JYVÄSKYLÄ STUDIES IN EDUCATION, PSYCHOLOGY AND SOCIAL RESEARCH
366

Tiina Huttunen-Scott

Auditory Duration Discrimination in Children with Reading Disorder, Attention Deficit or Both

JYVÄSKYLÄN YLIOPISTO

JYVÄSKYLÄ STUDIES IN EDUCATION, PSYCHOLOGY AND SOCIAL RESEARCH 366

Tiina Huttunen-Scott

Auditory Duration Discrimination in Children with Reading Disorder, Attention Deficit or Both

Esitetään Jyväskylän yliopiston yhteiskuntatieteellisen tiedekunnan suostumuksella
julkisesti tarkastettavaksi yliopiston Villa Ranan Blomstedtin salissa
elokuun 1. päivänä 2009 kello 12.

Academic dissertation to be publicly discussed, by permission of
the Faculty of Social Sciences of the University of Jyväskylä,
in the Building Villa Rana, Blomstedt Hall, on August 1, 2009 at 12 o'clock noon.

UNIVERSITY OF JYVÄSKYLÄ

JYVÄSKYLÄ 2009

Auditory Duration Discrimination in Children with Reading Disorder, Attention Deficit or Both

JYVÄSKYLÄ STUDIES IN EDUCATION, PSYCHOLOGY AND SOCIAL RESEARCH 366

Tiina Huttunen-Scott

Auditory Duration Discrimination in Children with Reading Disorder, Attention Deficit or Both

UNIVERSITY OF JYVÄSKYLÄ

JYVÄSKYLÄ 2009

Editors
Aarno Laitila
Department of Psychology, University of Jyväskylä
Pekka Olsbo, Marja-Leena Tynkkynen
Publishing Unit, University Library of Jyväskylä

URN:ISBN:978-951-39-3658-7
ISBN 978-951-39-3658-7 (PDF)

ISBN 978-951-39-3628-0 (nid.)
ISSN 0075-4625

Jyväskylä University Printing House, Jyväskylä 2009

ABSTRACT

Huttunen-Scott Tiina
Auditory duration discrimination in children with reading disorder, attention deficit or both
Jyväskylä: University of Jyväskylä, 2009, 68 p.
(Jyväskylä Studies in Education, Psychology and Social Research
ISSN 0075-4625; 366)
ISBN 978-951-39-3658-7 (PDF), 978-951-39-3628-0 (nid.)
Yhteenveto: Kuulonvarainen keston erottelu lapsilla, joilla on lukemisvaikeus, tarkkaavaisuuden ongelma tai molemmat
Diss.

Auditory processing has been suggested as one possible cause of reading disorder (RD). The studies of this thesis investigated auditory processing skills in children with RD as well as in children with attention deficit (AD). Duration was chosen as the stimulus feature since duration has a central role in Finnish. In the first study an event-related potential (ERP) peak called mismatch negativity (MMN) was measured in controls and children with RD or AD. No significant differences in the MMN amplitudes or latencies were found between the groups, but the MMN peaks were more lateralized to the right hemisphere in the control group, whereas in the RD group the MMNs were more pronounced in the left hemisphere. In the second study stricter group selection criteria were applied in order to investigate whether the MMNs of participants with either "pure" RD or AD differ from those of participants with both types of problems. MMNs of participants with RD were diminished as compared to those of the controls in frontal and central channels in the right hemisphere. In addition, the MMNs of the participants with comorbid RD and AD were attenuated in both hemispheres and in all frontal and central channels. The MMNs of the AD group did not differ from those of controls in amplitude, but the MMN peaks appeared earlier in this group. In the third study a behavioral speech duration discrimination task was introduced to the participants in addition to the MMN experiment in order to explore whether behavioral and ERP tasks provide similar results concerning the duration processing in participants with either RD, AD or both. MMNs with bigger amplitudes as well as superior behavioral duration discrimination were observed in participants with AD as compared to those with RD or both RD and AD. The results of the studies suggest a duration discrimination deficiency in Finnish children with RD, whereas no such problem was found in children with "pure" AD.

Keywords: Mismatch negativity (MMN), reading disorder (RD), attention deficit (AD), auditory processing, duration discrimination

Author's address Tiina Huttunen-Scott
Department of Psychology
P.O. Box 35
FIN-40014 University of Jyväskylä
tihuttun@psyka.jyu.fi

Supervisors Professor Heikki Lyytinen
Department of Psychology
University of Jyväskylä, Finland

Doctor Jukka Kaartinen
Department of Psychology
University of Jyväskylä, Finland

Professor Timo Ahonen
Department of Psychology
University of Jyväskylä, Finland

Reviewers Professor George Hynd
Arizona University

Professor Thomas Lachmann
University of Kaiserslautern

Opponent Professor George Hynd
Arizona University

ACKNOWLEDGEMENTS

I would like to thank my supervisors Prof. Heikki Lyytinen, Dr. Jukka Kaartinen, and Prof. Timo Ahonen for their guidance and valuable time. I would also like to thank co-authors Anne Halonen, Dr. Asko Tolvanen and Dr. Ulla Richardson for their contributions to the articles as well as the reviewers Prof. George Hynd and Prof. Thomas Lachmann for their helpful comments. I also wish to thank Michael Freeman for checking the language, Lauri Viljanto for his technical assistance, and Raija Mehto for her help and support. A special thank you to my family members who have always been there for me. This dissertation is dedicated to my husband Ira who has been my most devoted motivator and supporter. Thank you for giving me "sisu" to go on and especially for making me so happy.

The studies of this doctoral dissertation were financially supported by the Finnish Graduate School of Psychology and the Faculty of Social Sciences, University of Jyväskylä.

LIST OF PUBLICATIONS

Study I Huttunen, T., Halonen, A., Kaartinen, J. & Lyytinen, H. (2007). Does mismatch negativity show differences in reading-disabled children compared to normal children and children with attention deficit? *Developmental Neuropsychology*, 31, 453-470.

Study II Huttunen-Scott, T., Kaartinen, J., Tolvanen, A., & Lyytinen, H. (2008). Mismatch negativity (MMN) elicited by duration deviations in children with reading disorder, attention deficit or both. *International Journal of Psychophysiology*, 69, 69-77.

Study III Huttunen-Scott, T., Kaartinen, J., Tolvanen, A., Richardson, U., & Lyytinen, H. (submitted). A study of electrophysiological and behavioral aspects of auditory duration discrimination in children with reading disorder, attention deficit or both.

CONTENTS

1 INTRODUCTION

The theme of this dissertation is auditory duration processing in children with reading disorder (RD), attention deficit (AD), or both as compared to children with no problems in these skills. The first two studies of the dissertation applied electrophysiological approach to auditory duration processing evaluation, and the third study combined results from both electrophysiological and behavioral duration discrimination tasks. The electrophysiological measure used in evaluating auditory processing was an event-related potential (ERP) called mismatch negativity (MMN), which is elicited by a change in some feature of a repeated auditory stimulus. The behavioral auditory duration discrimination was examined by a task requiring categorization of pseudowords (/ata/-/atta/) varying in duration of stop closure.

Two main factors make these studies unique. First, although auditory processing has been studied actively in children with reading problems, relatively few studies have investigated this kind of processing in participants with attention problems, and to the knowledge of the author no study has studied auditory duration processing indexed by MMN in children with pure reading disorder as well as in those with pure attention deficit versus children with problems in both reading and attention. Attention problems often co-occur with reading problems, and thus it is important to include participants showing both kinds of problems in studies probing auditory deficit as one possible cause of reading problems. Second, these studies apply duration as the key auditory feature under investigation. Duration is a semantically central feature in Finnish language, and thus auditory duration processing is especially critical for Finnish speakers. I therefore wished to examine whether Finnish participants with RD show auditory duration processing problems, which have not been always shown in English speaking participants with similar problems (Baldeweg, Richardson, Watkins, Foale, & Gruzelier, 1999).

1.1 Reading disorder

Specific reading disorder refers to deficiencies in the development of reading skills that are not explained by mental age, visual acuity problems, or inadequate schooling (WHO, 1990). Another term often used when discussing problems in reading is dyslexia, which refers to a specific learning disability that has neurobiological origin and is characterized by difficulties with accurate and/or fluent word recognition and by poor spelling and decoding abilities (Lyon, Shaywitz, & Shaywitz, 2003). Throughout history terms used to describe problems in reading have varied depending on the focus of the research, and there is still plenty of variation in the terms and definitions used. In this thesis the word reading disorder (RD) is used to refer to specific difficulty in reading, whereas reading problems are applied as a more general term including also cases possibly showing other kinds of language problems in addition to reading disorder. When referring to other studies, the terms used by the authors are used.

Reading disorder affects approximately 6% of the population (Lyytinen, Leinonen, Nikula, Aro, & Leiwo, 1995; Pennington, 1990; Shaywitz, Shaywitz, Fletcher, & Escobar, 1990), and due to its high prevalence and profound effects on academic performance it has been actively studied from different perspectives. Reading disability also seems to continue to manifest in adulthood (Felton, Naylor, & Wood, 1990). Independent of the achieved reading level adults with history of reading disability continue to show deficiencies in neuropsychological measures of language skills (Felton, Naylor, & Wood, 1990). Thus, reading problems seem to display a profound deficit that can still be verified even if reading does not appear to be causing profound problems anymore.

Phonological processing is considered to be the core deficit of reading problems (Bradley & Bryant, 1983; Brady & Shankweiler, 1991; Wagner & Torgesen, 1987). Phonological processing refers to the ability to process the smallest units of language, phonemes, which constitute the whole linguistic system. In order to operate on the phonemes, phonological awareness must be developed, which means understanding that the language is constructed of phonemes: any word can be broken into separate phonemes. In addition to phonological processing another significant component of the reading process is orthographic processing, which refers to the ability of an individual to identify words or meaningful word parts on the basis of a visual representation formed in the memory (Barker, Torgesen, & Wagner, 1992).

