Electrophysiological studies on visual information processing in dyslexia and ADHD
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Chapter 1

General Introduction
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Background of the project

Developmental dyslexia is an inherited specific reading disability that is defined as a disturbance in learning to read and write, though there has been sufficient opportunity to learn the necessary skills and the child has had adequate education. In addition, there should be an absence of physical and intellectual disability (American Psychological Association [APA], 2000). Dyslexics in many cases fail to acquire phonological skills, which are necessary to automatise reading.

At present, dyslexia can be diagnosed in school-aged children, when a child’s reading level lags two years behind its chronological reading age. In 1995, the Dutch Health Council indexed the magnitude of the problem and stated that approximately 3% of the Dutch population is severely affected by dyslexia and that children with dyslexia continue to experience problems throughout their lifetime. A warning was issued that dyslexia is a growing problem, not only for the individuals who suffer from the symptoms but also for society as a whole, considering the emphasis today’s society places on reading and writing skills. Addressing these educational and socio-economic issues, this report ultimately led to the funding of a large-scale prospective study of dyslexia ‘Identifying the core features of developmental dyslexia: A multidisciplinary approach’.

The main goals of the study were to discover biological markers that may well allow for an early identification of dyslexia and to redefine dyslexia as a biological dysfunction. To achieve this goal, infants from 225 dyslexic families and another 120 from control families participated in the study, which started in 1998 and is still ongoing. Starting from the age of 2 months, the infants, who are being followed over a period of 10 years, are regularly assessed on measures of linguistic skills, auditory and visual processing. For the first 5 years the data acquisition was centred on measuring cortical responses to stimuli. Thereafter, behavioural measures were introduced, as the children were able to perform tasks. When the children are older, they will be tested for dyslexia. Until then we will refer to the infants from dyslexic families as at-risk.

Another goal of the prospective study was to gain insight into the frequent co-occurrence of dyslexia and ADHD. The current Ph.D. project was launched within this context. The primary goal of this Ph.D. study was to investigate information processing problems in adults with dyslexia. Secondly, as dyslexia shows significant overlap with attention-deficit hyperactivity disorder (ADHD),
another goal was to investigate the nature of the overlap between these two disorders and to possibly differentiate the disorders based on their presumably unique information processing profiles. Moreover, infants from the prospective study were investigated for early precursors of attention problems. All studies were conducted using electrophysiological methods, as these provide a window on brain functioning.

Comorbidity of dyslexia and ADHD

Developmental dyslexia and ADHD are two of the most commonly diagnosed developmental disorders, each affecting approximately 3% to 10% of school-age children (Anderson, Williams, McGee, & Silva, 1987; Shaywitz, et al., 1990; APA, 2000). These disorders co-occur in both clinical and epidemiological samples at a rate greater than would be expected by chance. The co-occurrence is estimated at 25 to 50%, depending on the criteria for inclusion (August & Garfinkel, 1990; Dykman & Ackerman, 1991; Shaywitz, Shaywitz, Fletcher, & Escobar, 1995; Willcutt & Pennington, 2000). Around 80% of children with ADHD and 60% of dyslexic children are diagnosed with more than one comorbid disorder (Willcutt, Pennington, Olson, & DeFries, 2007), inevitably complicating interpretation of research data. The occurrence of more than one condition also has implications for treatment strategy.

Both dyslexia and ADHD have a strong heritability of respectively 70% and 70-80% (DeFries, Fulker, & LaBuda, 1987; Gilger, Pennington, & DeFries, 1992) and pleiotropic loci have been identified suggesting a common genetic etiology (Gayán et al., 2005; Willcutt et al., 2007). A multifactorial model was proposed by Pennington (2006), in which comorbidity is viewed as a result of overlapping risk factors that in turn interact with genetic vulnerability, causing altered neurocognitive development and consequently deviant behaviour.

Diagnosis of dyslexia

The DSM-IV-TR (APA, 2000) defines dyslexia as a persistent reading disorder with the following three criteria:
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A. Reading achievement, as measured by individually administered standardised tests of reading accuracy or comprehension, is substantially below that expected given the person’s age, measured intelligence, and age-appropriate education.

B. The disturbance in reading ability interferes with academic achievement or activities of daily living that require reading skills.

