- Snowling, M. J. (1995). Phonological processing and developmental dyslexia. *Journal of Research in Reading*, 18, 132-138.
- Snowling, M. J. (2000). Dyslexia. UK, Oxford: Blackwell Publishing.
- Snowling, M. J., & Hulme, C. (2005). *The science of reading: A handbook*. UK, Oxford: Blackwell Publishing.
- Snowling, M. J., & Hulme, C. (2003). A critique of claims from Reynolds, Nicolson & Hambly (2003) that DDAT is an effective treatment for children with reading difficulties: 'Lies, damned lies and (inappropriate) statistics?' *Dyslexia*, 9, 127-133.
- Sperling, A. J., Lu, Z. L., Manis, F. R., & Seidenberg, M. S. (2003). Selective magnocellular deficits in dyslexia: A "Phantom contour" Study. *Neuropsychologia*, 41, 1422-1429.
- Stanovich, K. E. (1988a). The right and wrong places to look for the cognitive locus of reading disability. *Annals of Dyslexia*, *38*, 154-177.
- Stanovich, K. E. (1988b). Explaining the differences between the dyslexic and the gardenvariety poor reader: The phonological-core variable-difference model. *Journal of Learning Disabilities*, 21, 590.
- Stanovich, K. E. (1992). Speculation on the causes and consequences of individual differences in early reading acquisition In P. Gough, L. Ehri & R. Treiman (Eds.), *Reading acquisition* (pp. 307-342). Hillsdale, NJ Laurence: Erlbaum.
- Stanovich, K. E., Siegel, L. S., & Gottardo, A. (1997). Converging evidence for phonological and surface subtypes of reading disability. *Journal of Educational Psychology*, 89, 114-127.
- Stein, J., & Talcott, J. (1999). Impaired neuronal timing in developmental dyslexia: The magnocellular hypothesis. *Dyslexia*, *5*, 59-77.
- Stein, J. (2001). The sensory basis of reading problems. *Developmental Neuropsychology*, 20, 509-534.
- Stein, J. (2003). Evaluation of an exercise based treatment for children with reading difficulties. *Dyslexia*, *9*, 124-126.
- Stein, J., & Fowler, S. (1981). Visual dyslexia. Trends in Neurosciences, 4, 77-80.

- Stein, J., Talcott, J., & Walsh, V. (2000a). Controversy about the visual magnocellular deficit in developmental dyslexics. *Trends in Cognitive Sciences*, *4*, 209-211.
- Stein, J. F., Richardson, A. J., & Fowler, M. S. (2000b). Monocular occlusion can improve binocular control and reading in dyslexics. *Brain*, 123, 164-170.
- Stelmack, R. M., & Miles, J. (1990). The effect of picture priming on event-related potentials of normal and disabled readers during a word recognition memory task. *Journal of Clinical and Experimental Neuropsychology*, 12, 887-903.
- Stelmack, R. M., Saxe, B. J., Noldycullum, N., Campbell, K. B., & Armitage, R. (1988). Recognition memory for words and event-related potentials: A comparison of normal and disabled readers. *Journal of Clinical and Experimental Neuropsychology*, 10, 185-200.
- Stuart, M., & Coltheart, M. (1988). Does reading develop in a sequence of stages? *Cognition*, 30, 139-181.
- Studdert-Kennedy, M., & Mody, M. (1995). Auditory temporal perception deficits in the reading-impaired: A critical review of the evidence. *Psychonomic Bulletin & Review*, 2, 508-514.
- Sundheim, S., & Voeller, K. K. S. (2004). Psychiatric implications of language disorders and learning disabilities: Risks and management. *Journal of Child Neurology*, *19*, 814-826.
- Swanson, H. L., & Hoskyn, M. (1998). Experimental intervention research on students with learning disabilities: A meta-analysis of treatment outcomes. *Review of Educational Research*, 68, 277-321.
- Talcott, J. B., Hansen, P. C., Willis-Owen, C., McKinnell, I. W., Richardson, A. J., & Stein, J. F. (1998). Visual magnocellular impairment in adult developmental dyslexics. *Neuro-Ophthalmology*, 20, 187-201.
- Tallal, P. (1980). Auditory temporal perception, phonics, and reading disabilities in children. Brain and Language, 9, 182-198.
- Tarkiainen, A., Helenius, P., Hansen, P. C., Cornelissen, P. L., & Salmelin, R. (1999). Dynamics of letter string perception in the human occipitotemporal cortex. *Brain*, 122, 2119-2131.
- Taylor, M. J., & Keenan, N. K. (1990). Event-related potentials to visual and language stimuli in normal and dyslexic-children. *Psychophysiology*, 27, 318-327.
- Temple, C. M. (1985). Reading with partial phonology: Developmental phonological dyslexia. *Journal of Psycholinguistic Research*, 14, 523-541.
- Temple, C. M. (1997). Reading disorders. In C. M. Temple (Ed.), Developmental clinical neuropsychology (pp. 163-223). East Sussex, UK: Psychology Press.

