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Neurophysiological studies of reading fluency

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Chapter 1
General Introduction

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1.1. Developmental dyslexia: general introduction

Developmental dyslexia, or specific reading difficulty, is a learning disorder that is neurobiological in origin. Traditionally, it is defined as a persistent failure to acquire efficient reading skills, which is not the direct result of sensory impairments, intellectual deficits or lack of educational opportunities (Lyon, Shaywitz, & Shaywitz, 2003; Word Health Organization, 1993). Dyslexia is one of the most common developmental disorders. It occurs in all known languages, affecting 5 – 12% of school-aged children (Elizabeth, Beach, & Gabrieli, 2014; Peterson & Pennington, 2012). In families with a history of dyslexia, the estimate of prevalence surges to 34 – 66% (Grigorenko, 2001; Puolakanaho et al., 2007; Snowling, Callagher, & Frith, 2003), pointing to a genetic basis for the disorder. Dyslexia affects many different aspects of daily living, reading retardation being merely one of its manifestations. The disorder is a lifelong condition, posing a severe risk to academic attainments, occupational perspectives and psychosomatic well-being.

As noted by Schulte-Körne and Bruder (2010), a poor start with reading acquisition may lead to increasing backwardness in reading, demotivation, and ultimately, functional illiteracy. Therefore, in order to minimize the detrimental effects of dyslexia, it is important to provide dyslexic children with optimal intervention at the youngest possible age. Early diagnosis of the disorder, which is a prerequisite for early intervention, is thus called for. In this dissertation, we aim to reveal early neurophysiological markers of developmental dyslexia that can be detected during the first two years of reading acquisition, a critical time window wherein formal literacy education starts and key cognitive skills supporting fluent reading are developed.

During the past few decades, psycho- and neurolinguistic research, aided by neurophysiological and neuroimaging techniques, has significantly advanced our understanding of how reading is organized from the cognitive to the brain level, and how this complex process could break down in developmental dyslexia. Such knowledge is essential for the diagnosis of dyslexia, and may eventually set forth the theoretical basis for optimizing intervention programs. In this introductory chapter, we first present a dual-route model depicting the cognitive architecture of skilled reading, which provides a conceptual framework for investigating normal and impaired reading acquisition. This directs us to our main lines of research, i.e., abnormalities in auditory/phonological processing and visual word recognition. We then present major theories of cognitive deficits underlying dyslexia in these two domains, and identify their neuroanatomical origins. Finally, we close this introduction with an overview of upcoming chapters in this dissertation.

1.2. The dual-route model of skilled reading

As a hallmark in the evolutionary history of human intelligence, reading is a multi-modular cognitive operation integrating lower-level perceptual analyses and higher-level linguistic computations. Lying at the crux of this complex mechanism is the ability to decipher visual language codes and to relate them to units of spoken language, which, in order to be produced and comprehended, need to be mentally represented and connected to units of meaning (Schlaggar & McCandliss, 2007). This interactive network underpinning reading is among the most extensively studied topics in psycho- and neurolinguistics.

One of the most influential theories investigating the translation of print into speech or meaning is the dual route (DR) model. First proposed by Coltheart (1978), Patterson and Shewell (1987), and Ellis and Young (1998), among others, the DR model contends that reading proceeds along two alternative routes: a sublexical, or indirect route, and a lexical, or direct route. The essential step in reading via the sublexical route involves using “grapheme-phoneme correspondence rules” (GPCs; Coltheart, 1978) to convert letter strings into phonological representations, which can then be used to generate overt speech (i.e., to read aloud). In addition, via this sublexical route the auditory word forms being addressed can be connected to corresponding items in the semantic lexicon, in a similar way to spoken word recognition. The sublexical route works well for reading words with regular spelling-sound correspondences (e.g., “mint”, “tint”, and “hint”, in which the rhyme is pronounced /Int/; examples were taken from Cortese & Balota, 2013), and is necessary for pronouncing novel words and nonwords, i.e., words that sound like normal words but do not correspond to a lexical entry in the phonological or the semantic lexicon (e.g., “plitharize”). The lexical route, on the other hand, makes use of an orthographic input lexicon that directly recognizes whole-word orthographic patterns. The recognized item is then connected to corresponding items in the phonological output lexicon for reading aloud, and in the semantic lexicon to attach meaning. Since reading via the lexical route implicates recognition of patterned orthographic input, the development of this route requires exposure to print: only words that the reader has seen at least once can be decoded this way. Because of its dependency on familiarity, the lexical route cannot process novel words or nonwords; nevertheless, it is particularly needed to pronounce irregular words (e.g., “pint”, in which the rhyme is pronounced in an idiosyncratic way: /aint/).