Although problems in phonological processing seem to be a common problem to individuals with RD, there are differences between different language speaking individuals in the development of reading skills: children learning to read more consistent orthographies (i.e. Greek, Finnish) seem to develop grapheme-phoneme recoding skills faster than children learning to read less consistent orthographies (i.e. English, French). According to Goswami

(2002) children learning to read more consistent orthographies have the advantage of being able to focus exclusively on the phonemes without making many errors, whereas readers of less consistent orthographies have to adopt several parallel strategies of different grain sizes (whole word, onset, rhyme, phoneme) in order to read the exception words correctly.

There are also differences in the appearance of RD in different languages: in languages with less consistent orthographies the most pronounced difficulty is accuracy of phonological processing, whereas in languages with more consistent orthographies reading problems show more in the skills needed for automatization of these processes (Grigorenko, 2001; Serrano, & Defior, 2008). Thus, in orthographically more difficult languages problems in phonological processing show more as mistakes in reading, whereas in easier languages problems are more apparent in speed (Landerl, 1997).

More evidence of the role of phonological skills in reading problems comes from intervention studies: Training methods based on phonology have been shown to be effective in improving reading skills (Hatcher & Hulme, 1999; Torgesen et al., 1999). The effects of training have even been reported to show in brain correlates (Kujala et al., 2001; Richards et al., 2000; Temple et al., 2003).

Participants with RD often also experience other problems for example in working memory (e.g. Baddeley, 2003; Brady, 1991; Cohen & Netley, 1981; De Jong, 1998; Nation, Adams, Bowyer-Crane, & Snowling, 1999; Willcutt et al., 2001), eye movement control (Eden, Stein, Wood, & Wood, 1994; Fischer & Weber, 1990; Klicpera, Wolff, & Drake, 1981; Martos & Vila, 1990), and motor skills (e.g. Chiarenza, 1990; Fawcett, Nicolson, & Dean, 1996; Kinsbourne, Rufo, Gamzu, Palmer, & Berliner, 1991; Nicolson & Fawcett, 1994). The motor problems often seen in participants with RD have inspired some researchers to look for the cause of these problems in cerebellar functions (Fawcett, Nicolson, & Dean, 1996; Nicolson, Fawcett, Berry, Jenkins, Dean, & Brooks, 1999, Nicolson, Fawcett, & Dean, 2001). According to the cerebellar hypothesis the main cause of RD and the related problems is the dysfunction of cerebellum, which hinders the acquisition and automatisation of auditory, articulatory and visual skills, which in turn shows as poor phonological processing and finally as RD (Nicolson, Fawcett, Berry, Jenkins, Dean, Brooks, 1999; Nicolson, Fawcett, & Dean, 2001).

The left hemisphere plays a significant role in the language functions and several important language areas typically reside in this hemisphere. Already Orton (1925) suggested that reading problems are related to failure of the left hemisphere to dominate language functions. Typically the cortical areas critical for language functions show greater left than right asymmetry (Falzi, Perrone, & Vignolo, 1982; Geschwind, & Levitsky, 1968; Weinberger, Luchins, Morihisa, & Wyatt, 1982). However, in participants with language deficits deviations from the normal pattern of lateralization have been reported (Haslam, Dalby, Johns, & Rademaker, 1981; Hier, LeMay, Rosenberger, & Perlo, 1978; Plante, Swisher, Vance, & Rapcsak, 1991; Rosenberger, & Hier, 1980). According to the Geschwind-Behan-Galaburda (GBG) hypothesis (Geschwind & Behan, 1982;

Geschwind & Galaburda, 1985), the abnormal cerebral lateralization is due to high levels of prenatal testosterone. Several studies have reported abnormalities in the brain structures of participants with RD, especially in the left hemisphere.

1.1.1 Genetic and neuroanatomical findings

Based on observations of families (DeFries, Singer, Foch, & Lewitter, 1978; Hallgren, 1950; Pennington et al., 1991; Scarborough, 1989; Vogler, DeFries, & Decker, 1985) and twins (Alarcón & DeFries, 1997; Bakwin, 1973; DeFries & Alarcón, 1998; DeFries, Fulker, & Labuda, 1987; Gayan & Olson, 2001) of participants with reading disorder, the strong inheritance of these problems has been acknowledged for a long time. Especially regions of chromosome 1 (Grigorenko et al., 2001; Rabin et al., 1993; Tzenova, Kaplan, Petryshen, & Field, 2004), chromosome 2 (Fagerheim et al., 1999; Petryshen, Kaplan, Hughes, Tzenova, & Field, 2002), chromosome 3 (Nopola-Hemmi et al., 2001), chromosome 6 (Cardon et al., 1994; Fisher et al., 1999; Grigorenko et al., 1997, 2000), chromosome 15 (Grigorenko et al., 1997; Morris et al. 2000; Nöthen et al., 1999; Schumacher et al., 2008; Smith, Kimberling, Pennington, & Lubs 1983), and chromosome 18 (Fisher et al., 2002) have been found to contain genes related to reading disability (For a review, see Fisher & DeFries 2002). It has also been found that phonological decoding and orthographic coding have significant independent genetic variance (Olson, Datta, Gayan, & DeFries, 1999), and that this genetic variance differs as a function of IQ (Knopik et al., 2002; Wadsworth, Olson, Pennington, & DeFries, 2000). It seems that the genetic influences might play a more important role in participants with RD with higher IQ scores (Knopik et al., 2002; Wadsworth, Olson, Pennington, & DeFries, 2000). Although there seems to be a genetic basis for reading disability, or at least for some proportion of it, the exact transmission mechanisms are not known.

Post-mortem studies have shown that brains of individuals with reading problems often show anomalies in cortical areas central to language processing, which points to the neurobiological origin of the problem. Post-mortem studies have found brains of individuals with reading problems to have shown neuronal ectopias (malpositioned neuron groups) and architectonic dysplasias (absence of typical architectural organization of cortical neurons) especially in the perisylvian regions of the left hemisphere; lack or reduction of the typical cerebral asymmetry in the planum temporale (Galaburda, Sherman, Rosen, Aboitiz, & Geschwind, 1985; Hughdahl et al., 1998; Humphreys, Kaufmann, & Galaburda, 1990); as well as lesions in the inferior frontal gyrus (Galaburda et al., 1985). Also, differences in the cell sizes have been reported. Participants with dyslexia have been reported to show increased proportion of large neurons and fewer small neurons in cerebellar cortex and inferior olivary nucleus (Finch, Nicolson, & Fawcett, 2002), whereas the medial geniculate nucleus (MGN) of thalamus has been found to show an opposite pattern of fewer large neurons and more small neurons in left hemisphere (Galaburda, Menard, & Rosen, 1994). In animal studies this type of difference in the cell sizes of the MGN has been induced in male rats by microgyria and post-operation rats have shown deficient fast auditory temporal processing as a result of the injury (Herman, Galaburda, Fitch, Carter, & Rosen, 1997). This kind of processing deficit has been suggested to play a critical role in reading problems (Tallal, 1980) and will be discussed later in detail. Another deviation reported in the MGN of dyslexics is smaller neurons in this area in the left hemisphere as compared to the right hemisphere (Galaburda, Menard, and Rosen, 1994). No such asymmetry was found in controls (Galaburda, Menard, & Rosen, 1994).

Smaller size of neurons in magnocellular layers of another thalamic nucleus, lateral geniculate nucleus (LGN), has been similarly reported in participants with dyslexia (Livingstone, Rosen, Drislane, & Galaburda, 1991; Galaburda & Livingstone, 1993). This finding together with the discovery of lack of typical leftward neuronal size asymmetry of the primary visual cortex (Jenner, Rosen, & Galaburda, 1999) are of special interest when considering the theory of magnocellular function deficit in participants with dyslexia. Galaburda and colleagues have speculated that the phonological abnormalities found in participants with dyslexia might result from abnormal development of the left hemispheric auditory system, which is due to neuronal migration disturbances caused by abnormal gene function and produces deficiencies especially in the subsystems processing rapid temporal transitions (Galaburda, 1995; Galaburda, Menard, & Rosen, 1994). The magnocellular theory will be further discussed later.

In addition to the post-mortem studies several other brain imaging methods (MRI, fMRI, PET, MEG, EEG) have revealed different kinds of abnormalities in both structural and activation properties of the brains of individuals with reading problems. Structural differences have been found both in cerebral cortex (Brown et al., 2001, Clark & Plante, 1998; Eckert et al., 2003; Eliez et al., 2000; Leonard et al., 2001; 2002) as well as in deeper structures, such as thalamus (Brown et al., 2001) and cerebellum (Brown et al., 2001; Eckert et al., 2003). Studies of planum temporale have produced contrasting results: while some studies have confirmed the postmortem findings of Galaburda and colleagues (1985; 1989) of lack of typical leftward asymmetry in participants with reading disorder (Hynd, Semrud-Clikeman, Lorys, Novey, & Eliopulos, 1990; Larsen, Høien, Lundberg, & Odegaard, 1990), other studies have found this group of participant to show normal asymmetry (Best & Demb, 1999; Heiervang et al., 2000; Robichon, Levrier, Farnarier, & Habib, 2000; Rumsey et al., 1997a; Schultz et al., 1994) or even exaggerated leftward asymmetry of planum temporale (Leonard et al., 1993; 2001; 2002). The finding of extreme leftward asymmetry of planum in participants with dyslexia may seem puzzling. One reason for the contrasting findings might be differences in the measurement techniques. Another reason might be differences in the participants under study. According to Leonard and colleagues (Leonard, 2001; Leonard et al., 2001) planar symmetry is typical only for participants with reading problems who show poor verbal ability or delayed language development, whereas marked leftward planar asymmetry is seen in participants with poor phonological skills only. In contrast, in the study of Larsen, Høien, Lundberg, and Odegaard (1990) especially participants with pure phonological deficits showed planum symmetry. However, there are also other possibly affecting differences in the results, for example native language and comorbid disorders of the participants.