- Temple, E., Deutsch, G. K., Poldrack, R. A., Miller, S. L., Tallal, P., Merzenich, M. M., et al. (2003). Neural deficits in children with dyslexia ameliorated by behavioral remediation: Evidence from functional MRI. *Proceedings of the National Academy of Sciences of the* United States of America, 100, 2860-2865.
- Temple, E., Poldrack, R. A., Protopapas, A., Nagarajan, S., Salz, T., Tallal, P., et al. (2000). Disruption of the neural response to rapid acoustic stimuli in dyslexia: Evidence from functional MRI. Proceedings of the National Academy of Sciences of the United States of America, 97, 13907-13912.
- Tijms, J., & Hoeks, J. (2005). A computerized treatment of dyslexia: Benefits from treating lexico-phonological processing problems. *Dyslexia*, 11, 22-40.
- Timmann, D., & Daum, I. (2007). Cerebellar contributions to cognitive functions: A progress report after two decades of research. *Cerebellum*, *6*, 159-162.
- Torgesen, J., Alexander, A., Wagner, R., Rashotte, C., Voeller, K., & Conway, T. (2001). Intensive remedial instruction for children with severe reading disabilities: Immediate and long-term outcomes from two instructional approaches. *Journal of Learning Disabilities 34*, 33-58.
- Torgesen, J. K., Rashotte, C. A., & Alexander, A. (2003). Progress towards understanding the instructional conditions necessary for remediating reading difficulties in older children. In B. R. Foorman (Ed.), *Preventing and remediating reading difficulties: Bringing science to scale* (pp. 275-298). Timonium, MD: York Press.
- Torgesen, J. K., Wagner, A. D., & Rashotte, C. A. (1997a). Prevention and remediation of severe reading disabilities: Keeping the end in mind. *Scientific Studies of Reading*, 1, 217-234.
- Torgesen, J. K., Wagner, R. K., Rashotte, C. A., Burgess, S., & Hecht, S. (1997b).
  Contributions of phonological awareness and rapid automatic naming ability to the growth of word-reading skills in second-to fifth-grade children. *Scientific Studies of Reading*, 1, 161-185.
- Torgesen, J. K., Wagner, R. K., Rashotte, C. A., Rose, E., Lindamood, P., Conway, T., et al. (1999). Preventing reading failure in young children with phonological processing disabilities: Group and individual responses to instruction. *Journal of Educational Psychology*, 91, 579-593.
- Turkeltaub, P. E., Gareau, L., Flowers, D. L., Zeffiro, T. A., & Eden, G. F. (2003).Development of neural mechanisms for reading. *Nature Neuroscience*, 6, 767-773.
- Van den Berg, R. M., & Te Lintelo, H. G. (1977). A.V.1-pakket. Netherlands: K.P.C.: Den Bosch.
- Van Petten, C. (1993). A comparison of lexical and sentence-level context effects in eventrelated potentials. *Language and Cognitive Processes*, *8*, 485-531.