The DR model has undergone several major modifications over the past few decades. Remarkably, Coltheart and colleagues (Coltheart, Rastle, Perry, & Langdon, 2001) developed the DR model into the dual-route cascaded (DRC) model. In contrast to the original DR model, which assumes serial grapheme-to-phoneme processing in the indirect route, the DRC model postulates bidirectional excitation and inhibition connections between the orthographic, the phonological and the semantic lexicon. This transition was inspired by the notion of interactive activation (IA) from the parallel distributed processing (PDP) model (Harm & Seidenberg, 1999, 2004; Seidenberg & McClelland, 1989). Another major change the notion of IA entails, is that the scope of the direct route is extended beyond whole words to incorporate orthographic patterns smaller than words, such as morphemes, syllables, and letter strings at the sub-syllable level (e.g., syllable-onset clusters and syllable rhymes).

The DRC model has been tested in numerous studies, and gained support from a large body of psycholinguistic data (e.g., Coltheart & Rastle, 1994; Rastle & Coltheart, 1999; Ziegler et al., 2008; Ziegler, Perry, & Coltheart, 2000, 2003). There are, of course, theoretical accounts that take very different approaches to modeling the cognitive architecture of reading, for example, the PDP model (a full description of the PDP model, which is also highly influential, falls beyond the scope of this introduction; for reviews see, e.g. Seidenberg, 2013). However, a broad consensus has been reached, i.e., two routes can be employed to transform print into speech or meaning, one hinging on grapheme-to-phoneme conversion and the other on fast, accurate recognition of visual word forms (Coltheart, 2005). As we will see in the next section, the DRC model provides useful guidelines for research on normal and disordered reading acquisition.

1.3. Cognitive deficits underlying developmental dyslexia and their neuroanatomical origins

Under the framework of the DRC model, fluent reading proceeds in two pathways and implicates the following components and processes: an orthographic lexicon storing the visual form of words, a phonological lexicon storing the sound structure of words, a semantic lexicon storing the meaning of words, and a grapheme-to-phoneme conversion route that translates orthographic representations into their phonological counterparts. The possession of such a dual-route architecture is the end-result of successful reading acquisition. Accordingly, if any of the component(s) has not been acquired to an age-appropriate level, reading will prove abnormal for age, and the way in which the anomaly manifests itself will depend on which component(s) has been affected (Coltheart, 2005). For example, if the orthographic lexicon is developmentally impaired, the child will have particular difficulty reading irregular words, despite relatively preserved reading of words and nonwords; this is the case for *developmental surface dyslexia* (McDougall, Borowsky, MacKinnon, & Hymel, 2005; Valdois, Bosse, & Tainturier, 2004; Ziegler et al., 2008). If the sublexical procedure is poor for age, on the other hand, the child will have a selective deficit in reading nonwords and novel words (real words encountered for first time by the reader are processed in the same way as nonwords, because they do not have a corresponding entry in the orthographic input lexicon yet); this is the case for *developmental phonological dyslexia* (McDougall et al., 2005; Muneaux, Ziegler, Truc, Thomson, & Goswami, 2004; White et al., 2006; Ziegler et al., 2008).

In the current study, we therefore focus on two lines of research to investigate early indicators of developmental dyslexia, i.e., phonological/auditory processing and visual word recognition, the former underpinning the sublexical route of the DRC model and the latter underpinning the lexical route. Indeed, as the DRC model predicts, impairments in these two domains have been shown to play an important role in dyslexia. In the rest of this section, we present major theories of phonological/auditory-processing and visual-word-recognition deficits underlying dyslexia, and discuss their origins in the brain.