Reduced activity has been most often found in left temporoparietal regions (Breier et al., 2003; Brunswick, McCrory, Price, Frith, & Frith, 1999; Georgiewa et al., 1999; Meyler et al., 2007; Paulesu et al., 1996; 2001; Rumsey et al., 1992; 1997b; Schulz et al., 2008; Simos et al., 2000; Shaywitz et al., 1998; 2002; Temple et al., 2001), occipitotemporal areas (Helenius, Tarkiainen, Cornelissen, Hansen, & Salmelin, 1999; Kronbichler et al., 2006; McCrory, Mechelli, Frith, & Price, 2005; Schulz et al., 2008; Shaywitz et al., 1998; 2002), and Broca's area (Georgiewa et al., 1999), although also increased activity has been reported in left inferior frontal gyrus (Kronbichler et al., 2006; Shaywitz et al., 1998) and more specifically in Broca's area (Brunswick et al., 1999; Georgiewa et al., 2002; Kronbichler et al., 2006) as well as in right temporoparietal region (Simos et al., 2000). The reported hyperactivities might be related to greater effort invested in language tasks and compensatory mechanisms in participants with reading problems (Georgiewa et al., 2002; Shaywitz et al., 1998). The EEG study of Penolazzi, Spinorelli, and Angrilli (2008) suggested greater left anterior delta amplitude in dyslexic participants to index inhibition of left frontal linguistic areas. The left temporoparietal areas and Broca's area seem to be related to phonological processing (Démonet, Fiez, Paulesu, Petersen, & Zatorre, 1996; Moore & Price, 1999; Pugh et al., 1996; Simos et al., 2002; Zeffiro & Eden, 2000), whereas the occipitotemporal areas are suggested to be more involved in the fast automatic orthographic processing of the whole word (Cohen et al., 2002; 2003; Dehaene et al., 2001; Dehaene, Le Clec'H, Poline, Le Bihan, & Cohen, 2002; McCandliss, Cohen, & Dehaene, 2003; Pugh et al., 1996).

Reading problems have been also portrayed as a disconnection syndrome, which refers to the possibility that participants with reading problems might experience weak connections between the frontal and posterior brain areas (Paulesu et al., 1996). PET studies of weaker correlations in the activity between the left angular gyrus and inferior frontal cortex (Horwitz, Rumsey, & Donohue, 1998), and left angular gyrus and occipitotemporal areas (Horwitz, Rumsey, & Donohue, 1998; Pugh et al., 2000) in participants with dyslexia have supported the idea of connection disruptions in this subject group. More support comes from diffusion tensor magnetic resonance imaging (DTI) studies showing disruption in temporoparietal white matter in participants with reading problems, especially in the left hemisphere (Deutsch et al., 2005; Klingberg et al., 2000; Steinbrink et al., 2008). The brain connectivity in the left temporoparietal white matter as measured by DTI has also been found to correlate with reading ability (Beaulieu et al., 2005; Deutsch et al., 2005; Steinbrink et al., 2008) Since white matter plays a central role in connecting different brain areas, this kind of disruption could explain the functional connection problems found in participants with reading problems.

In addition to the intrahemispheric connections differences have also been found in the interhemispheric connections in participants reading problems. MRI studies have found differences in the size and shape of corpus callosum, the longitudinal fissure connection left and right hemisphere in participants with reading disorder (Duara et al., 1991; Hynd et al., 1995; Robichon & Habib, 1998; Rumsey et al., 1996; von Plessen et al., 2002). Also a DTI study has shown a correlation between the diffusion properties of the temporal-callosal fiber tract of corpus callosum and phonological awareness, which might be due to higher proportions of large axons in the good readers (Dougherty et al., 2007). This kind of a deficit might affect the hemispheric specialization due to reduced communication between the hemispheres (Dougherty et al., 2007). The DTI studies of disruptions in intra- and interhemispheric connections might explain the problems of participants with reading disorder in processing fast visual and auditory information: The larger axons in corpus callosum and intact myelination help carry signals faster and more effectively. However, some studies have not found differences in the corpus callosum in participants with reading disorder (Larsen, Høien, & Ødegaard, 1992; Penninton et al., 1999). The contrasting findings make it difficult to draw clear conclusions about the neuroanatomical differences between participants with reading problems and controls. Leonard (2001) has stressed the fact that participants with reading disabilities do not form a homogeneous group, and the neuroanatomical findings may depend on the specific type of reading disability.

1.1.2 Auditory processing and reading disorder

Although most researchers agree on the central role of phonological processing in reading problems, there are different opinions of the origin of these problems in reading disorder. Some researchers think that phonological problems might be caused by problems in basic auditory processing (Farmer & Klein, 1995; Goswami et al., 2002; Talcott et al., 2000; Tallal, 1980; Tallal & Benasich, 2002; Tallal, Sainburg, & Jernigan, 1991), whereas according to others reading problems are related to language processes, and basic auditory processing deficiencies are not the cause of phonological problems (Blomert & Mitterer, 2004; Brady, Shankweiler, & Mann, 1983; Mody, Studdert-Kennedy, & Brady, 1997). Auditory processing has been widely studied in participants with reading problems.

Participants showing reading problems have been found to perform poorly in several types of auditory processing tasks. Lower than normal performance has been found for example in temporal order judgment (TOJ) tasks demanding participants to perceive the order of rapidly presented complex tones differing in fundamental frequency (Heiervang, Stevenson, & Hughdahl, 2002; Nagarajan et al. 1999; Reed, 1989; Tallal, 1980), tone frequency discrimination tasks (Ahissar, Protopapas, Reid, & Merzenich, 2000; De Weirdt, 1988; Heirvang, Stevenson, & Hughdahl, 2002), frequency- (Witton et al., 1998; Witton, Stein, Stoodley, Rosner, & Talcott, 2002) and amplitude modulation tasks (Goswami et al., 2002; Witton, Stein, Stoodley, Rosner, & Talcott, 2002), and auditory localization tasks (Hari, & Kiesilä, 1996). In the study of Ahissar and colleagues (2000) adults with childhood history of reading difficulties still showed problems in auditory processing abilities tested with several types of tasks, although their reading skills were higher than expected on the basis of the auditory processing skills. Thus it seems that compensating reading skills do not necessary affect the auditory deficit.

Auditory processing problems have also been found in studies applying language stimuli. Participants with reading problems have been found to have difficulties in for example categorical speech perception (Breier, Fletcher, Denton, & Gray, 2004; De Gelder & Vroomen, 1998; Steffens, Eilers, Gross-Glenn, & Jallad, 1992). In the study of Breier and colleagues (2004) children showing weak language skills in the beginning of first grade showed poor auditory categorical discrimination of tokens crossing phonemic boundary (/ga/-/ka/). Authors suggested deficient categorical perception to affect the process of extracting phonetic information from speech, and cause more attention being directed to uninformative subtle acoustic differences. Categorical phonemic discrimination was also found to be associated with phonological processing skills (Breier et al., 2004), which might implicate the importance of categorical perception abilities in developing efficient language processing. Steffens, Eilers, Gross-Glenn, and Jallad (1992) found adults with dyslexia to need longer silence duration between two speech stimuli (/sa/-/sta/) in order to perceive a change in the stimuli. Also the study of De Gelder and Vroomen (1998) found deficient categorical speech perception (/ba/-/da/) in children with poor reading skills.

Similar results of deficient categorical speech perception have been reported in adults with dyslexia and their infants who are at-risk for reading problems (Richardson, 1998; Richardson, Leppänen, Leiwo, & Lyytinen, 2003). In these studies categorical perception was investigated by pseudoword stimuli with varying durations (/ata/-/atta/). Duration is a crucial semantic feature in Finnish language that can distinguish two otherwise similar words. For example '*mato*' means worm in Finnish, wheras '*matto*' is the word for carpet. Quantity errors are the most typical error type made by Finnish speaking individuals with deficient reading skills (Lyytinen et al., 1995).

1.1.2.1 Rapid auditory processing theory

According to the studies of Tallal and colleagues (Tallal, 1980; Tallal & Piercy, 1974; 1975; Tallal & Stark, 1981), participants with language problems have a basic auditory processing problem, which shows as a disability in processing rapidly presented auditory stimuli. In the studies of Tallal and colleagues participants had to either discriminate two stimuli (same/different task) or judge the temporal order (temporal order judgment task, TOJ) of two stimuli. The stimuli were either complex tones varying in fundamental frequency, synthetic syllables, or synthetic steady-state vowels. Although several studies of Tallal and colleagues were executed with participants with more severe language problems, in 1980 Tallal found children with RD to experience similar problems. Reed (1989) and Farmer and Klein (1993) provided further support of auditory processing problems observed in TOJ tasks in children with RD.

Brain activation abnormalities in response to rapid stimuli have been also reported. The MEG study of Nagarajan and colleagues (1999) found adult participants with poor reading abilities to show reduced auditory cortex responses to brief and rapidly successive auditory stimuli in addition to the poor behavioral performance in TOJ task. Temple and colleagues (2000) applied fMRI to study the brain activity during rapidly and slowly changing, nonlinguistic stimuli. They found normal adult readers to show left prefrontal activity in response to the rapidly changing stimuli relative to the slowly changing stimuli, whereas no such differential left frontal response was observed in participants with dyslexia. The left prefrontal response also correlated with performance in behavioral rapid processing task, and two of three subjects participating in rapid auditory processing training showed increased left prefrontal activity as well as improvement in behavioral performance. These results were recently replicated in children by Gaab, Gabrieli, Deutsch, Tallal, and Temple (2007).

Tallal (1980; 2000) has suggested that the fast temporal auditory processing deficit might possibly cause problems in speech perception and in formation of phonological representations, which in turn leads to reading problems. Indeed, performance in auditory temporal processing tasks has been found to correlate with reading measures (Au, & Lovegrove, 2001; Tallal, 1980; Walker, Hall, Klein, & Phillips, 2006), although some studies have not been able to find a strong relationship between these skills (Heath, Hogben, & Clark, 1999; Heiervang, Stevenson, & Hugdahl, 2002; White et al., 2006b). Further support for Tallals theory comes from intervention studies suggesting auditory training to improve the performance in tasks demanding discrimination of nonspeech and speech stimuli (Merzenich, et al., 1996; Tallal et al., 1996). Tallal's view of basic auditory processing deficit as a cause of reading problems has been however challenged by researchers who have not been able to replicate her results (Breier, Gray, Fletcher, Foorman, & Klaas, 2002; Mody, Studdert-Kennedy, & Brady, 1997).