- Van Petten, C. (1995). Words and sentences: Event-related brain potential measures. *Psychophysiology*, 32, 511-525.
- Van Petten, C., & Kutas, M. (1987). Ambiguous words in context: An event-related potential analysis of the time course of meaning activation. *Journal of Memory and Language*, 26, 188-208.
- Van Petten, C., & Kutas, M. (1990). Interactions between sentence context and wordfrequency in event-related brain potentials. *Memory & Cognition*, 18, 380-393.
- Van Petten, C., & Kutas, M. (1991). Influences of semantic and syntactic context on openclass and closed-class words. *Memory & Cognition*, 19, 95-112.
- Vaughn, S., & Fuchs, L. S. (2003). Redefining learning disabilities as inadequate response to instruction: The promise and potential problems. *Learning Disabilities Research & Practice, 18*, 137-146.
- Vellutino, F. R. (1979). Validity of perceptual deficit explanations of reading disability: A reply. *Journal of Learning Disabilities*, *12*, 160-167.
- Vellutino, F. R., & Fletcher, J. M. (2005). Developmental dyslexia. In M. J. Snowling & C. Hulme (Eds.), *The science of reading: A handbook* (pp. 362-379). UK, Oxford: Blackwell Publishing.
- Vellutino, F. R., Scanlon, D. M., Sipay, E. R., Small, S. G., Pratt, A., Chen, R. S., et al. (1996). Cognitive profiles of difficult-to-remediate and readily remediated poor readers: Early intervention as a vehicle for distinguishing between cognitive and experiential deficits as basic causes of specific reading disability. *Journal of Educational Psychology*, 88, 601-638.
- Vellutino, F. R., Scanlon, D. M., & Tanzman, M. S. (1991). Bridging the gap between cognitive and neuropsychological conceptualization of reading disability. *Learning and Individual Differences*, 3, 181-203.
- Voeller, K. S. K. (2004). Dyslexia. Journal of Child Neurology, 19, 740-744.
- von Suchodoletz, W. (2007). Which therapy is effective in children with dyslexia? *Monatsschrift Kinderheilkunde*, 155, 351-356.
- Vukovic, R. K., & Siegel, L. S. (2006). The double-deficit hypothesis: A comprehensive analysis of the evidence. *Journal of Learning Disabilities*, *39*, 25-47.
- Wagner, R. K., & Torgesen, J. K. (1987). The nature of phonological processing and its causal role in the acquisition of reading skills. *Psychological Bulletin*, 101, 192-212.
- Wanzek, J., Vaughn, S., Wexler, J., Swanson, E. A., Edmonds, M., & Kim, A. H. (2006). A synthesis of spelling and reading interventions and their effects on the spelling outcomes of students with LD. *Journal of Learning Disabilities*, 39, 528-543.
- Watson, B. U. (1992). Auditory temporal acuity in normally achieving and learning-disabled college students. *Journal of Speech and Hearing Research*, 35, 148-156.
- Westwood, P. S. (2002). *Reading and learning difficulties: Approaches to teaching and assessment*. Australia: Australian Council for Education Research ACER.
- Wigfield, A., & Guthrie, J. T. (1997). Relations of children's motivation for reading to the amount and breadth of their reading. *Journal of Educational Psychology*, 89, 420-432.
- Wigfield, A., Guthrie, J. T., Tonks, S., & Perencevich, K. C. (2004). Children's motivation for reading: Domain specificity and instructional influences. *Journal of Educational Research*, 97, 299-309.
- Wilkinson, G. S. (2006). *Wide range achievement test 4 (WRAT- 4)*. London, UK: Harcourt Assessment.
- Williams, M., Lecluyse, K., & Rock-Faucheux, A. (1992). Effective interventions for reading disability. *Journal of the American Optometric Association* 63, 411-417.
- Williams, M. J., Stuart, G. W., Castles, A., & McAnally, K. I. (2003). Contrast sensitivity in subgroups of developmental dyslexia. *Vision Research*, 43, 467-477.
- Witton, C., Talcott, J. B., Hansen, P. C., Richardson, A. J., Griffiths, T. D., Rees, A., et al. (1998). Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexic and normal readers. *Current Biology*, 8, 791-797.
- Wolf, M. (1999). What time may tell: Towards a new conceptualization of developmental dyslexia. *Annals of Dyslexia*, 49, 3-28.
- Wolf, M., Bally, H., & Morris, R. (1986). Automaticity, retrieval processes, and reading: A longitudinal study in average and impaired readers. *Child Development*, 57, 988-1000.
- Wolf, M., & Bowers, P. G. (2000). Naming-speed processes and developmental reading disabilities: An introduction to the special issue on the double-deficit hypothesis. *Journal of Learning Disabilities*, 33, 322-324.
- Wolf, M., Bowers, P. G., & Biddle, K. (2000). Naming-speed processes, timing, and reading: A conceptual review. *Journal of Learning Disabilities*, 33, 387-407.
- Wolf, M., Miller, L., & Donnelly, K. (2000). Retrieval, automaticity, vocabulary elaboration, orthography (RAVE-O): A comprehensive, fluency-based reading intervention program. *Journal of Learning Disabilities 33*, 375-386.
- Wolf, M., & Segal, D. (1999). Retrieval rate, accuracy and vocabulary evlaboration (rave) in reading-impaired children: A pilot intervention programme. *Dyslexia*, *5*, 1-27.
- Woodcock, R. W. (1987). *The woodcok reading mastery test- revised*. USA: American Guidance Service, Inc.
- Worthy, J., Patterson, E., Salas, R., Prater, S., & Turner, M. (2002). "More than just reading": The human factor in reaching resistant readers. *Reading Research and Instruction*, 41, 177-201.
- Zeffiro, T., & Eden, G. (2000). The neural basis of developmental dyslexia. *Annals of Dyslexia*, 50, 3-30.

Ziegler, J. C., Besson, M., Jacobs, A. M., Nazir, T. A., & Carr, T. H. (1997). Word, pseudoword, and nonword processing: A multitask comparison using event-related brain potentials. *Journal of Cognitive Neuroscience*, 9, 758-775.