1.3.1. Phonological/auditory processing in developmental dyslexia

1.3.1.1. The phonological-core deficit theory

Historically, the aetiology of dyslexia has been sought in the visual, the auditory, and the cognitive-linguistic domains (for a review see, e.g., Vellutino, Fletcher, Snowling, & Scanlon, 2004). After decades of discussion, the consensus has been reached that, though other systems may each contribute to dyslexia to a certain extent, the core difficulty underlying dyslexia is phonological. This is in line with the claims of the DRC model regarding learning to read: as discussed in previous sections, an important task of reading acquisition in the context of the DRC model is to build up a fully functioning sublexical route, which is needed for reading nonwords and novel words (the latter type is particularly important in reading acquisition).

To acquire the sublexical route, which rests heavily on grapheme-to-phoneme conversion, a child must develop phonological awareness, i.e., the awareness that speech is combinatorial in nature, comprising a limited number of sounds (phonemes) that are combined to make words (Lyon et al., 2003; Ramus, et al., 2003). Moreover, the child must realize that written words bear the same internal phonological structure

as the spoken words. It is phonological awareness and the understanding that constituents of written words are systematically related to spoken units that eventually allows the child to connect written words to corresponding items in the phonological lexicon (Pugh et al., 2001). Failure in the acquisition of this sublexical/phonological route may lead to difficulties reading nonwords and newly encountered words, which in turn hinder the development of the orthographic lexicon and subsequently the acquisition of the lexical/orthographic route. This way of looking at the origin of reading difficulty lays the basis for the phonological-core deficit theory. Specifically, supporters of this theory claim that developmental dyslexia arises causally and directly from a cognitive deficit in phonological awareness, which renders the learning of grapheme-to-phoneme conversion rules difficult, hence weakening the very basis of reading alphabetic scripts (Bishop & Snowling, 2004; Lyon et al., 2003; Snowling, 2001; Vellutino et al., 2004; Ziegler & Goswami, 2005).

Support for the phonological theory comes from behavioral findings that children with dyslexia perform poorly on tasks requiring phonological awareness such as nonword reading and phoneme deletion (for reviews see, e.g. Ramus, 2004). Problems as such are believed to reflect impairments in conscious segmentation and manipulation of phonemes. In addition to impoverished phonological awareness, there are other problems associated with phonological processing in dyslexia, which may point to a more basic deficit in the quality of phonological representations, or their access and retrieval (Dandache, Wouters, & Ghesquière, 2014; Ramus et al., 2003): these include poor verbal short-term memory, as exemplified in nonword repetition and digit span task (e.g., Catts, 1989), and slow retrieval of phonological codes from long-term memory, as reflected by the word-finding difficulties frequently observed clinically and in experiments using rapid naming tasks¹ (e.g., Denckla & Cutting, 1999; Vellutino et al., 1996). In addition to findings of deficient phonological processing in individuals with dyslexia, evidence for the phonological-core deficit theory comes from longitudinal prospective studies that have reported a causal link between early sensitivity to the phonological structure of words and later literacy performance (for reviews see, e.g., Castles & Coltheart, 2004).

In short, there is good evidence for a phonological deficit as a primary source of reading difficulty. According to a recent review by Schulte-Körne and Bruder (2010), a phonological deficit is found in 30 – 60% of the dyslexics depending on the study. Indeed, the phonological-core deficit theory has remained the predominant etiological view of dyslexia for decades. However, despite the consensus that dyslexia results largely, if not exclusively, from a phonological deficit, the origin of this deficit is still a matter of debate: is it specific to phonology, as proposed by the phonological core-deficit theory, or is it part of a more general sensorimotor deficit? One of the theories supporting the latter view is the auditory deficit theory, which will be reviewed next.