C. If a sensory deficit is present, the reading difficulties are in excess of those usually associated with it.

Dyslexics often show difficulty with verbal short-term memory, word and non-word repetition, and rapid automatised naming (Paulesu, et al., 1996), although dyslexia can manifest itself differently throughout an individual’s development into adulthood, as one learns to cope with the disorder. It must be noted that dyslexia concerns a heterogeneous group. Consequently, subtypes of dyslexics have been defined, based on the specific processing deficits involved, although the DSM-IV-TR does not acknowledge these as such. Out of those diagnosed with the disorder, 60-80 % is male.

Dyslexia: a biological perspective

Post-mortem and animal model studies have related behaviour symptoms in dyslexics to neuro-anatomical anomalies, including reduction in cell size of thalamic cells, cortical microgyria and heterotopias due to deviant cell migration, and altered callosal morphology (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Hynd et al., 1995; Kaufmann & Galaburda, 1989; Livingstone, Rosen, Drislane, & Galaburda, 1991; Njokiktjien, De Sonneville, & Vaal, 1994; Rumsey et al., 1996). These data suggest that neuronal development may be altered. In addition, the animal model (cf. chapter 5) suggests that functional connectivity between brain regions may be different in dyslexics. The abnormalities, along with the finding of visual deficits in dyslexia, have been explained in terms of a processing deficit related to the magnocellular division of the visual system (Lovegrove, Martin, & Slaghuis, 1986; Stein & Walsh, 1997). This particular theory of dyslexia will be given special consideration, as it may offer insight into the biological mechanism for the visual deficits seen in dyslexia.
The visual system

The magnocellular system, or transient visual system, is sensitive to low contrast, low spatial and high temporal frequencies, whereas a second system, the parvocellular system is responsible for processing of detailed spatial information and colour. The visual system has a hierarchical structure. The magnocellular pathway (M-pathway) begins in the retinal ganglion cells and projects to the lateral geniculate nucleus in the thalamus. From there the visual information is transferred to the primary visual cortex and on to secondary and higher order visual areas to terminate in the parietal cortex (dorsal stream), unlike the parvocellular stream, which ends in the temporal cortex (ventral stream). Feedforward and feedback signals in the visual pathways can provide enhancement of attention in a particular location.

Reading, attention and the magnocellular theory

Reading entails the decoding of graphemes and the coupling of graphemes to phonemes. An experienced reader achieves fluency by using the graphemic route of reading, thus reading a word as whole. This graphemic route of reading (Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001) has been found to rely on visual attentional mechanisms and allows for high processing speed whilst reading (Casco, Tressoldi, & Dellantonio, 1998; Omtzigt, Hendriks, & Kolk, 2002; Valdois et al., 2003).

A model was proposed (Vidyasagar & Pammer, 1999; Vidyasagar, 2004) explaining how visuo-spatial attentional resources are used in reading. In this model, the M-pathway projects to the posterior parietal cortex producing a spatial map of the visual field. Information from the parietal cortex is then fed back to the ventral cortex where detailed parvocellular processing occurs. There, the spatial information is bound with detail information and the position of letters is encoded. So, letter encoding is essentially proposed to be controlled by the posterior parietal cortex (Kinsey, Rose, Hansen, Richardson, 2004; Steinman, Steinman, & Garzia, 1998).

Deficits seen in dyslexics, such as sluggish attention shifting (Hari & Renvall, 2001), automatised naming of words or figures (Denckla & Rudel, 1976), have been proposed to be the result of deficient feedforward and feedback signals in the visual system (Laycock & Crewther, 2008).
Diagnosis of ADHD

ADHD is a behavioural disorder, which is defined by either an attentional dysfunction, hyperactive/impulsive behaviour or both. The diagnosis of ADHD therefore has three subtypes: the inattentive subtype, the hyperactive/impulsive subtype and the combined inattentive-hyperactive/impulsive subtype. Most children with ADHD suffer from the combined subtype. The diagnostic criteria state that the disorder should manifest itself before the age of 7 and it should be present in more than one situation, for example at home and at school. Also, there must be a clinically significant dysfunction and the disorder may not be secondary to a psychotic or pervasive developmental disorder (DSM-IV-TR, 2000). In children 6 or more inattentive or hyperactive/impulsive symptoms must be reported to occur often.