Zuma. (2002). Zuma deluxe. Retrieved 01/02, 2008, from http://www.realarcade.com

## **APPENDIX A: MEDICAL QUESTIONNAIRE**

## **Child Medical History Questionnaire**

Participant's ID..... Phase: Pre Post Follow-up

Date: ...../..../.....

Child's name.....

This questionnaire asks some questions about your child's health.

| i his questionnaire asks some questions about your child's health.   |  |  |  |  |
|--|--|--|--|--|
| Handedness child: Right Left   |  |  |  |  |
| 1. Is your child currently suffering from  |  |  |  |  |
| <ul> <li>□ Anxiety</li> <li>□ Depression</li> <li>□ Schizophrenia</li> </ul>   |  |  |  |  |
| 2. Does your child have any serious physical condition?<br>If yes, please describe:  |  |  |  |  |
| 3. Is your child currently taking any prescription medication? Yes No<br>If yes, please give details of the medication:                |  |  |  |  |
| 4. Has your child in the past taken any medications for psychological conditions?<br>Yes No  |  |  |  |  |
| If yes, please give details of the medications:  |  |  |  |  |
| 5. Does your child have any difficulties with vision (e.g. blurred vision, watery eyes, bothered by glare)? $\square$ Yes $\square$ No |  |  |  |  |
| If yes, has the condition been diagnosed medically? $\Box$ Yes $\Box$ No   |  |  |  |  |
| If yes, are these difficulties corrected? $\square$ Yes $\square$ No   |  |  |  |  |
| 6. Does your child have any difficulties with hearing?   |  |  |  |  |

| If yes, has the condition been diagnosed medically? $\square$ Yes $\square$ No   |        |  |  |
|--|--------|--|--|
| If yes, are these difficulties corrected? $\Box$ Yes $\Box$ No.  |        |  |  |
| 7. Did the mother of the child have any difficulties   |        |  |  |
| a) during pregnancy? T Yes T No<br>If yes, please describe:  |        |  |  |
| b) while giving birth? Yes No<br>If yes, please describe:  |        |  |  |
|  | •••••• |  |  |
| 8. Has your child had any of the following? (Please tick)  |        |  |  |
| <ul> <li>8. Has your child had any of the following? (Please tick)</li> <li>Middle ear infections ('glue ear')</li> <li>Fits or convulsions</li> </ul>   |        |  |  |
| <ul> <li>8. Has your child had any of the following? (Please tick)</li> <li>Middle ear infections ('glue ear')</li> <li>Fits or convulsions</li> <li>Epilepsy</li> </ul>   |        |  |  |
| <ul> <li>8. Has your child had any of the following? (Please tick)</li> <li>Middle ear infections ('glue ear')</li> <li>Fits or convulsions</li> <li>Epilepsy</li> <li>Giddiness</li> </ul>  |        |  |  |
| <ul> <li>8. Has your child had any of the following? (Please tick)</li> <li>Middle ear infections ('glue ear')</li> <li>Fits or convulsions</li> <li>Epilepsy</li> <li>Giddiness</li> <li>Concussion</li> </ul>  |        |  |  |
| <ul> <li>8. Has your child had any of the following? (Please tick)</li> <li>Middle ear infections ('glue ear')</li> <li>Fits or convulsions</li> <li>Epilepsy</li> <li>Giddiness</li> <li>Concussion</li> <li>Headaches</li> </ul>                             |        |  |  |
| <ul> <li>8. Has your child had any of the following? (Please tick)</li> <li>Middle ear infections ('glue ear')</li> <li>Fits or convulsions</li> <li>Epilepsy</li> <li>Giddiness</li> <li>Concussion</li> <li>Headaches</li> <li>Severe head injury</li> </ul> |        |  |  |

9. If you answered yes to any of the above, please describe giving the age of the child at the time this occurred:

.....

# **APPENDIX B: PARENTS' QUESTIONNAIRE**

| Personal details: parents         |                          |                 |
|-----------------------------------|--------------------------|-----------------|
| Form filled out by: father / male | ;uardian 🗖 mother / f    | female guardian |
| Your name:                        |                          |                 |
| Age father / male guardian: A     | ge mother / female guard | lian:           |
| Address                           |                          |                 |
| Email and/or                      | ••••••••••••••••••       |                 |
| phone number                      |                          |                 |
| l                                 | arents' Questionnaire    |                 |

Participant's ID..... Phase: Pre

Date: ...../..../.....

Child's name.....