1.3.1.2. The auditory deficit theory

Challenging the central and causal role of phonology in dyslexia, the rapid auditory temporal processing theory (RATP; Tallal & Piercy, 1973 a, 1973 b) claims that the phonological deficit is secondary to a more fundamental deficit in auditory

¹ Alternatively, the rapid naming deficit is considered as an independent source of reading difficulty (see, e.g., Kirby, Silvestri, Allingham, Parrila, & La Fave, 2008; Wolf & Bowers, 2000; Wolf, Bowers, & Biddle, 2000).

mechanisms. Initially proposed to account for deficient auditory processing in specific language impairment (SLI), the RATP was later extended to explain dyslexia (Farmer & Klein, 1995; McArthur & Bishop, 2001; Stein, 2001; Tallal, 1980; Temple et al., 2000). Specifically, the RATP contends that children with developmental language and literacy disorders have difficulties perceiving transient or rapidly changing sounds. In support of the RATP, it has been shown that individuals with dyslexia have particular difficulties discriminating phonemes involving rapid spectro-temporal transitions (e.g., Mody, Studdert-Kennedy, & Brady, 1997; Schulte-Körne, Deimel, Bartling, & Remschmidt, 1998).

During the past forty years, the temporal demand the RATP initially stipulated has been called into question, and research on deficient auditory processing in dyslexia has been extended to incorporate aspects of auditory analyses that do not tax temporal processing. The auditory deficit among dyslexic readers has been demonstrated in a wide range of tasks, including temporal-order judgment (e.g., Ben-Artzi, Fostick, & Babkoff, 2005; Chung et al., 2008), pitch discrimination (e.g., Baldeweg, Richardson, Watkins, Foale, & Gruzelier, 1999; Kujala, Belitz, Tervaniemi, & Näätänen, 2003), and frequency/amplitude-modulation detection (Menell, McAnally, & Stein, 1999; Talcott et al., 1999, 2000, 2002; van Ingelghem et al., 2005; Witton et al., 1998). In addition to analyses of general acoustic information, the processing of speech signals also proves to be impaired in dyslexic readers, as they exhibit difficulties discriminating between speech sounds contrasting various acoustic features, such as frequency transition (spectral changes, e.g. /da/ vs. /ga/; Kraus et al., 1996; Meng et al., 2005; Schulte-Körne, Deimel, Bartling, & Remschmidt, 2001), and voice-onset timing (temporal changes, e.g. /ba/ vs. /pa/; Cohen-Mimran, 2006; Moisescu-Yiflach & Pratt, 2005).

According to the auditory deficit theory, problems with auditory processing as reviewed above cause a cascade of effects, starting with fuzzy, underspecified phonological representations, leading to disrupted phonological processing and finally bringing on developmental dyslexia (Ben-Artzi et al., 2005; Boets, Wouters, van Wieringen, & Ghesquière, 2007; Chung et al., 2008). Therefore, the auditory deficit theory does not deny the existence of a phonological deficit or its contribution to reading difficulty. Rather, it argues that the phonological deficit is part of a more general auditory processing deficit. Next, we elucidate the neuroanatomical networks that underlie auditory/phonological processing, and review findings from neuroimaging and neurophysiological studies that suggest impairments in this network.

1.3.1.3. The neuroanatomical networks underlying auditory and phonological processing (and how they may break down in dyslexic readers)

At the neuroanatomical level, two parallel pathways are plausibly implicated in auditory/phonological processing of speech and nonspeech stimuli (Boets et al., 2007; Hickok & Poeppel, 2000; Scott & Johnsrude, 2003). The first is an antero-ventral auditory-to-meaning pathway, which extends from bilateral dorsal superior temporal gyrus (STG) to the left anterior superior temporal sulcus (STS) and middle temporal gyrus (MTG). Functional imaging data have suggested hierarchical processing along this pathway: while the processing of pure tones occurs mainly in the primary auditory cortex, complex spectro-temporal signal analyses (e.g., amplitude/frequency modulation) are underpinned by the antero-lateral association cortex including

bilateral dorsal STG and STS (Hart, Palmer, & Hall, 2003; Humphries, Sabri, Lewis, & Liebenthal, 2014; Liebenthal, Binder, Spitzer, Possing, & Medler, 2005; Scott, 2003; Scott & Wise, 2003). Crucially, temporal fluctuations in amplitude and spectral shape are at the core of speech perception, which is primarily accomplished in the left middle and anterior STS (Liebenthal et al. 2005; Scott & Wise, 2003).