The items of inattention include: failure to pay attention to details or carelessness, difficulty in sustaining attention, not listening when spoken to, lack of persistence, difficulty organising tasks, avoidance or dislike of tasks involving sustained mental effort, often losing things necessary for tasks, distractibility, and forgetfulness. The hyperactive/impulsive dimension encompasses: fidgeting, not remaining seated, excessive motor activity, difficulty engaging in activities quietly, excessive talking, blurting out answers before questions have been completed, difficulty awaiting ones turn, and interrupting or intruding on others.

In roughly half of the children diagnosed with ADHD, symptoms persist into adulthood (Spencer, Biederman, Wilens, & Faraone, 1998). Therefore, ADHD has also been validated as an adulthood disorder (Faraone et al., 2000), with remaining symptoms in adults including distractibility and difficulties with maintaining goal-directed behaviour rather than hyperactivity. Self-report rating scales, based on the DSM-IV-TR, have been developed to quantify symptoms of ADHD in adults (e.g. Kooij et al, 2004).

Executive functioning in ADHD

ADHD has been conceptualised as a deficit in executive functioning such as planning, response inhibition and maintaining goal-directed behaviour. These deficits in ADHD are thought to stem from an underlying deficit in prefrontal lobe function. Barkley (1997) hypothesised that deviant behaviour seen in ADHD is
caused by a deficit in behavioural inhibition, which was defined as the ability to suppress, delay, or alter an action that is provoked by internal or external stimulation. Impaired inhibitory control was therefore proposed to be the core deficit in ADHD, due to the observation that subjects with ADHD show a slower inhibition process on neuropsychological tasks measuring executive functions (e.g. Wisconsin Card Sorting Test, Stroop, Tower of London). Fronto-striatal pathways have been assumed to be involved in the inhibitory deficit.

Reaction time (RT) tasks have been devised to tap inhibitory control in ADHD. However, serious doubts have been expressed as to whether inhibitory control should be viewed as the only core deficit in ADHD (Scheres et al., 2004), as inhibitory deficits on these tasks turned out not to be specific to ADHD children, but have been seen in children with ODD and CD (Oosterlaan, Logan & Sergeant, 1998; Wiersema, van der Meere, Roeyers, Van Coster, & Baeyens, 2006) as well as dyslexics (Donfrancesco, Mugnaini, & Dell’Uomo, 2005; Purvis & Tannock, 2000; Van der Schoot, Licht, Horsley, & Sergeant, 2000). Thus, despite attempts to reveal a fundamental core deficit for ADHD, so far there is still uncertainty as to whether a deficit in inhibitory control can explain attentional and behavioural deficits sufficiently.

Two models of attention

Cognitive-energetic model
Van der Meere (2002) employed a cognitive-energetic model by Sanders (1983, 1998) to explain the deficits seen in ADHD. He suggested that problems in inhibitory control could be the result of the energetic state of the child, influenced by variables such as presentation rate of stimuli, reward, and presence or absence of the experimenter. The cognitive-energetic model describes information processing as a series of computational input, central cognitive, and output stages: stimulus pre-processing, feature extraction, response choice, and motor adjustment, which determine the efficiency of performance. In turn, the efficiency of these stages depends on the availability of energetic sources identified as arousal and activation.

The first, arousal, is described as a phasic physiological response, time-locked to incoming stimuli. Arousal mediates the input side of information-processing and is related to the novelty, complexity or surprise of the input (Pribram & McGuiness, 1975). Brain structures related to this system are the spinal
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cord, reticular formation, hypothalamus, amygdala, and frontal cortices. Noradrenaline and serotonin are the principal neurotransmitters in these pathways.

The second system, activation is tonic in nature and affects the readiness to respond. This system is controlled by thalamic and fronto-striatal structures. Dopamine is the dominant neurotransmitter in the activation system.

The model is hierarchic, in that a third energetic resource, effort, governed by an evaluation mechanism, modulates computational processing through arousal and activation. The evaluation mechanism gauges arousal and activation levels and activates or inhibits the effort pool to (re)turn the individual to an optimal state.

Within this model ADHD is explained as a dysfunction in state regulation and thus allocation of attentional resources rather than an attentional deficit (Sergeant & van der Meere, 1988; van der Meere, 2002; Sergeant, Geurts, Huijbregts, Scheres, & Oosterlaan, 2003). This model offers a framework for studying information processing in ADHD, as it incorporates distinguishable stages of input, central cognitive, and output related processes, as well as the energetic factors influencing them.