## PART 1 Personal details: father

Please indicate your marital status

| П | married/de facto |
|---|------------------|
|   | single           |
| П | divorced         |
|   | widowed          |

## Please indicate the father / male guardian's employment status

| Г | full-time                |
|---|--------------------------|
| Ē | part-time                |
|   | casual work              |
|   | retired                  |
| D | not employed/home duties |
|   | disabled                 |
| Ð | other (please specify):  |

## Please indicate the father / male guardian's educational level

| <u>,</u> | year 11 or less   |
|----------|---|
|          | year 12 or equivalent   |
|          | apprenticeship, technical trades certificate, special training        |
|          | diploma or associate diploma (e.g. in nursing, accountancy, teaching) |
| F        | bachelor degree   |
|          | postgraduate degree/graduate diploma                                  |

# PART 2 Personal details: mother

Please indicate your marital status

- married/de facto
- □ single
- divorced
- widowed

Please indicate the mother / female guardian's employment status

|          | full-time                |
|----------|--------------------------|
|          | part-time                |
|          | casual work              |
|          | retired                  |
| <u>.</u> | not employed/home duties |
|          | disabled                 |
| Ε        | other (please specify):  |
|          |                          |

Please indicate the mother / female guardian's educational level

|        | year 11 or less   |
|--------|---|
| L<br>L | year 12 or equivalent   |
|        | apprenticeship, technical trades certificate, special training        |
| F.1    | diploma or associate diploma (e.g. in nursing, accountancy, teaching) |
|        | bachelor degree   |
|        | postgraduate degree/graduate diploma                                  |

Please estimate your average household income (before tax) per week

(Sum of all individual incomes in the household, for individuals aged 15 years and

over).....

ie indicate your family situation child/ren live/s in a two-parent family

|     | child/ren live/s in a one-parent family              |
|-----|--|
|     | child/ren live/s with one parent and one step-parent |
| How | many people are living in your household?            |
| How | many bedrooms are there in your house?               |

(Please note for Part 3, 4 and 5 "Child" refers to your child: the child who is participating in the study)

| PART 3 Your child's educational history          |
|--|
| Current Grade School's name                      |
| Please list the schools your child has attended: |
| Grade to Grade School's name:                    |
| Grade to Grade School's name:                    |
| Grade to Grade School's name:                    |
|  |

Has your child had any long absences (a month or more) from school (due to a medical condition, moving etc.)? Please add up the months if more than one long absence.

No Yes\_\_\_\_month(s).

If Yes, during what Grade/s?.....

### PART 4 Your child's reading and developmental history

Did your child meet all the milestones for language development at the appropriate ages?

| Use of single words as names of things or<br>actions by the age of 12 months | П  | Yes | Π | No F | Don't remember |
|--|----|-----|---|------|----------------|
| Talks clearly in two-three word sentences by the age of two                  |    | Yes |   | No 🗖 | Don't remember |
| Follows a series of three simple instructions by the age of four             | E. | Yes | Γ | No 🗖 | Don't remember |
| Reads a few letters by the age of five                                       |    | Yes | Π | No 🗖 | Don't remember |

Do the father and/or the mother have any difficulties with reading?

|   | No                   | Yes, the mother only        |
|---|----------------------|-----------------------------|
| Γ | Yes, the father only | Yes, both mother and father |

Is there a history of learning/reading difficulties or dyslexia in either parent's families?

| □ <sub>No</sub> | <b>L</b> , | Yes, the mother's family only |
|-----------------|------------|-------------------------------|
|-----------------|------------|-------------------------------|

Yes, the father's family only Yes, both families

Has your child ever been assessed for learning/reading difficulties?

| If yes, was there a diagnosis?  Yes  No.  |
|---|
| If yes, please describe the diagnosis:  |
|   |
| Has your child ever been assessed for speech and oral language problems?<br>Yes No  |
| If yes, please describe the diagnosis:  |
|   |
| Has your child ever been assessed for Attention Deficit Hyperactivity Disorder (ADHD)?  |
| Yes No  |
| If yes, please describe the diagnosis:  |
|   |
| Has your child ever been treated for dyslexia or specific learning needs (including special lessons in school) in the past?   |
| Yes E No. Please specify:   |
|   |
| PART 5 Please give details on your family's reading environment   |
|   |
| About how many newspapers or magazines does your family get regularly (on a weekly, fortnightly or  |
| monthly basis?  |
| never   |
| Importing basis?     Import basis?       Import basis? <t< td=""></t<>  |
| Importing basis?     Importing basis?       Impo  |
| Image: provide basis?     Image: provide basis?   |
| Importing basis?   |
| Importing basis?       Importing basis?         Importing basis?  |
| Importing basis?   |
| Imported basis?   |
| Importing basis?   |
| Do you' basis)?         Image: provide basis   |
| Importing Basis?         Importing Basis and Basis  |
| Importing basis)?         Importing basis)? <t< td=""></t<>   |
| Importing basis/2       Importing basis/2   |
| Provide Plasticity         Image: Provide Plasticity         Image: Plasticity  |
| Provide Basis/2012       Unit of read a book for preasure?         Image: Provide Basis/2012       Image: Provide Basis/2012  |
| Provide Beausyles your child to read a book for pleasure?         Image: Image |
| Howking beging ge your child to read a book for pleasure?         Image: Image |

How often do you read a book for pleasure?

| Û | once a year or less       |
|---|---------------------------|
|   | once a week or less       |
|   | two to three times a week |
|   | daily                     |

Do you enjoy reading?

yes sometimes

no

Would you describe yourself as a good reader?