Given the role of the auditory-to-meaning pathway in analyzing acoustic features that are critical to phoneme identity, the auditory deficit theory of dyslexia would postulate a dysfunction in this pathway. Indeed, there is good evidence for deficient neurophysiological processing in dyslexic readers with respect to speech perception and auditory temporal analyses (for reviews see Boets et al., 2007; Lytinen et al., 2005). Functional imaging studies, on the other hand, did not find consistent evidence for reduced activity in the auditory-to-meaning pathway, but rather reported anomalies in left frontal regions. Such findings, nevertheless, are consistent with evidence gained from normal subjects showing involvement of left frontal regions in processing rapidly changing auditory stimuli (e.g., Joanisse & Gati, 2003; Müller, Kleinhans, & Courchesne, 2001).

The second pathway engaged in auditory/phonological processing is an auditory-to-motor stream that links posterior temporal cortex to inferior parietal (i.e., angular gyrus and supramarginal gyrus) and inferior frontal regions (Boets et al., 2007). This network is known to support a wide range of speech- and language-related functions, including articulation and naming (primarily in frontal regions; e.g., Fiez & Peterson, 1998), lexical retrieval (e.g., Misra, Katzir, Wolf, & Poldrack, 2004), grapheme-to-phoneme mapping (e.g., Jobard, Crivello, & Tzourio-Mazoyer, 2003; Simons et al., 2002), and phonological working memory (e.g., Démonet, Thierry, & Cardebat, 2005). Not surprisingly, the phonological-core deficit theory predicts anomalies in this pathway. In support of this perspective, a large literature on phonological processing in dyslexia has shown reduced activation in left temporoparietal regions of dyslexics' brain, often accompanied by increased, compensatory activation in the inferior frontal gyrus (for reviews see, e.g., Paulesu, Danelli, & Berlinger, 2014; Pugh et al., 2001; Temple, 2002).

To sum up, both the phonological and the auditory theory provide a feasible account of developmental dyslexia, including a possible neurobiological origin. It is beyond the scope of this dissertation to assess these two accounts against each other. Rather, our primary objective, as illustrated at the beginning of this chapter, is to reveal robust indicators of dyslexia that can be detected at an early age. Given this goal, we will focus specifically on the auditory deficit of dyslexia, which can easily be assessed with beginning readers using a non-attentive paradigm (for more details see 1.4.2.).

1.3.2. Visual word recognition in developmental dyslexia

The phonological/auditory deficit theory defines dyslexia as primarily a problem with spoken language. However, dyslexia is after all a disorder of deciphering visual language codes. Under the framework of the DRC model, a parallel, complementary counterpart to the sublexical/phonological route is the lexical/orthographic route, which is necessary for the correct pronunciation of irregular words. Since the lexical route requires an orthographic input lexicon, i.e., a store of the visual form of whole-word and sublexical orthographic patterns, this route develops only with exposure to print. Once established, the lexical route functions faster than the sublexical route, because it allows reading comprehension to proceed via direct connections between the orthographic and the semantic lexicon, and reading aloud via direct connections

between the orthographic and the phonological lexicon. Consequently, fluent adult readers rely heavily on the lexical route for normal reading, both silently and aloud, and resort to the sublexical route only when encountering low-frequency words, nonwords and novel words.

A critical first step in reading via the lexical route is visual word recognition. To recognize an orthographic pattern fast and accurately, the reader needs to effectively extract invariant features from print input while ignoring large variants in font, color and other low-level stimulus features. In this way, the reader forms an abstract representation of letter/letter-string identity (also known as the visual word form; Warrington & Shallice, 1980), which can then be connected to corresponding items in the phonological and the semantic lexicon. Failure in this initial visual-orthographic analysis would hinder the computation of visual word-forms and hence lead to reading difficulties. So far, a well-formulated theory describing the visual-orthographic deficit of dyslexia at the cognitive level has yet to be developed (but see Maurer & McCandliss, 2007; McCandliss & Noble, 2003; Valdois, Lassus-Sangosse, & Lobier, 2012, discussed later). Nevertheless, a large number of neuroimaging studies have investigated the neural substrate of visual-orthographic processing and how it may break down in dyslexia.