Posner’s networks of attention

In understanding spatial attention, an analogy is often made to a spotlight that enhances attention in a spatial location (Posner, 1980). Tasks of spatial attention may use a cue to act as a spotlight, facilitating selective attention at a certain location. On these visuo-spatial orienting tasks, the focus of attention is captured by a cue before eye movements can take place. Posner described three brain networks of attention to be involved in directing selective attention: a posterior attention, anterior attention, and a vigilance network (Posner and Petersen, 1990; Posner & Raichle, 1994).

The posterior attention network involves the superior colliculus, pulvinar nucleus and the parietal lobe, and is involved in covert shifts of attention. These structures are respectively responsible for disengaging, moving and engaging of attention to a new location.

The anterior attention network is active when information is processed actively and is mediated by the anterior cingulate gyrus as well as the prefrontal cortex. This network is involved in executive control of information processing and also exerts control over the posterior network.

The third, vigilance, network has a role in maintaining alertness. The vigilance network can modulate the other two networks in order to increase
alertness, thereby increasing efficiency of orienting to stimuli. This network is thought to be modulated by noradrenergic input from the locus coeruleus (Aston-Jones, Chiang, & Alexinsky, 1994), which exerts influence over frontal and parietal lobes.

Attention tasks

In the present Ph.D., two tasks were used that demand the use of all three networks and tap all three energetic pools. These tasks will be described in this section.

**Visuo-spatial orienting tasks**
The attention networks described by Posner can be manipulated by employing visuo-spatial attention tasks, which use a cue to evoke a covert orienting response to a certain location. The cue can be either endogenous, i.e. a centrally presented arrow, pointing toward the visual hemifield that is to be attended to or exogenous (e.g. located peripherally). Peripheral cues and short cue-target intervals evoke automatic attention, controlled by the posterior attention network, while longer cue-target intervals and central cues require endogenous attention, provided by the anterior attention network. In addition, cues can be valid, in that they correctly predict the target location, or invalid when the target appears in the opposite visual hemifield. A validly cued target facilitates information processing at the cued location.

In extensive research by Facoetti and colleagues (Facoetti, Paganoni, Turatto, Marzola, & Mascetti, 2000; Facoetti, 2001; Facoetti et al., 2006), covert orienting of attention has been studied in dyslexic children using RT tasks. In these studies, evidence was found for orienting deficits to exogenous peripheral cues, suggesting problems with automatic visuo-spatial attention. Endogenously controlled attention was found to be intact. Moreover, attention was found to be distributed asymmetrically across the visual field, suggesting that dyslexic individuals have a right hemisphere processing deficit in addition to slower processing speed (Hari & Renvall, 2001). In line with this suggestion, patients with damage to posterior regions of the right parietal lobe have been found to suffer from acquired dyslexia (Kinsbourne & Warrington, 1962; Ruddock, 1991).

Many studies of visuo-spatial attention in ADHD have been conducted. A meta-analysis, revealed that individuals with ADHD responded slower when
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endogenous attention was required, suggesting an anterior attention deficit (for a review see Huang-Pollock & Nigg, 2003).

Thus, so far, the evidence suggests deficits in visuo-spatial orienting in dyslexia and, in contrast, deficits in controlled attention in ADHD.

Continuous Performance Tests
One of the most frequently used tasks for measuring deficits in executive control and attention in ADHD is the Continuous Performance Test (CPT) developed by Rosvold and colleagues (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). In this computer task, the subject monitors sequentially presented stimuli and is asked to respond to a target stimulus with a button press. In another version of the CPT, the A-X CPT the subject is asked to identify a target letter X, but to respond only when it follows the letter A, which acts as a cue. Using an A-X CPT allows a distinction between orienting to the cue, attentional processes related to the target and preparatory processes related to action.

Performance deficits observed in CPTs have been related to deficiencies in cognitive processing. Firstly, errors of commission are assumed to reflect impulsivity, whereas errors of omission reflect inattention (Halperin, Wolf, Greenblatt, & Young, 1991). These types of errors are common in both children and adults with ADHD (Epstein & Sitarenios, 1998). Furthermore, participants with ADHD generally respond slower and more variably than normal controls on CPTs (Klein, Wendling, Huettner, Ruder, & Peper, 2006). Increased RT and variability of RT on these CPTs have been thought to reflect difficulties allocating processing resources (Riccio, 2002). In ADHD, these performance deficits have been related to deficient arousal levels mediated by ascending and descending pathways from the reticular activating system (Voellar, 1991), and deficient state regulation in terms of the Sanders model (van der Meere, Stemerdink, & Gunning, 1995). Rothlind and colleagues (1991) suggested that problems in sustaining attention to a certain location pointed towards a problem in the vigilance network (Rothlind, Posner, Schaugnecy, 1991).