□ yes

no

Thank you for your time!

# APPENDIX C: WORD STIMULI PRESENTED FOR THE PHONOLOGICAL AND

## LEXICAL TASKS

# Phonological Task

| Pse        | udo homor | hones |        | Non | words   |    |        |
|------------|-----------|-------|--------|-----|---------|----|--------|
| 1          | whilde    | 21    | whercs | 1   | whilge  | 21 | whervs |
| 2          | ceetes    | 22    | mighn  | 2   | ceeths  | 22 | mighbs |
| 3          | skaile    | 23    | strete | 3   | skaims  | 23 | screte |
| 4          | kought    | 24    | ghetts | 4   | zought  | 24 | ghetch |
| 5          | farste    | 25    | whaugh | 5   | narste  | 25 | cwaugh |
| 6          | klarse    | 26    | gnawth | 6   | klarbs  | 26 | gnawls |
| 7          | dighed    | 27    | smourl | 7   | dights  | 27 | smourp |
| 8          | treign    | 28    | pseame | 8   | treives | 28 | pseafe |
| 9          | whirse    | 29    | werled | 9   | whirps  | 29 | gwerls |
| 10         | kloazz    | 30    | phound | 10  | kloabz  | 30 | phounn |
| 11         | paidge    | 31    | whunse | 11  | paides  | 31 | whunge |
| 12         | torked    | 32    | koarld | 12  | jorked  | 32 | koarve |
| 13         | poaced    | 33    | skoole | 13  | spoace  | 33 | skoode |
| 14         | naimbs    | 34    | ghrait | 14  | naimth  | 34 | ghraib |
| 15         | phaice    | 35    | rowned | 15  | phaiph  | 35 | rownse |
| 16         | senned    | 36    | taique | 16  | sennth  | 36 | gaique |
| 17         | phawm     | 37    | pherst | 17  | phawch  | 37 | pherse |
| 18         | shautt    | 38    | rheedd | 18  | shaugg  | 38 | rheend |
| 1 <b>9</b> | shoart    | 39    | whonte | 19  | shoarf  | 39 | whonce |
| 20         | fealed    | 40    | coarce | 20  | fealts  | 40 | woarce |

Lexical Task

| Rea        | al Words |    |        | Pseudo homophones |        |    |        |
|------------|----------|----|--------|-------------------|--------|----|--------|
| 1          | prayed   | 21 | skills | 1                 | praide | 21 | sckils |
| 2          | floors   | 22 | glance | 2                 | florze | 22 | glanse |
| 3          | stones   | 23 | fought | 3                 | stoans | 23 | faught |
| 4          | scored   | 24 | rolled | 4                 | skoard | 24 | roalde |
| 5          | slowed   | 25 | boards | 5                 | sloade | 25 | bordes |
| 6          | earned   | 26 | screen | 6                 | irgned | 26 | screan |
| 7          | curves   | 27 | sports | 7                 | kerves | 27 | spauts |
| 8          | guards   | 28 | courts | 8                 | ghards | 28 | cortes |
| 9          | grains   | 29 | taught | 9                 | granes | 29 | tourte |
| 10         | scared   | 30 | choose | 10                | scaird | 30 | chooze |
| 11         | wheels   | 31 | troops | 11                | weeles | 31 | treups |
| 12         | warned   | 32 | wished | 12                | wawned | 32 | wyshed |
| 13         | hearts   | 33 | sought | 13                | hartes | 33 | sourte |
| 14         | shapes   | 34 | please | 14                | shaips | 34 | pleazz |
| 15         | scenes   | 35 | search | 15                | seenes | 35 | sertch |
| 16         | shorts   | 36 | claims | 16                | shawts | 36 | klaims |
| 17         | nights   | 37 | rights | 17                | knytes | 37 | rhytes |
| 1 <b>8</b> | scheme   | 38 | forced | 18                | sckeem | 38 | fauced |
| 19         | phrase   | 39 | caused | 19                | fraizz | 39 | corzed |
| 20         | stores   | 40 | bridge | 20                | storze | 40 | brydge |