The visual-orthographic system of the reading network is located ventrally in the extrastriate cortex. Within this system, the bilateral areas are thought to support initial visual analyses. The outcome of this preliminary processing is subsequently fed into the left anterior part of the system, a region widely referred to as the visual word form system (VWFS; Cohen et al., 2000). The VWFS is postulated to underpin the computation of visual word-forms, and the interaction between the VWFS and classical language areas, on the other hand, serves to relay the visual word-forms to phonological and semantic processing (Bruno, Zumberge, Manis, Lu, & Goldman, 2008).

Just as a radio can be tuned to receive a particular range of frequencies, the VWFS can be tuned to process print. Specifically, fMRI studies have identified two levels of VWFS tuning in fluent adult readers. First, a fast, coarse form of tuning can be found when contrasting print with visual baseline under equivalent conditions: the former consistently elicits stronger activation in the VWFS (e.g., Brem et al., 2006; van der Marker et al., 2009; Vinckier et al., 2007). The second level of VWFS tuning, i.e., the fine tuning, concerns differential processing of familiar and unfamiliar word forms (e.g., words vs. pseudo words, or high-frequency vs. low-frequency words): the former are processed more efficiently and therefore evoke lower activation in the VWFS (e.g., Bruno et al., 2008; Devlin, Jamison, Gonnerman, & Matthews, 2006; Kronbichler et al., 2004, 2007). When reported, the tuning effects (both the coarse and the fine form) were typically described in terms of relative strengths of activation. Thereby, the evidence supports preferential, but not exclusive, processing of visual word-forms in the VWFS. It has been suggested that this region embraces a form of perceptual expertise for reading a particular script, which supports fast recognition of visual words presented in that script. Putting it metaphorically, the VWFS serves as a "gateway", through which invariant features extracted from the print input come into contact with higher-level linguistic representations (Schlaggar & McCandliss, 2007).

From a developmental perspective, the functional specialization of the VWFS is thought to develop with learning to read. Initially, recognizing visual word-forms is just the same as recognizing general objects, and may engage a wide range of visual processing mechanisms. With increasing exposure to print and gradual maturation of the underlying neural circuitry, the system "learns" to make effective use of

mechanisms focusing on invariant visual features that are critical to letter identities, while reducing the usage of less appropriate mechanisms. Supporting this perspective, developmental functional imaging studies have demonstrated a gradual transition from bilateral extrastriate activations for the task of reading to more focal, left ventral occipitotemporal activations. Failure in this specialization process thus leads to deficient visual word recognition, and ultimately, to impaired reading. Accordingly, both the coarse and the fine form of VWFS specialization seem to be disrupted in dyslexia (Kronshabel, Schmid, Maurer, & Brandeis, 2013; Paulesu et al., 2001; Richlan, Kronbichler, & Wimmer, 2011; Rumsey et al., 1997; Schulz et al., 2008; Shaywitz et al., 2002; van der Mark et al., 2009; Wimmer et al., 2010).

As mentioned earlier, a well-established theory describing deficient visual word recognition at the cognitive level is still to be developed. So far, two major accounts have been put forward. The phonological mapping hypothesis, proposed by McCandliss and Noble (2003) and Maurer and McCandliss (2007), suggests that the visual-tuning deficit can be linked to the phonological-core deficit of dyslexia, as the grapheme-to-phoneme conversion process that lies at the basis of alphabetic reading could map phonological difficulties to reading-related visual processing. Alternatively, the visual-tuning deficit may constitute an independent source of reading difficulties (Valdois et al., 2012). In this dissertation, we present a cross-linguistic study aiming to investigate the underlying mechanism of specialized visual processing for print (see Chapter 4). A more detailed discussion of these two theoretical accounts is provided in the general discussion (see Chapter 5).