With respect to dyslexia, a CPT study conducted on dyslexic adolescents pointed towards impaired attentional processing compared to controls due to parietal lobe problems (Taroyan, Nicolson, & Fawcett, 2007). The data also suggested an altered interhemispheric collaboration in dyslexics, as demonstrated by abnormalities in lateralisation of the P3.
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Studying attention with electrophysiological methods

To a large extent, researchers have applied neuropsychological tests to distinguish cognitive impairments. However, one complicating aspect is that the performance measured often relies on several underlying cognitive processes, e.g. orienting, selection, working memory, response selection, and motor planning. So a deficit in measured performance, as reflected by errors, RT, and variability of responses, may signify impairment in any one of these underlying cognitive processes, complicating interpretation of behavioural data. Using the ERP methodology has several advantages over neuropsychological testing. First of all, it lends insight into the timing of neural information processing due to its high temporal resolution (milliseconds), enabling an isolation of various aspects of information processing. Another advantage is that covert differences, if present, can be detected in the absence of overt responses. Additionally, energetic aspects of attention, i.e. the amount of allocated attention, can be measured.

In order to obtain robust ERPs, the small voltage changes, measured from the scalp EEG, are time-locked to the onset of a stimulus. Many stimuli must be averaged to enhance signal-to-noise ratio. ERPs are characterised by their latency, amplitude and topography.

The names of ERP components are derived from the polarity and latency at which they occur. For example, the N2 is a negative-going deflection, which occurs around 200 ms post stimulus. Furthermore, components can be categorised along a continuum from exogenous to endogenous. Exogenous components are elicited by a stimulus and reflect characteristics (quality and intensity) of the stimulus. They are related to automatic, input related, sensory processes. Endogenous components occur from some 200 ms post stimulus and reflect demands on controlled central processing. They can be related to the central cognitive processes of comparison, evaluation, decision-making and response preparation.

The components investigated in the current Ph.D. study will concisely be described below. The N2 is a component that is considered to be a correlate of selective attention and stimulus identification (for a review see Patel & Azzam, 2005), or response conflict (Nieuwenhuis, Yeung, & Cohen, 2004). Several studies considered a frontocentral N2, elicited by Nogo trials of a Go/Nogo task, to reflect inhibitory control (Pfefferbaum & Ford, 1988; Eimer, 1993).
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The P3 with a parietal maximum between 300 and 700 ms after stimulus onset, is said to reflect context updating and has often been used as a measure of attentional resource allocation (Donchin & Coles, 1988). A smaller than normal P300 has been postulated to index either decreased availability of attentional resources or the inability to allocate them appropriately. The P3 amplitude is larger for attended stimuli than for non-targets. The latency of the P3 provides a measure of stimulus evaluation timing (Kok, 2001). On tasks measuring response inhibition, the P3 was shown to have a more enhanced fronto-central maximum and a longer latency when a response had to be inhibited (Bokura, Yamaguchi, Matsubara, & Kobayashi, 2001; Eimer, 1993; Fallgatter, Brandeis, & Strik, 1997; Tekok-Kilic, Shucard, & Shucard, 2001). This Nogo P3 was found to give an accurate estimation of inhibitory control.

The Contingent Negative Variation (CNV) consists of two consecutive parts respectively reflecting input- and output-related processes. The first part provides a measure of orienting to a cue, whereas the second part reflects motor preparation of the response to this stimulus (Van Boxtel & Böcker, 2004).

All of the described ERP components were elicited by the two tasks, a visuo-spatial orienting task and an OX-CPT, that were used in this project.