1.1.2.2 The magnocellular theory and rapid processing

The magnocellular theory has been used as an explanation for the processing deficits found in participants with dyslexia. Visual system can be divided to two pathways, magnocellular and parvocellular, based on their sensitivity to visual features. Parvocellular pathway is sensitive to form and color and has poor temporal but good spatial resolution, whereas magnocellular has good temporal but poor spatial resolution and is sensitive to contrasts, direction and movement. The magnocellular and parvocellular pathways orginate in retina and project to separate layers in the lateral geniculate nucleus (LGN) of the thalamus (Livingstone & Hubel, 1987). From the LGN the visual information is transmitted to magnocellular and parvocellular sublayers and to visual cortex (Livingstone & Hubel, 1987).

Several studies have shown participants with reading problems to experience problems in visual tasks (e.g. contrast sensitivity, flicker sensitivity, motion detection) taxing the magnocellular pathways (Ben-Yehudah, Sackett, Malchi-Ginzberg, & Ahissar, 2001; Borsting et al., 1996; Cornelissen, Hansen, Hutton, Evangelinou, & Stein, 1998b; Cornelissen et al 1998a; Cornelissen, Richardson, Mason, Fowler, & Stein, 1995; Demb, Boynton, Best, & Heeger, 1998; Eden, VanMeter, Rumsey, & Zeffiro, 1996; Felmingham & Jakobson, 1995; Johnston et al., 2008; Lovegrove, Bowling, Badcock, & Blackwood, 1980; Lovegrove et al., 1982; Martin & Lovegrove, 1984; Martin & Lovegrove, 1987; Ridder, Borsting, Cooper, McNeel, & Huang, 1997; Slaghuis & Ryan, 1999; Talcott et al., 1998; Witton et al., 1998), and to show smaller amplitudes of visually evoked potentials (VEP) in these tasks (Livingstone et al., 1991; May, Lovegrove, Martin, & Nelson, 1991). Also abnormal activation in fMRI studies has been reported: During visual motion processing tasks less activation has been found in primary visual cortex (V1) and extrastriate visual areas (V2, V3, V3A, V4v, V5/MT) (Demb, Boynton, & Heeger, 1998; Eden et al., 1996). The study of Demb, Boynton, and Heeger (1998) also found the measured brain activity to be correlated with motion discrimination performance and reading speed. However, these results were not supported by the study of Vanni, Uusitalo, Kiesilä, and Hari (1997). In a post-mortem study reduced cell size in the magnocellular layers of the LGN in participants with dyslexia has been reported (Livingstone et al., 1991).

Cornelissen and colleagues (1998a; 1998b) found a positive relationship between children's motion detection thresholds and the likelihood of making letter errors, which lends further support to the role of magnocellular processing in reading skills. According to Stein and Walsh (1997) the effects of magnocellular impairments to reading problems might be due to anatomical connections from the magnocellular laminae of the LGN to the posterior parietal cortex, which is important for normal eye-movement control, visuospatial attention and peripheral vision. Also other researchers have suggested that the reading problems are due to the role of the magnocellular pathway in attention allocation required in reading (Hari, Valta, & Uutela, 1999; Laycock, & Crewther, 2008; Omtzigt, Hendriks, & Kolk, 2002)

It has been suggested that auditory system shows a deficit similar to the visual magnocellular deficit (Stein & Walsh, 1997). Indeed, the magnocells of the medial geniculate nucleus have been found to be abnormal in dyslexic brain (Galaburda et al., 1994). Stein and Walsh proposed that the magnocellular temporal processing deficit might affect vision and audition and also for example vestibular and motor systems, which might explain the co-occurrence of motor problems in participants with reading disorder. Boets, Wouters, van Wieringen, De Smedt, and Ghesquiére (2008) found evidence of a global magnocellular model in a study assessing dynamic auditory and visual processing, speech-in-noise perception, phonological ability and orthographic ability in preschool children. The model connects dynamic auditory processing to speech perception, which is related to phonological awareness, whereas dynamic visual processing is connected to orthographic skills. Finally, phonological awareness and orthographic ability together with verbal short-term memory predict literacy development.

Some studies have not found evidence of a magnocellular deficit in participants with reading disorder (Hayduk, Bruck, & Cavanagh, 1996; Spinelli et al., 1997; Walther-Müller, 1995). Amitay, Ahissar, and Nelken (2002) found only six of 30 participants with RD to be impaired in tasks of magnocellular functions, and these subjects were consistently impaired in several other perceptual tasks as well. The rest of the participants with RD did not differ significantly from controls in magnocellular tasks but showed deficient performance in tasks requiring fine visual and auditory frequency discrimination. Amitay and colleagues (2002) concluded that some participants with RD have generally impaired perceptual skills, whereas some have more specific perceptual deficits. Ramus and colleagues (2003) provided similar results of relatively low incidence (2 of 16) of magnocellular deficit in participants with dyslexia. Magnocellular deficits have been also found in participants with other types problems, which suggests that these problems are not specific to participants with reading disorder (Skottun & Skoyles, 2008).

1.1.2.3 Evaluation of the rapid auditory processing theory

Although some participants with reading disorder seem to show rapid auditory processing problems, not all of them do. This was already noticed by Tallal (1980), and has been confirmed in other studies (Amitay et al., 2002; Bishop, Carlyon, Deeks, & Bishop, 1999; Boets, Wouters, van Wieringen, & Ghesquiére, 2007; Heath, Hogben, & Clark, 1999; Ramus et al., 2003; White et al., 2006a). Similarly, there are normally reading participants with deficient sensory processing (Bishop et al., 1999; Boets et al., 2007). Twin studies have shown sensory skills not to be as strongly genetically influenced as phonological skills (Bishop et al., 1999; Olson & Datta, 2002). This seems also to point to the conclusion that phonological skills are affected also by other things than pure sensory abilities. Thus, rapid auditory processing problems can not be considered neither a necessary nor the sole cause of reading problems. Instead auditory processing problems have been suggested to act as moderators compromising already poor reading skills (Bishop et al., 1999), co-occuring with reading disability and possibly aggravating phonological problems (Ramus et al., 2003), or the phonological problems have even been proposed to affect negatively to basic sensorimotor skills (White et al., 2006b).

Amitay and colleagues (2002) argued that instead of a rapid auditory processing deficit reading problems might be caused by accumulative result of different kinds of auditory processing deficits that are not restricted to tasks demanding fast stimulus processing. Their study applying several kinds of auditory tasks with a wide range of stimulus constants failed to find a single common impairment in all of the participants with RD although significant portion of the RD group had auditory difficulties of some kind.

According to Mody, Studdert-Kennedy, & Brady (1997) and Mody (2003) participants with reading problems experience difficulties in discriminating phonetically similar stimuli instead of rapid auditory processing problems and consider the deficit to be purely linguistic. In the study of Mody, Studdert-Kennedy, and Brady (1997) participants with poor reading skills did not differ from controls when easily discriminable syllables were used. Also Banai and Ahissar (2006) have stated that the auditory processing deficits found in participants with RD are actually due to task complexity. They showed task complexity to affect the performance of participants with dyslexia and additional learning disabilities in tasks demanding frequency and phoneme discrimination.

Another interesting alternative explanation for the auditory discrimination problems in participants with dyslexia was recently proposed by Ahissar (2007). He suggested that these participants with dyslexia have an anchoring deficit, which causes problems in retaining and retrieving recently presented stimuli. According to the hypothesis, perception of repeated stimuli leads to the formation of stimulus anchors that are used to predict future stimuli and to avoid distractions caused by irrelevant stimuli. However, in participants with dyslexia the anchoring system does not work normally and the perceptual system is more sensitive to external noise. Thus, in the case of auditory discrimination studies controls would benefit from the repetition of the same stimuli, whereas no such benefit would be present in participants with dyslexia. The study of Ahissar, Lubin, Putter-Katz, and Banai (2006) using varying reference tones instead of a repeated reference in frequency discrimination task support the theory but more research is needed to test the theory using different stimulus types and experimental conditions.

The inconsistency of the results concerning the auditory processing problems in participants with dyslexia may be due to many factors. One is the heterogeneity of the subject groups. The definitions of the reading problems and diagnostic procedures vary from one study to another. Also, languages differ in many ways and reading problems do not appear exactly the same way in participants speaking different languages (Grigorenko, 2001; Landerl, 1997). Since the demands and critical features vary, it seems possible that different auditory problems might disturb normal development of reading skills in different languages. It has been also argued that the behavioral auditory processing found in participants with reading disorder might be caused by nonperceptual factors, like attention and motivation (Bishop, 2007). Due to the poor verbal skills it might be harder for these participants to develop effective listening strategies in psychoacoustic tasks and thus it might be harder for them to sustain attention and to be motivated (Bishop, 2007). Since attention problems often co-occur with reading problems it is important to take these factors into account.

1.1.3 Comorbidity of reading disability and attention deficit

Attention-deficit hyperactivity is a neurodevelopmental disorder characterized by impulsivity, inattention and often also by overactivity (American Psychiatric Association (APA), 2000: Diagnostic and Statistical Manual of Mental Disorders, DSM-IV-TR). Attention deficit can, however, also appear without overactivity as an inattentive type. In brain imaging studies (MRI, EEG) especially dysfunction in the frontostriatal circuits connecting frontal lobe with basal ganglia has been often reported (Willis, &Weiler, 2005 for a review). Attention deficits are relatively common affecting approximately 5% of population (Lambert, Sandoval, & Sassone, 1978; Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007). The prevalence seems to be approximately the same worldwide although varying diagnostic practise may cause differences in prevalence estimates (Faraone, Sergeant, Gillberg, & Biederman, 2003; Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007).

In a Finnish study nearly 20% of children with reading disability showed symptoms of ADHD as well (Lyytinen et al., 1998). Also other studies have found attention problems and reading problems to often coexist (Dykman & Ackerman, 1991; Knivsberg & Andreassen, 2008; Willcutt & Pennington, 2000a&b). According to the study of Knivsberg and Andreassen (2008) attention problems are not often thoroughly assessed in children showing reading problems. In part the high comorbidity seems to be due to common genetic influences (Gayán et al., 2005; Willcutt et al., 2002).