1.4. The current study

The objective of the current study is to reveal neurophysiological markers of developmental dyslexia in beginning readers of Dutch, thus contributing to the early diagnosis and intervention of the disorder. To fulfill this objective, we focus on two lines of research: auditory discrimination of speech and nonspeech stimuli, and visual word recognition. Specifically, we use electroencephalography (EEG) to record brain activities of children with varying degrees of reading fluency, while they are doing tasks assessing auditory/visual processing. Using generalized additive modeling (GAM; Wood, 2006), we characterize specific patterns of brain activities linked to reading abilities, thus revealing neurophysiological markers that may help distinguish poor from normal readers. Below we briefly introduce our study population, as well as the neurophysiological technique (EEG) and the statistical modeling we used to collect and analyze the data. We then close this introductory chapter with an overview of upcoming chapters in this dissertation.

1.4.1. Study population

Participants in the current project were recruited from children who participated in the Diagnosis of Dyslexia study (WakelKamp, 2015; MA-thesis), a behavioral study investigating the cognitive profiles of beginning readers of Dutch, with a focus on early diagnosis of dyslexia. In total, 75 children were recruited from 9 primary schools in various cities and villages surrounding the city of Groningen. Schools were approached through existing contacts that the Center for Language and Cognition Groningen (CLCG) had with EDventure, an association of education consultancy firms in the Netherlands. Teachers in participating schools received information about the study, and were asked to refer especially poor-reading children. Parents of the

children who were referred then received information about the study. Only children whose parents gave informed consent for participation were approached further. For these children, teachers filled out questionnaires about general learning progress and reading performance, whereas parents filled out questionnaires about neurological disorders, family history of dyslexia and other possibly relevant cognitive problems, handedness and visual/auditory acuity. Our exclusion criteria are the following:

- neurological disorders (e.g., traumatic brain injury or epilepsy)
- serious mental or psychiatric problems, emotional disturbances or attentional deficits (e.g., autism or ADHD)
- speech and language problems (e.g., specific language disorder)
- uncorrected vision or hearing problems
- bilingual background

Based on these criteria, 75 children were selected for the Diagnosis of Dyslexia study. All of them were neurologically health, right-handed and had normal or corrected-to-normal vision and hearing. A comprehensive behavioral test battery was administered to the children during the first 1.5 year of formal reading education (between the end of Grade 1 and halfway Grade 2). The test battery comprised assessments of reading skills, phoneme awareness, rapid automatized naming (RAN), and nonverbal intelligence (See Appendix A.1 for a detailed description of the tests). MA students of neurolinguistics tested the children in a silent classroom at school.

All participants of the Diagnosis of Dyslexia study and their parents were asked whether they were interested in taking part in two ERP studies. Due to several reasons (the major reason being the long-distance parents would have to travel to the ERP-laboratory), of the 75 children only 18 agreed to participate. To increase the sample size, another 3 children were recruited from a dyslexia center in the city of Zwolle, the Netherlands. Therefore, a total number of 21 children participated in the ERP experiments (9 males and 12 females; mean age [\pm SD] = 7.7 [\pm 0.46] years). Since the statistical-modeling approach we employed (generalized additive modeling; see 1.4.3.) can accommodate continuous, nonlinear predictors, we used raw reading scores to index the children's reading ability without resorting to group dichotomy (poor-reading vs. control). Behavioral measurements of individual participants are summarized in Table A.4.4 in Appendix A.4.

1.4.2. Neurophysiological measurements

Electroencephalography (EEG) is a non-invasive method to record electrical activities in the human brain using electrodes placed on the scalp. The event-related potential (ERP) represents electrophysiological response in the EEG signal that is time-locked to the onset of a particular event. Traditionally, ERPs are obtained by averaging across a large number of EEG epochs, a process that presumably removes background noise in the ongoing EEG that is unrelated to the event. The peaks and troughs in the resulting ERP waveform, typically referred to as *components*, are thought to reflect underlying neural activities (Luck, 2005). An important advantage of the ERP is that it measures neural activities with millisecond precision. Furthermore, some ERP components (including those investigated in the current study, see below) can be elicited irrespective of subject's attention or behavioral task. Therefore, compared with behavioral measures, ERPs are less likely to be contaminated by confounding factors such as task demand, attention and motivation.

Such characteristics render the ERP a particularly suitable tool for investigating basic aspects of visual and auditory processing in beginning readers. In the current study, we focus on the following ERP components to assess auditory discrimination and visual word recognition. Detailed descriptions of these components can be found in relevant chapters.