# APPENDIX D: SENTENCES PRESENTED FOR THE SENTENCE TASK

|    | Congruent Sentences                         |    |                                       |
|----|---|----|---------------------------------------|
| 1  | You can leave the door open.                | 26 | You and I can run very fast.          |
| 2  | The prisoner feels lonely in his cell.      | 27 | Wake up and open your eyes.           |
| 3  | She is upstairs and he is downstairs.       | 28 | Sit down I'll tell you a story.       |
| 4  | I have planted flowers in my garden.        | 29 | Cars cost a lot of money.             |
| 5  | The colour of grass is green.               | 30 | Every morning Dad goes to work.       |
| 6  | English is not her first language.          | 31 | Winter is cold and summer is hot.     |
| 7  | Have some more there is plenty.             | 32 | Peas and carrots are both vegetables. |
| 8  | She never takes a day off.                  | 33 | She cooked it on the stove.           |
| 9  | He was tired so he slept.                   | 34 | That lucky boy won first prize.       |
| 10 | After wiping, the floor is clean.           | 35 | Babies drink milk from a bottle.      |
| 11 | An electric guitar is a musical instrument. | 36 | Our hands have exactly ten fingers.   |
| 12 | Football is a very popular sport.           | 37 | Bags of rocks are very heavy.         |
| 13 | He buys dog food for his dog.               | 38 | The sea is also called the ocean.     |
| 14 | Last night I had a terrible dream.          | 39 | Snails and turtles move very slowly.  |
| 15 | The beach has very white sand.              | 40 | Run round and round in a circle.      |
| 16 | Every morning the children go to school.    | 41 | The sun went behind a cloud.          |
| 17 | Sesame Street is my favourite TV show.      | 42 | He died of a heart attack.            |
| 18 | Apples grow on an apple tree.               | 43 | We have to wear a school uniform.     |
| 19 | Dad likes to drive his car.                 | 44 | The bad boy stuck out his tongue.     |
| 20 | At night I sleep in my bed.                 | 45 | The man had a long grey beard.        |
| 21 | John likes to read his new book.            |    |                                       |
| 22 | She waited at the bus stop.                 |    |                                       |
| 23 | The girls went outside to play.             |    |                                       |
| 24 | The stars come out at night.                |    |                                       |

,

25 She's not a boy, she's a girl.

# **Incongruent Sentences**

|            | 8   |
|------------|---|
| 1          | You can leave the door fast.              |
| 2          | The prisoner feels lonely in his money.   |
| 3          | She is upstairs and he is off.            |
| 4          | I have planted flowers in my stove.       |
| 5          | The colour of grass is slow.              |
| 6          | English is not her first dog.             |
| 7          | Have some more, there is garden.          |
| 8          | She never takes a day plenty.             |
| 9          | He was tired so he pulled.                |
| 10         | After wiping, the floor is green.         |
| 11         | An electric guitar is a musical girl.     |
| 12         | Football is a very popular language.      |
| 13         | He buys dog food for his story.           |
| 14         | Last night I had a terrible ocean.        |
| 15         | The beach has very white instruments.     |
| 16         | Every morning the children go to uniform. |
| 17         | Sesame Street is my favourite TV car.     |
| 18         | Apples grow on an apple school.           |
| 1 <b>9</b> | Dad likes to drive his vegetables.        |
| 20         | At night I sleep in my sport.             |
| 21         | John likes to read his new sand.          |
| 22         | She waited at the bus dream.              |
| 23         | The girls went outside to book.           |
| 24         | The stars come out at cell.               |

25 She's not a boy, she's a work.

- 26 You and I can run very clean.
- 27 Wake up and open your cloud.
- 28 Sit down I'll tell you a tongue.
- 29 Cars cost a lot of trees.
- 30 Every morning Dad goes to bottle.
- 31 Winter is cold and summer is heavy.
- 32 Peas and carrots are both eyes.
- 33 She cooked it on the beard.
- 34 That lucky boy won first fingers.
- 35 Babies drink milk from a show.
- 36 Our hands have exactly ten attacks.
- 37 Bags of rocks are very hot.
- 38 The sea is also called the play.
- 39 Snails and turtles move very open.
- 40 Run round and round in a stop.
- 41 The sun went behind a bed.
- 42 He died of a heart night.
- 43 We have to wear a school stairs.
- 44 The bad boy stuck out his circle.
- 45 The man had a long grey prize.