Participants with problems both in reading and attention seem to show the typical cognitive deficits linked to both types of problems. Studies of Willcutt and colleagues (2001, 2005) as well as Purvis and Tannock (1997; 2000) found both phonological and inhibition problems in participants experiencing comorbid attention and reading problems. However, the studies of Bental and Tirosh (2007) and Toplak, Rucklidge, Hetherington, John, and Tannock (2003) proved that the participants with both problem types may differ from the "pure" groups and show for example more severe deficits in working memory. This highlights the importance of clearly defined participant groups as well as comparing the groups with "pure" and mixed disorders. In addition to the phenotypical problems of phonological processing in individuals with reading problems and disrupted executive functions in individuals with attention deficiencies, processing speed has been suggested as a cognitive risk factor shared by both groups (Shanahan et al., 2006; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005)

Since reading disability and attention deficit often co-occur, differential diagnostics and careful investigation of both types of problems is important. Unfortunately the characteristic symptoms of attention deficit make it relatively hard to reliably estimate the reading skills of participants with such problems. Similarly, learning problems like reading disorder may cause distress and restlessness in participants in situations demanding the use of the deficient skills, which may be faultily interpreted as attention problems. Thus, methods that do not demand behavioral response and active attention allocation might be useful tools in the differential diagnostics of attention problems and learning disabilities. A good recent example of such effort is the EEG study of Dhar, Been, Minderaa, and Althaus (2008) who found participants with reading problems to show problems during covert orienting task in early information processing (smaller N2), whereas those with attention deficit displayed abnormalities only in later, response-related processing stages (larger P3).

The deficient performance in auditory tasks reported in participants with RD could be caused by problems in attention and motivation (Bishop, 2007). In behavioral tasks the role of attention is hard to estimate. In order to study auditory processing without such confounding effects, event-related potentials (ERPs) can be used. One of the advantages of brain measurements in providing further information about auditory processing is that children participating do not have to pay attention to the task, and thus the role of attention can be ruled out as one of the main factors affecting the results.

1.2 Mismatch negativity and auditory processing

Mismatch negativity (MMN) is an event-related potential (ERP) that occurs when a deviant sound occurs among repeated standard stimuli and breaks the auditory regularities of the auditory stream (Näätänen, Gaillard, & Mäntysalo, 1978). The deviation can occur in different stimulus parameters, such as frequency, intensity, duration, or sound location. MMN can also be elicited by changes in phonemes or even by violations in abstract stimulus patterns (Paavilainen, Arajärvi, & Takegata, 2007; Tervaniemi, Maury, & Näätänen, 1994). The MMN response is elicited approximately 100-350 ms, but there have been reports of a second mismatch related peak called late discrimination negativity (LDN) around 400-600 ms (Ceponiené, Cheour, & Näätänen, 1998; Ceponiené et al., 2002; Ceponiené et al., 2004; Kilpeläinen, Partanen, & Karhu, 1999; Korpilahti, Krause, Holopainen, & Lang, 2001; Martynova, Kirjavainen, & Cheour, 2003; Schulte-Körne, Deimel, Bartling, & Remschmidt, 2001). According to Ceponiené and colleagues (2004) LDN reflects preattentive sound change processing at a more cognitive level than MMN.

Amplitude of the MMN is around -3µV (Licht & Horsley, 1998) and it consists of frontal and central subcomponents (Alho, 1995; Giard et al., 1990; Näätänen & Michie, 1979). The central component is generated in the auditory cortex, and the frontal subcomponent is suggested to be related to involuntary attention switch due to the deviant stimuli (Giard et al., 1990). The MMN scalp distribution is slightly different for different stimulus features (Giard et al., 1994).

The elicitation of MMN is thought to be based on a neuronal process in which the deviating stimulus is compared to the memory trace formed by the repetition of the standard stimuli (Näätänen, 1992), and thus the time elapsing between the standard and the deviant stimulus is crucial for the MMN elicitation. In addition to the increase of the magnitude of the deviation and decrease of the probability of the deviant occurrence, also shortening of the interstimulus interval (ISI) causes larger MMN. MMN has been elicited in adults with 10 second ISI (Sams, Hari, Rif, & Knuutila ,1993). In children the duration of the memory trace has been found to prolong: MMN can be elicited with 1-2 seconds ISI in children of 2 and 3 years, with more than 2 seconds ISI in 4-year-olds and 3-5 secons in 6-year-olds (Glass, Sachse, & von Suchodoletz, 2008).

MMN tends to overlap with another negative peak, N1, which appears in response to any notable sound change. It has been even proposed that MMN is actually a temporally modulated N1 response that is suppressed and delayed by stimulus specific adaptation (Jääskeläinen et al., 2004). However, this theory has been refuted by studies showing for example that MMN can be elicited even with stimulus omissions (Oceák, Winkler, Sussman, & Alho, 2006; Yabe et al., 1998), the latencies, scalp distribution as well as generator loci of MMN and N1 differ (Näätäläinen, Paavilainen, & Reinikainen, 1989; Winkler, Tervaniemi, & Näätänen, 1997; Alho, 1995; Korzyukov et al., 1999; Opitz, Schröger, & von Cramon, 2005) and that there is a difference in the effect of N1 and MMN on memory disruption (for a review of the evidence, see Näätänen, Jacobsen, & Winkler, 2005).

MMN can be recorded in children (Kraus et al., 1993; Ceponiené, Cheour, & Näätänen, 1998; Gomot, Giard, Roux, Barthelemy, & Bruneau, 2000; Korpilahti et al., 2001; Cheour et al., 2002b) and even in infants (Alho, Sainio, Sajaniemi, Reinikainen, & Näätänen, 1990; Cheour et al., 1998a; Cheour et al., 1998b; Cheour-Luhtanen et al., 1996; Pang et al., 1998; Trainor, Samuel, Desjardins, & Sonnadara., 2001; Ceponiené et al., 2002; Cheour et al., 2002a; Kushnerenko, Ceponiené, Balan, Fellman, & Näätänen, 2002; Martynova, Kirjavainen, & Cheour, 2003). However, some infant studies have found a positive response to deviations instead of a MMN (Benasich et al., 2006; Leppänen, Eklund, & Lyytinen, 1997; Leppänen, Pihko, Eklund, & Lyytinen, 1999; Pihko et al., 1999). The findings of positive response to auditory stimulus change have been explained by greater positivity elicited by the deviant than the standard sound due to the excitement of new afferent elements (Leppänen, Eklund, & Lyytinen, 1997). MMN of school-age children seems to resemble that of adults, although some developmental changes can still be seen (Gomot et al., 2000; Kraus, Koch, McGee, Nicol, & Cunningham, 1999; Shafer, Morr, Kreuzer, & Kurtzberg, 2000).

MMN elicitation does not demand attention, which makes MMN studies much easier to conduct with different patient groups and especially children. MMN has been even elicited during sleep (Campbell, Bell, & Bastien, 1991; Martynova, Kirjavainen, & Cheour, 2003; Nashida et al., 2000; Sallinen, Kaartinen, & Lyytinen, 1994). Although MMN elicitation does not depend on attention (Alho, Woods, & Algazi, 1994; Näätänen, 1992; Näätänen, Paavilainen, Tiitinen, Jiang, & Alho, 1993), it can affect MMN amplitude in experiments that present competition for the processing capacities (Szymanski, Yund, & Woods, 1999; Woldorff, Hackley, & Hillyard, 1991; Sabri, Liebenthal, Waldron, Medler, & Binder, 2006). Attention also seems to affect differently MMNs in adult and child participants: Gomes and colleagues (2000) found attention to affect the amplitude of frequency MMNs in child participants when comparing ignore and attend conditions, whereas no such effect was found in adults. It may be that difficult auditory discrimination demands attention resources in children, whereas the process becomes more automatic over the course of development.

MMN amplitude has been shown to increase with discrimination training (Kraus et al., 1995; Kujala et al., 2001; Näätänen, Schröger, Karakas, Tervaniemi, & Paavilainen, 1993; Tremblay, Kraus, Carrell, & McGee, 1997; Stack & Dalebout, 1998), which suggests that MMN elicitation is related to participants' ability to behaviorally discriminate between presented standard and deviant sounds. Although some studies have found behavioral discrimination ability to correlate with MMN (Kraus et al., 1996; Novitski, Tervaniemi, Huotilainen, & Näätänen, 2004; Pakarinen, Takegata, Rinne, Huotilainen, & Näätänen, 2007), not all of them have found a clear connection between the behavioral and electrophysiological discrimination measures (Paavilainen, Arajärvi, & Takegata, 2007; Paul, Bott, Heim, Wienbruch, & Elbert, 2006; Sharma et al., 2006; Uwer, Albrecht, & von Suchodoletz, 2002).

MMN is affected by the familiarity of speech stimuli used for its elicitation. If a phoneme is a prototype of subjects' native language MMN tends to be larger as compared to a phoneme that is not a prototype of the language (Cheour et al., 1998b; Näätänen et al., 1997; Tervaniemi et al., 2006; Ylinen, Shestakova, Huotilainen, Alku, & Näätänen, 2006). In the study of Tervaniemi and colleagues (2006) speakers of Finnish showed significantly larger MMNs to duration changes as compared to German speaking subjects, whereas no differences in the MMNs elicited by frequency changes were found. The same subjects also differed in the behavioral duration discrimination to the advantage of Finnish speaking subjects. In the study of Ylinen, Shestakova, Huotilainen, Alku, & Näätänen (2006) native speakers of Finnish showed enhanced MMN amplitudes in response to change in vowel duration when compared to both non-native speakers of Finnish and non-Finnish-speaking participants. Similarly, native speakers performed better in the behavioral discrimination of the phonological quantity. It has been also shown that when learning a new language cortical memory representations of the phonemes of that language are formed and MMN can be elicited by the phoneme contrasts of the new language (Cheour et al., 2002b; Winkler et al., 1999). The language specific memory traces affecting MMN formation seem to emerge in infants already during the first 12 months of life (Cheour et al., 1998b). Thus it seems that experience of certain type of stimulus features can affect MMN elicitation.

1.2.1 Mismatch negativity and reading disorder

One of the first studies to encourage researchers to use MMN as a tool for studying learning disabilities was conducted by Kraus and colleagues (1996). In their study children with learning disabilities were studied with both MMN and behavioral discrimination task. Kraus and colleagues found that the children with poor behavioral discrimination of synthetic speech stimuli (/da/-/ga/) also showed smaller MMNs than children with good discrimination skills. MMN measures and behavioral discrimination scores were also significantly correlated.