The EEG approach also allows for studying the functional connectivity of neural networks through which communication between cortical regions occurs. This can be accomplished by measuring the level of coherence of EEG frequencies between regions of interest, whereby information on the coupling of synchronous neural firing in separate regions is utilised. Using this method, dysfunctions in connectivity between specific cortical regions have been found in clinical populations with neurocognitive impairments, such as autism spectrum disorder (e.g. Coben, Clarke, Hudspeth, & Barry, 2008) and schizophrenia (e.g. Higashima et al., 2007). Also, evidence has been found for abnormal cortico-cortical coherence in dyslexia. However, coherence studies on dyslexia are scarce and have not always proved to be methodologically sound (French and Beaumont, 1984; Leisman, 2002). Yet, there is reason to suspect that in dyslexia functional connectivity between distinct cortical regions may be affected. This method, thus, may help to explain some of the specific neurophysiological mechanisms underlying cognitive dysfunction in dyslexia.
Aims and outline of the thesis

Aims

The magnocellular account describes how visuo-spatial attention deficits may affect reading in dyslexics and predicts that visuo-spatial orienting will be deficient in dyslexics. In terms of information processing, deficits are expected in early information processing, as most evidence has been found for input related problems. Besides these deficits, strong evidence has also been found for inhibition problems in dyslexia. This thesis investigates whether ERP evidence can be found for input related, visual orienting problems and inhibition deficits in dyslexics.

Furthermore, as evidence from an animal model of dyslexia predicts that dyslexic brains may have had an alternative development, leading to altered interhemispheric wiring, a second objective of this project will be to seek evidence for this hypothesis by determining functional connectivity between the hemispheres. This can be achieved by measuring the interhemispheric EEG coherence, whereby the level of coherence reflects the amount of information relayed between the regions, thus providing a measure of functional connectivity.

In ADHD, information processing problems have been related to an individual’s underlying state. For the most part, research on the comorbidity of dyslexia and ADHD has been undertaken using neuropsychological tests or RT paradigms, while measuring performance, to investigate brain functioning. By using psychophysiological research methods insight may be gained into how the groups differ in various stages of visual information processing. The third objective of this project was thus to gain insight into visual information processing differences and similarities between dyslexia and ADHD. It is expected that adults with ADHD will demonstrate central cognitive (attention allocation) and output related (inhibition) impairments and that dyslexic adults will show input-related (orienting) deficits as well as output-related (inhibition) deficits.

The comorbidity of dyslexia and ADHD suggests that this group may show additive effects of both disorders. The fourth objective of this dissertation will be to evaluate whether electrophysiological evidence of such additivity can be found, or whether the comorbid group can in fact be discriminated from a ‘pure’ dyslexic and ‘pure’ ADHD group.

Finally, as described earlier, the Dutch Prospective Study may be able to provide early neurobiological markers for dyslexia. Hence, electrophysiological
processing of infants at risk for dyslexia will be investigated on paradigms depending on magnocellular functioning. Visuo-spatial orienting in these infants may reveal precursors of deficiencies seen in adults with dyslexia.

To sum up, the research questions were:

- Do dyslexic adults show visuo-spatial attention difficulties?
- Can dyslexic adults be discriminated from adults with ADHD by the type and timing (early vs. late) of information processing deficits?
- What profile do individuals with comorbid dyslexia and ADHD show compared to a ‘pure’ dyslexic and ‘pure’ ADHD group?
- Is there evidence for abnormal interhemispheric processing in dyslexics, as predicted by neuro-anatomical and animal studies?
- Is it possible to find an ERP marker for visuo-spatial orienting deficits in infants at risk for dyslexia?

Outline of the thesis

Chapter 2 presents a study on information processing of adults with dyslexia, ADHD, and comorbid adults. A visuo-spatial orienting task was used to compare ERP characteristics. Furthermore, based on differences in information processing, participants with dyslexia and ADHD will be discriminated from each other and from comorbid participants.

Chapter 3 again centres on information processing differences between dyslexia and ADHD, but now a continuous processing task is administered, whereby participants are required to sustain attention to a task, hence probing more effortful processing.

Chapter 4 returns to visuo-spatial attention in dyslexic adults. This time the interhemispheric communication involved during the processing of this task will be considered. This study was based on an animal model of dyslexia. Results of the study will be discussed in terms of a possible deficit in magnocellular processing.

In chapter 5 a study is reported that investigated infants from the Prospective Dyslexia Study at the age of 5 months on a visuo-spatial orienting task similar to the one administered to adults.
Finally, a summary of the results will be presented and discussed, concluding some with limitations of the present studies and suggestions for future research.

The chapters will be presented in their original form, as they were accepted or submitted for publication.
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