MMN

The mismatch negativity, widely known as the MMN, is a negative-going component of the auditory ERP that peaks between 160 and 220 ms, and is largest at central midline electrode sites. The MMN arises in response to any discriminable change in a repetitive chain of homogeneous auditory stimuli. Therefore, it is thought to provide an objective measure of the accuracy of auditory perception (Näätänen & Alho, 1997; Näätänen, Pakarinen, Rinne, & Takegata, 2004; Näätänen & Winkler, 1999). In accordance with the auditory deficit theory, reduced MMN to both speech and non-speech stimuli has been observed in dyslexic children, pointing to a basic deficit in auditory discrimination that underlies dyslexia (Cohen-Mimran, 2006; Kraus et al., 1996; Meng et al., 2005; Moisescu-Yiflach & Pratt, 2005; Schulte-Körne et al., 2001).

N170

The N170 (or N1) component of the visual ERP is a negative-going deflection that peaks between 150 and 200 ms over occipito-temporal electrode sites. The N170 is thought to index visual expertise of the viewer, as it is strongly elicited by familiar visual categories, such as faces, compared to low-level visual control-stimuli (Gauthier, Curran, Curby, & Collins, 2003; Latinus & Taylor, 2006). Specifically, enhanced N170 for print (words or word-like letter strings) has been associated with perceptual expertise for written language, which supports fast, automatized recognition of visual word-forms (Brem et al., 2005; Maurer, Brandeis, & McCandliss, 2005a; Maurer, Brem, Bucher, & Brandeis, 2005b; Maurer, Zevin, & McCandliss, 2008; Rossion, Joyce, Cottrell, & Tarr, 2003). In line with fMRI findings of disrupted VWFS specialization in dyslexia, the N170 tuning effect develops rapidly in typically developing children during the early phase of reading acquisition, but not in children with dyslexia (Araújo, Bramão, Faisca, Petersson, & Reis, 2012; Eberhard-Moscicka, Jost, Raith, & Maurer, 2015; Maurer et al., 2007, 2011).

1.4.3. Statistical modelling

We use generalized additive modelling (GAM; Wood, 2006) for offline analyses of the ERP data. The GAM is a novel statistical tool for analyzing time-series data. Different from linear regression, GAM provides flexible tools for modeling *nonlinear* relations, thus allowing us to assess the complete, nonlinear shape of the ERP signal over the entire timespan wherein the effect of interest might emerge. Crucially, by accommodating nonlinear interactions between multiple predictors, GAM avoids arbitrary dichotomy of inherently continuous variables (e.g., grouping the participants as control vs. dyslexic based on their reading scores), a practice that is likely to decrease statistical power and increase the possibility of finding spurious significances. Furthermore, GAM can account for systematic variations introduced by random-effect factors, e.g., subject and item, hence increasing the generalizability of

our results. A detailed introduction to GAM and its application to ERP data can be found in the Methods section of Chapter 2.

1.4.4. Outline of the dissertation

Following the lines of research outlined in this introductory chapter, Chapter 2 presents an ERP experiment investigating the relationship between reading performance and the size, as well as the lateralization pattern, of the N170 print-tuning effect. We hypothesize that the tuning effect grows larger as reading score increases, and that such correlation is particularly robust in the left hemisphere. If verified, the N170 print-tuning effect would prove to be a valid neurophysiological marker of dyslexia. In Chapter 3, we present another ERP experiment with the same group of children, which aims to assess their auditory discriminability using the mismatch negativity. Similarly, we expect a positive correlation between the size of the MMN and reading fluency, which would lend support to the validity of the MMN as a neurophysiological marker of dyslexia. In Chapter 4, to investigate whether the N170 print-tuning effect extends to logographic Chinese and to shed light on the underlying mechanism of the N170 print-tuning effect (i.e., whether it is triggered by grapheme-to-phoneme conversion or by visual familiarity with a particular script), we conducted a cross-linguistic ERP study contrasting readers of alphabetic language (Dutch) and readers of logographic language (Chinese). Finally, we conclude the thesis with a general discussion of all findings collected.