Several studies have found the MMN to be attenuated in participants with reading disorder. Differences have been found for example in studies applying changes in tone frequency (Baldeweg et al., 1999; Kujala, Belitz, Tervaniemi, & Näätänen, 2003; Kujala et al., 2006), duration (Corbera, Escera, & Artigas, 2006), tone omission (Fisher et al., 2006), and complex tone patterns (Kujala et al., 2000). Even children at familial risk for dyslexia have shown attenuated MMN to changes in tone frequency (Maurer, Bucher, Brem, & Brandeis, 2003), speech sound duration (Leppänen, Pihko, Eklund, & Lyytinen, 1999; Leppänen et al., 2002), and phonemes (van Leeuwen et al., 2006). However, some studies have found diminished MMNs in participants with reading disorder only when applying speech stimuli, whereas no impairment in responding to changes in tone stimuli was found (Schulte-Körne, Deimel, Bartling, & Remschmidt, 1998; 2001).

Because of its critical role in Finnish language it is interesting to study duration discrimination in Finnish participants with RD. Although several studies have examined duration discrimination with behavioral methods, few have investigated MMNs produced by tone duration changes, and the results vary widely (see table 1).

One of the first MMN studies applying changes in tone duration was executed by Korpilahti and Lang (1994). They studied children with dysphasia, who showed a diminished MMN elicited by a large duration change (50 vs 500 ms). However, Uwer, Albrecht, and von Suchodoletz (2002) did not find a significant difference in the MMNs between controls and children with specific language impairment (SLI) with a smaller duration change (175 vs 100 ms).

Similarly mixed findings have been reported in studies of participants with dyslexia. Baldeweg et al., 1999 (1999) studied both frequency and duration MMN in adult participants of this group. In their study only frequency changes showed significant differences in the MMNs of the controls and the clinical group. On the contrary, in the study of Corbera and colleagues (2006) children with dyslexia showed deficient tone duration processing. One factor affecting the discrepancy between the results of Corbera and colleagues (2006) and Baldeweg and colleagues (1999) might be the difference in the length of the stimuli. Baldeweg and colleagues (1999) applied significantly longer duration stimuli (standard 200 ms, deviants 160 ms, 120 ms, 80 ms & 40 ms) than Corbera and colleagues (2006) (standard 100 ms, deviant 33 ms).

The study of Kujala, Lovio, Lepistö, Laasonen, and Näätänen (2006) supported the negative findings of Baldeweg and colleagues (1999) of no significant differences in the MMNs of controls and adult participants with dyslexia. However, again the stimuli differed in an important way from those applied by Corbera and colleagues (2006): The differences between standards

TABLE 1 MMN studies using duration deviants in the study of participants with reading problems

Authors	Participants (diagnosis, age, language)	Stimuli (S:Standard D:Deviant)	Main findings
Korpilahti, & Lang (1994)	Dysphasia 7-13 years Finnish	Tone duration change S: 50 ms D: 110/ 500 ms	Dysphasic children had a significantly smaller MMN in response to the more deviant sound (500 ms), whereas the MMNs to the smaller duration deviant did not differ between the groups.
Baldeweg et al. (1999)	Dyslexia adults English	Tone duration change S: 200 D: 40/ 80/ 120/ 160 ms	No significant difference between the participants with dyslexia and controls.
Schulte-Körne et al. (1999)	Dyslexia adults German	Duration change in tones of a 4-tone pattern S: 50-90-25-50 ms D: 50-50-25-90 ms	Attenuated MMN in participants with dyslexia in a late time window (250-600 ms)
Uwer, Albrecht, & von Suchodoletz (2002)	Specific language impairment (SLI) 5-10 years German	Tone duration change S: 175 ms D: 100 ms	No significant differences between the participants with SLI and controls.
Corbera et al. (2006)	Dyslexia ~11 years Spanish	Tone duration change S: 100 ms D: 33 ms	Attenuated MMN in participants with dyslexia
Kujala et al. (2006)	Dyslexia adults Finnish	Tone duration change in 5-deviant and oddball tasks S: 100 ms, D: 65 ms S: 50 ms, D: 33 ms	No significant differences between the participants with dyslexia and controls.

and deviants were smaller (100 vs 65 ms or 50 vs 33 ms) than of Corbera and colleagues (2006) showing significant differences between the controls and the participants with dyslexia (100 vs 33 ms).

One other study in addition to the one reported by Corbera and colleagues (2006) providing evidence of a diminished duration MMN in participants with reading problems is the study by Schulte-Körne, Deimel, Bartling, and Remschmidt (1999). This study, however, applied duration changes appearing in a 4-tone pattern (standard 50-90-25-50 ms, deviant 50-50-25-90 ms) instead of a change in a single pure tone. According to the authors the study implicates a problem in temporal information processing, whereas the tone processing per se might be normal in participants with dyslexia. The behavioral studies have similarly produced both positive (Banai & Ahissar, 2006; Thomson, Fryer, Maltby, & Goswami, 2006) and negative (Baldeweg et al., 1999) results concerning a duration discrimination deficit in children with reading problems.

Some MMN studies have also found Late Discriminative Negativity (LDN) arising at 400-500 ms to be diminished in subjects with reading disorder (Schulte-Körne et al., 1998; 1999; Alonso-Búa, Díaz, & Ferraces, 2006). In the study of Alonso-Búa and colleagues (2006) mismatch responses were elicited with both syllable (/ba/ vs /da/) and frequency changes. Only LDN was found to be decreased in participants with RD, whereas no significant MMN amplitude difference in comparison to controls was observed. However, the latencies of both MMN (syllables) and LDN (both syllables and tones) were significantly delayed in participants with RD. In addition, Alonso-Búa and colleagues reported differences in the lateralization of LDN: the LDN elicited by linguistic stimuli was significantly larger over the right hemisphere in the group with RD.

Sharma and colleagues (2006) used both MMN and behavioral tasks to study auditory processing in control children and children with reading problems as well as in compensated readers. The behavioral auditory processing tasks included both tone and speech stimuli, and in the MMN measurements tone, chord, and speech stimuli were used. All participants with RD failed at least one of the behavioral auditory processing tests. Participants with RD also had significantly smaller MMN area evoked by speech stimuli (/da/ vs /ga/) than the control children. However, when applying the same speech and chord stimuli in behavioral task significantly poorer performance in participants with RD was only observed in chord discrimination. The behavioral and MMN results did not support each other at the individual level: about half of the children with RD had MMN present but showed poor behavioral discrimination and about half showed the opposite pattern.

The study of Kujala and colleagues (2006) also did not support the idea of a direct relationship between MMN and behavioral discrimination in participants with dyslexia. In their study changes in vowel durations embedded in syllable strings were used, and no difference in the MMN responses between participants with dyslexia and normal reading skills were found whereas the behavioral discrimination performance of the former group was significantly worse. Similarly Paul and colleagues (2006) did not find correlations between MMN and behavioral discrimination in participants with dyslexia when applying speech stimuli (/ba/-/da/). This study found the MMNs of children with dyslexia were not attenuated, whereas the participants of this group showed problems in behavioral discrimination as compared to controls.

It seems surprising that some studies have found MMN and behavioral discrimination to correlate, whereas some have not. Most likely in the behavioral tasks also many other processes in addition to the auditory ones play a role. One possible factor might be attention. Not all studies have provided information about the attention capacity of the participants. Another possible factor is the performance strategy. Shafer, Morr, Datta, Kurtzberg, and Schwartz (2005) reported a participant showing robust MMN but poor behavioral performance to have adopted fast but inaccurate strategy in the behavioral task. Shafer and colleagues (2005) did not report any specific screening test for attention deficit.

1.2.2 Auditory processing and attention deficit

MMN has not been as actively studied in participants with attention deficit as in those with reading disorder. The few studies available have not produced sound evidence of abnormal MMNs in this participant group. Studies of Winsberg, Javitt, Silipo, and Doneshka (1993), Winsberg, Javitt, and Shanahan/Silipo (1997), Oades, Dittmann-Balcar, Schepker, Eggers, and Zerbin (1996), and Kilpeläinen, Partanen, and Karhu (1999) used frequency changes to elicit MMN, whereas speech stimuli changes (/oy/-/ay/) were applied by Kemner and colleagues (1996). Winsberg and colleagues (1993) found hyperactive children to show diminished MMNs in their first study, but the difference may have been caused by the peak latency differences: hyperactive children showed a marked negativity later, outside the time window applied in scoring. In the second study by Winsberg and colleagues (1997) a larger sample of hyperkinetic children showed no difference in the MMNs. Kilpeläinen, Partanen, and Karhu observed only the second phase (at 300-500 ms) of a two-phasic MMN to be smaller in highly distractible children, whereas the first peak (at 220 ms) showed a nonsignificant tendency to be larger in this group. Also Oades and colleagues (1996) found frontal part of MMN to be insignificantly larger in children with ADHD. In the speech stimuli study of Kemner and colleagues (1996) children with ADHD displayes marginally but not significantly smaller MMNs. In addition, MMNs showed lateralization to the left hemisphere in participants with ADHD, whereas in the control group MMNs were right lateralized.

Based on the studies reviewed above it seems that abnormal MMNs have not been convincingly documented in participants with attention problems. The participant selection procedures were also not completely uniform. In the study of Kilpeläinen, Partanen, and Karhu (1999), for example, the participants were "highly distractible" children, and the criteria of high distractibility was based on two subtests (arithmetic and digit span) of Wechsler Intelligence Scale for Children –Revised (WISC-R). These subtests do not provide a direct measure of attention, but on the contrary, measure several other skills as well. Thus, it can be questioned whether the participants of this study had genuine attention problems. On the other hand, children in the study of Oades and colleagues (1996) had an ADHD diagnosis based on DSM-III-R criteria and were inpatients, which implicate that they had problems severely affecting their daily functions. Also studies of Winsberg and colleagues (1993; 1997) and Kemner and colleagues (1996) applied DSM criteria in the participant selection.