# **APPENDIX E: MONITOR SHEET FOR HOME READING PRACTICE**

# UTAS Cellfield research 2007: Reading and spelling practice record

1.1

We would like you to practice reading and/or spelling with your child for 5-10 minutes daily

|                              |             |        | ↓ <u> </u>                            |        |  |
|------------------------------|-------------|--------|---------------------------------------|--------|--|
| DATE                         |             |        |                                       |        |  |
|                              |             |        |                                       |        |  |
| <b>1.Type of text for</b>    |             |        |                                       |        |  |
| reading practice             |             |        |                                       |        |  |
| (e g magazine book)          |             |        |                                       |        |  |
|                              |             |        | · · · · · · · · · · · · · · · · · · · |        | ······································ |
| Amount read                  |             |        |                                       |        |  |
| (section/pages)              |             |        |                                       |        |  |
| 2. Spelling practice?        |             |        |                                       |        | E C                                    |
| (please tick)                | Yes No      | Yes No | Yes No                                | Yes No | Yes No <sup></sup>                     |
|                              |             |        |                                       |        |  |
| Amount of new words          |             |        |                                       |        |  |
| spelled correctly            |             |        |                                       |        |  |
| - <b>F</b>                   |             |        |                                       |        |  |
|                              | · · · · · · |        |                                       |        |  |
| 3. Any comments?             |             |        |                                       |        |  |
| (reads better, able to spell |             |        |                                       |        |  |
| a new word etc.)             |             |        |                                       |        |  |
| ,                            |             |        |                                       |        |  |
|                              |             |        |                                       |        |  |
|                              |             |        |                                       |        |  |
|                              |             |        |                                       |        |  |

#### **APPENDIX F: STANDARD INSTRUCTIONS FOR THE ERP TASKS**

#### **Sentence Task**

You will be presented with a series of sentences one word at a time. The sentences are similar to one another in grammatical structure and some might be very familiar to you. Read each of the sentences silently. At the end of each sentenced a slide with XXXXX will appear, followed by a question mark (???) slide. When you see the '???' I want you to decide if the sentence made sense or not by pressing Z (yes it made sense) or X (no it did not make sense). Please use your dominant hand and keep your two fingers close to the buttons. There will be a break after half of the sentences. Try not to make errors, but also try to be quick.

Run through the practice items and ensure that the child understands the task. Then go on to the experimental tasks.

#### **Phonological Decision Task**

Say to the child: "Some words are going to come up on the computer screen one at a time. Neither of the words are real words, but some sound like a real word. If you think the word on the screen sounds like a real word press button Z. If you think it does not sound like a real word press button X. A cross will appear briefly on the screen between the words. You do not need to respond to that. Try not to make errors, but also try to be quick.

Run through the practice items and ensure that the child understands the task. Then go on to the experimental tasks.

#### **Lexical Decision Task**

Say to the child: "Some words are going to come up on the computer screen one at a time. All words sound like a real word but only some of them are real words. If you think the word spells a real word press the button Z. If you think the word on the screen does not spell a real word press button X. A cross will appear briefly on the screen between the words. You do not need to respond to that. Try not to make errors, but also try to be quick.

Run through the practice items and ensure that the child understands the task. Then go on to the experimental tasks.

#### **General Instructions**

Please try to remain as still as you can. Keep your eyes focused on the screen. Do not move your head.

## APPENDIX G: STEM AND LEAF PLOTS FOR LITERACY DATA AT PRE-TEST

.

# Table 10

1

Stem and Leaf Plots for the Literacy Measures for the Cellfield and Placebo Group at Pre-test

|                                    | Cellfield |       | Placebo |
|------------------------------------|-----------|-------|---------|
| Variable name                      | Leaf      | Stem  | Leaf    |
| WRMT-R WI (SS <sup>1</sup> )       | 5         | 4     | 6       |
|                                    | 5         | 5     |         |
|                                    | 447       | 6     |         |
|                                    | 6         | 7     | 588     |
|                                    | 6         | 8     | 4       |
|                                    | _1        | 9     |         |
| WRMT-R WA (SS <sup>1</sup> )       |           | 5     | 1       |
|                                    | 028       | 6     |         |
|                                    | 16        | 7     | 8       |
|                                    | 33        | 8     | 114     |
| WRAT-4 Spelling (SS <sup>1</sup> ) | 2449      | 7     | 0       |
|                                    | 288       | 8     | 2368    |
| Neale Accuracy (RA <sup>2</sup> )  | 9         | 7 – – |         |
|                                    | 58        | 8     | 3       |
|                                    | 7         | 9     | 6       |
|                                    | 112       | 10    | 77      |
|                                    |           | 11    | 2       |
| Neale Comprehension $(RA^2)$       | 47        | 8     | 5       |
|                                    |           | 9     | 5       |
|                                    | 11        | 10    | 4       |
|                                    | 36        | 11    |         |
|                                    | 0         | 12    | 8       |
|                                    |           | 13    | 9       |
| Neale Rate $(RA^2)$                | 35        | 8     |         |
|                                    | 0         | 9     | 24      |
|                                    | 12        | 10    | 4       |
|                                    |           | 11    | 36      |
|                                    | 1         | 12    |         |
|                                    |           | 13    |         |
|                                    | 9         | 14    |         |

1= SS (primary standard score; M=100, SD= 15); 2= RA (reading age in months)

.