Although MMN studies have not documented deficient auditory processing in participants with attention problems, in behavioral studies duration discrimination has been suggested to be impaired in individuals with attention problems (Smith, Taylor, Rogers, Newman, & Rubia, 2002; Toplak, Rucklidge, Hetherington, John, & Tannock, 2003; Toplak, & Tannock, 2005). However, the problem in examining behaviorally the skills of participants with attention problems is their hardship in focusing on the task at hand, when they are easily distractible, impulsive and have hard time sustaining attention. Due to the typical features of attention problems, behavioral tasks always have the risk of under evaluating the skills of this participant group.

1.3 The objectives of the studies

The first goal was to study, whether children with reading disorder show diminished MMNs in response to duration change appearing in continuous tone. Since studies displaying participants with different native languages have produced disagreeing results of the duration MMNs in participants with RD, it was interesting to study this peak in Finnish-speaking participants, since duration is such a critical feature in this language. One possible explanation for the disagreeing results might be the differences in the demands of languages.

Second goal was to compare the duration discrimination abilities of participants with reading disorder, attention deficit, or both types of problems. In the first study, control participants were compared to participants with reading disorder and to those with attention deficit. However, also a high proportion of the attention deficit group participants had comorbid reading problems, which were considered possible confounding factors in the study. Due to this possibility, in the second and third study, the group selection criteria were made stricter and three clinical groups formed: participants with reading disorder, attention deficit, or both. This way it was possible to explorewhether the “pure” groups differ from the group with comorbid problems as has been suggested by previous studies (Bental, & Tirosh, 2007; Toplak, Rucklidge, Hetherington, John, & Tannock, 2003).

Third, although several studies have found either MMNs (Baldeweg et al., 1999; Corbera et al., 2006; Fisher et al., 2006; Kujala et al., 2000; 2003; 2006) or behavioral duration discrimination (Steffens, Eilers, Gross-Glenn, & Jallad, 1992; Watson, 1992) to be impaired in participants with reading disorder, rarely both types of tasks have been displayed on the same participants. In the third study the aim was to explore whether duration MMNs and behavioral speech stimuli duration discrimination are related, and whether either one or both of the tasks would show differences between participants with reading disorder, attention deficit, and both.

2 SUMMARY OF THE STUDIES

2.1 Study I

Two competing views have been suggested to explain the phonological problems observed in indiviuduals with reading disorder (RD). Some researchers have proposed the core of the problems to be at the basic level of auditory processing (Tallal, 1980; Tallal, Stark, Kallman, & Mellits, 1980; Tallal, Sainburg, & Jernigan, 1991; Farmer & Klein, 1995), whereas others have advocated the speech-specific view, according to which phonological problems are related to higher order linguistic processes. One relatively objective way to assess basic auditory processing abilities is to measure brain potential called mismatch negativity (MMN), which is elicited by deviations in the repeated homogeneous auditory stimuli. Since duration is a central feature in Finnish, duration discrimination is especially interesting. The objective of the first study was to investigate auditory processing of tone duration changes in a control group as well as in children with reading disability or attention deficit (AD) in order to explore whether the children with RD actually show a basic level auditory processing problems and whether the possible problems are specific to this group. The hypothesis was that children with RD would show such a deficit.

2.1.1 Methods

Auditory event-related potential, mismatch negativity (MMN), was used to study children (8-14 years). MMNs were elicited by a duration change in a continuous sound, which consisted of two alternating 100 ms tones of 600 and 800 Hz. The 600 Hz tones were in 15% of the tones replaced randomly by a shorter tone of either 30 ms or 50 ms. The experiment included 700 trials, and after the application of eye movement artefact rejection criteria ($\pm$ 60 μV) 650 trials on average were analyzed. In total, 114 children were tested, but after data and participant screening, each of the three groups (controls, children with RD, and children with AD) contained 21 participants.

2.1.2 Results

In all three groups clear MMN peaks were observed. No significant and systematic differences in the MMNs of the control, RD, and AD group were found. The only significant difference was in the lateralization of the MMN peaks: In the control group the MMNs were more negative over the right hemisphere, whereas in the RD group the opposite hemisphere showed more pronounced MMN responses. The lateralization of the MMNs of the AD group did not differ significantly from either one of the other two groups (controls, RD), but still showed the same trend as controls towards more prominent MMN peaks over the right hemisphere.

2.1.3 Discussion

Opposite to expectations, MMNs of children with RD were not attenuated as compared to control children and children with AD. In previous studies (Kujala et al., 2000; Schulte-Körne et al., 1999) of duration change induced MMNs participants with reading problems have shown deficient duration processing, although also negative findings have been reported (Baldeweg et al., 1999). The two studies showing deficient duration MMNs were carried out with adult participants, which can be one of the factors affecting the difference in the results. The children participating in the present study were relatively young, and it is still impossible to estimate whether they will be able to compensate for their problems, and whether their reading problems are severe enough to still show in adulthood. Thus, the participants of the study might have not have exhibited as severe language problems as the participants of the adult studies. Several studies reporting diminished MMNs in children have had participants with more severe language problems than RD (e.g. Korpilahti & Lang, 1994; Korpilahti, 1995; Holopainen, Korpilahti, Juottonen, Lang, & Sillanpää, 1998).

Significant difference was, however, observed in the patterns of MMN peak lateralization between control group and RD group. Typically MMNs to non-speech sounds are more prominent over the right hemisphere, whereas the opposite is usually seen with speech stimuli (for a review, see Lyytinen et al., 2005). Similar findings of atypical lateralization of MMNs have been reported in dysphasic children (Korpilahti & Lang, 1994; Holopainen et al., 1998) and in newborns with familial risk for dyslexia (Pihko et al., 1999; Leppänen, Pihko, Eklund, & Lyytinen, 1999; Guttorm, Leppänen, Richardson, & Lyytinen, 2001). Thus, it seems that the MMN processing was atypical in the RD group, even though the MMN peak amplitude values or latencies did not differentiate the groups.

2.2 Study II

The first study raised the question, whether the variation in the severity of the reading problems examined in MMN studies might explain the mixed results concerning the existence of auditory duration processing deficit in this group. Thus, the second study was a reanalysis of the same MMN data, but stricter criteria for reading disorder (RD) were applied. In addition, in the first study many children with attention deficit (AD) had concurrent reading problems. Some studies have shown children with both RD and AD to differ from children showing "pure" RD or AD (Bental & Tirosh, 2007; Toplak, Rucklidge, Hetherington, John, & Tannock, 2003). Therefore, the main interest of the second study was to investigate, whether MMNs of control children would differ from those of children with relatively severe RD, AD or both (RD+AD).

2.2.1 Methods

In the second study the same MMN data pool elicited by duration changes (standard 100 ms, deviants 50 or 30 ms) in continuous sound was analyzed. Stricter criteria in RD screening were applied and three clinically referred groups with either RD, AD, or both were formed. After the eye movement artifact rejection (± 60 μV) 650 trials on average were analyzed.

2.2.2 Results

The MMNs elicited by the more deviant sound change (30 ms) showed significant differences between the control group and all clinically referred groups: The RD group showed smaller MMNs in frontal and central channels in the right hemisphere, the MMNs of the RD+AD group were attenuated in frontal and central channels in both hemispheres, and the MMN peaks of the AD group appeared earlier in the frontal channels. In addition, latency differences between the clinically referred groups were observed as well: The MMNs peaked later in the RD+AD group as compared to both RD group and AD group.

Lateralization of the MMN peaks differed significantly only between the RD and AD groups, although a nonsignificant trend to similar difference was found between the RD and control group. As in the study I, RD group showed more pronounced MMN responses over the left hemisphere, whereas the opposite pattern was found in the AD and control group. In the RD+AD group the MMN peaks did not appear in visual inspection to be clearly lateralized to either hemisphere.

2.2.3 Discussion

The changes in the participant selection criteria (stricter RD criteria, division of participants into "pure" AD and RD groups and a comorbid RD+AD group) revealed significant differences between the clinically referred groups and control group, whereas no such differences were witnessed in the first study. Based on the results it seems that the participant selection can strongly affect the results obtained in MMN studies. In addition to the severity of the language problems under study, comorbid attention problems may affect the results.

The studies of auditory duration processing in individuals with reading problems have presented mixed results. One possibly relevant factor explaning the contrasting results is the native language of the participants. Baldeweg and colleagues (1999) did not find differences in the duration MMNs of English speaking participants, whereas Corbera and colleagues (2006) reported attenuated duration MMNs in Spanish speaking participants. In both Spanish and Finnish duration is a critical feature, and repetition of consonant letters lengthens the phonemic duration of a sound and changes the meaning of a word. In English, on the other hand, consonant duration does not have such a role. Therefore, duration processing problems might have more devastating effects on language skills in Finnish and Spanish as compared to English.

Another possible explanation might be differences in the stimuli. Standard stimulus of Baldeweg and colleagues (1999) was longer (200 ms), wheres in the current study and in the study of Corbera and colleagues (2006) standard stimulus of shorter duration (100 ms was used). The basic auditory processing deficit has been suggested to only show with rapidly presented stimuli of short durations (Frumkin & Rapin, 1980; Tallal, 1980; Tallal & Benasich, 2002; Wright et al., 1997).

The second study stressed the importance of clearly defined, "pure" groups. If comorbid problems in attention are not screened, the results of a MMN study can be obscured. Also the severity of the RD seems to affect the findings concerning an auditory duration processing deficit in this group.

2.3 Study III

The aim of the third study was to explore, whether electrophysiological (MMN) and behavioral tasks would provide similar results concerning auditory duration discrimination in children with attention deficit (AD), reading disorder (RD) or both (AD+RD). Although several studies have found children with RD to show auditory processing problems when either MMN or behavioral tasks have been used, only rarely have both types of tasks been presented to the same participants. In one of the few studies of this kind, Sharma and colleagues (2006) measured MMNs elicited by changes in tone frequency, chord frequency, and speech stimuli as well as behavioral auditory processing by dichotic listening, frequency discrimination, gap detection, and an ipsilateral speech-in-noise task. The only difference between the control and RD groups in MMN studies was a significantly larger MMN area elicited by speech stimuli, whereas in all behavioral tests participants with RD performed worse than control participants. Although all children with RD showed auditory processing problems of some kind, no consistent relationship between MMNs and behavioral performance was observed.

2.3.1 Methods

The same MMN stimuli as in studies I and II with duration changes (standard 100 ms, deviant 30 or 50 ms) in continuous sound were presented to three groups of children showing either AD (N=5), RD (N=5) or both (N=5). Only the MMNs elicited by the more deviant sound (30 ms) were included in the analysis since the smaller deviance (50 ms) did not show any significant differences between the groups. After the eye movement artefact rejection criteria (±60 μV) were applied 637 trials on average were included in the analyses.

The behavioral duration discrimination was estimated with a speech task, in which the stimuli consisted of a continuum (/ata/-/atta/) structured by stimuli varying in the duration of the stop closure (Richardson, 1998; Richardson, Leppänen, Leiwo, & Lyytinen, 2003). Short duration of the stop induces the stimulus to be perceived as /ata/, whereas long duration is perceived as /atta/. As no differences in the MMN amplitudes of the AD group in comparison to control children was found in the two previous studies, this group acted as a clinically referred control group to the two other groups showing RD.

2.3.2 Results

Children with RD and RD+AD showed significantly smaller MMNs as compared to the children with AD. AD group also performed more accurately in the behavioral duration discrimination task. Also some significant associations between behavioral duration discrimination scores and the MMN peak values were observed.

Although no significant differences in the lateralization of MMN peaks were observed, a nonsignificant trend to more pronounced MMN responses over the left hemisphere as compared to the right one was seen in the RD group, and an opposite trend in the AD group. In the RD+AD group no noticeably lateralization of MMNs was observed. These findings are in accordance of the two previous studies.

2.3.3 Discussion

The third study provided further support of an auditory duration discrimination deficit in participants with RD. In addition to diminished MMN responses, also behavioral duration discrimination was compromised in this group. Richardson (1998) and Richardson, Leppänen, Leiwo, and Lyytinen (2003) used the same behavioral duration discrimination task to study infants with a familial risk for dyslexia and their parents with dyslexia. Both infants and adults were found to show worse speech duration discrimination skills as compared to controls. This study provides futher support of a speech duration discrimination deficit in individuals with RD.

Two views of auditory processing skills in participants with reading problems have been presented. According to the first view these participants experience a basic auditory processing problem that affects also tone processing (Tallal, 1980; 1984; Reed, 1989; Farmer & Klein, 1993). The other suggests only the auditory speech processing is deficient (Studdert-Kennedy & Mody, 1995; Mody, Studdert-Kennedy, & Brady, 1997; Adlard & Hazan, 1998; Studdert-Kennedy, 2002). The current study showed auditory processing problems in children with RD with both tone and speech stimuli, whereas no such problems were observed in children with "pure" AD.

Some significant associations between the MMN responses and behavioral task performance were found, even though the MMN responses were elicited by tone duration changes and in the behavioral task the participants had to discriminate changes in speech duration. The fact that associations between these different types of tasks were found suggests that the basic auditory processing problems covary with speech processing problems.

No genuine control group was available for the study, and therefore the AD group acted as a clinically referred control group. Although this might have affected the results of the study, more probable is that the problems of the RD participants were underestimated than overestimated, since some studies of participants with attention problems have found this group to have problems in behavioral duration discrimination. No sound evidence of abnormal MMN responses has been reported in this participant group.

3 GENERAL DISCUSSION

The differences in the experimental designs of studies applying MMN have made it very difficult to compare MMN studies and draw firm conclusions based on them. There are differences in, for example, stimulus characteristics and presentation rates, data processing, and MMN peak quantification. In a review of 26 MMN studies of participants with either dyslexia or specific language impairment, Bishop (2007) concluded that although the results were highly inconsistent, attenuation of the MMN and atypical lateralization in the clinical groups was most typically reported in studies applying fast stimulus presentation rates. According to the recommendation of Bishop (2007), difference between standard and deviant should be smaller than 10% in order to optimize the chance of finding differences between the controls and the participants with language problems. In the duration studies it seems, however, that with short standard stimuli the difference has to be relatively large in order to show differences between the groups (Huttunen-Scott, Kaartinen, Tolvanen, & Lyytinen, 2008; Huttunen-Scott, Kaartinen, Richardson, & Lyytinen, submitted; Korpilahti, & Lang, 1994). It seems very important to choose the stimulus parameters correctly to attain group differences. It would also be recommendable to use same experimental designs in studies changing/adjusting other variables, like participant features, in order to compare studies.

Another thing making it difficult to draw firm conclusions on the basis of the MMN studies is the variance in the subject groups studied. The diagnostic practise and assessment of dyslexia and reading disabilities varies from one country to another, and it is hard to ensure the similarity of the groups under study even if the diagnostic label is the same. This is also related to the differences in the languages and reading problem types typically encountered in different languages. For example, when comparing Finnish and English, spelling errors are more typically made by English speaking participants with reading disabilities due to the inconsistency of orthography in English, whereas Finnish has consistent orthography and thus the problems in reading mainly show as slowness of reading (Landerl, 1997).

It has been found that the subtypes of dyslexia may present different kinds of impairment profiles. The study of magnocellular deficit conducted by Borsting and colleagues (1996), for example, applied the subtyping of Boder (1971), which recognizes three dyslexia subtypes: dysphonetic, dyseidetic and dysphoneidetic. Participants with dysphonetic dyslexia have problems with matching graphemes with phonemes and have problems especially with unfamiliar words but whole word reading of familiar words might be relatively fluent, whereas participants with dyseidetic dyslexia show problems especially in whole word recognition and find especially phonetically irregular words difficult. The third type, dysphoneidetic, comprises problems shown by both of the two other subtypes. The study reported magnocellular deficit only in participants with dysphoneidetic dyslexia, whereas the dyseidetic participants did not show impaired performance in tasks stressing magnocellular processing. Similarly, in the MMN study of Lachmann and colleagues (2005) only a subgroup of participants with dyslexia showed diminished MMN. The participants were divided into dyslexics-1, a group showing impairments in non-word reading and frequent word reading and dyslexics-2, which experienced problems in only frequent word reading. Thus, the first group presented problems associated with dysphoneidetic dyslexia and the latter with dyseidetic dyslexia. Only the dyslexics-2 had diminished MMN. Also Leonard (2001) has stressed the homogeneousness of the group with reading disabilities when studying neuroanatomical markers of reading problems. In the study of Leonard and colleagues (2001) only participants with phonological deficit differed from controls in neuroanatomical comparisons. Heim and colleagues (2008) tested phonological awareness, auditory discrimination, motion detection, visual attention, and rhythm imitation in participants with dyslexia in a study aiming to identify different subtypes within the group. Three groups with distinct cognitive profiles were identified: participants with problems in phonological tasks only, those with difficulties only in attention, and participants with deteriorated performance in phonological as well as auditory and magnocellular tasks. Thus, the study provided evidence of problems described by many current theories striving to explain reading problems but only in subgroups of participants.

Although Tallal (1980) found children with reading problems to show problems in rapid auditory processing, Tallal and Stark (1982) later suggested that the finding might be explained by inclusion of children with oral language deficits. In the study of Tallal and Stark (1982) reading impaired children with no oral language deficit were not found to differ from controls in TOJ with varying ISIs. As well, the studies of this thesis only found significantly deficient duration MMN in participants showing relatively severe reading problems, whereas no such difference was found in the first study with milder problems. One possibility is that in the more severe cases of RD the auditory processing is more widely disturbed and deficiencies in several types of tasks is found in group comparisons, whereas in the milder cases the auditory processing problems might vary more and be less pronounced and thus harder to document. The current diagnostic practice might not be precise enough and therefore classify individuals with significantly different skill profiles to same diagnostic groups.

Still another important factor contributing to the differences in the studies is the issue of developmental factors. It has been suggested that children with language problems might show a maturational lag in their auditory processing (McArthur & Bishop, 2004; Wright & Zecker, 2004). According to Wright and Zecker (2004) puberty might play an important part in the development of language skills; because of a maturational lag in perceptual development, slowly developing auditory skills might not be achieved before puberty (when the brain plasticity decreases) and might thereby remain less developed. It seems that the MMN is still not fully matured by the age of 11 years (Martin, Shafer, Morr, Kreuzer, & Kurtzberg, 2003), although some studies have suggested a considerably earlier maturation (Kraus, Koch, McGee, Nicol, & Cunningham, 1999). Typically the MMNs of children are also larger in amplitude as compared with adult MMN (Gomot et al., 2000; Shafer et al., 2000) and the latency also diminishes with age (Shafer et al., 2000). Thus, in certain age groups the maturational lag in development might cause the results to suggest that participants with dyslexia actually have a bigger MMN. Another confusing factor is the often-reported positive change detection response in infants (Friederici, Friedrich, & Weber, 2002; He, Hotson, & Trainor, 2007; Leppänen, Guttorm, Pihko, Takkinen, & Lyytinen, 2004). Infant studies have also suggested that different sound features have different maturational timetables (He, Hotson, & Trainor, 2007).

Different features are semantically relevant in different languages, and the elicitation of MMN seems to be affected by the language background of the subjects. MMN elicitation depends on whether the stimulus change occurs in features relevant in subjects language (Näätänen et al., 1997; Tervaniemi et al., 2006; Ylinen et al., 2006). Difficulty discriminating a feature critical in ones native language might cause problems in language development, whereas the same feature might not be as central in another language and might not appear to be problematic to individuals with reading disorder. An example of this kind of difference between languages might be the importance of duration discrimination in languages like Finnish and Spanish as compared to English.

MMN has not been actively used as a clinical tool despite being studied in several clinical groups, for example in participants with schizophrenia, Alzheimer's or alcoholism. This is especially due to problems in interpreting individual MMN responses. There is much variation in MMNs between individuals and even the replicability of the peak does not seem very reliable (Picton, Alain, Otten, Ritter, & Achim, 2000). Of the different stimulus features duration seems to show fairly good test-retest stability (Uwer & von Suchodoletz, 2000). In order to be used more in clinical evaluation, more experiments combining ERP and behavioral tasks need to be conducted and a better understanding of the relationship between MMN and behavioral performance has to be reached.

MMN studies of participants with attention deficit are still scarce, but the results of the studies of this thesis support the view of normal MMNs in this participant group. The only study that has found significantly diminished MMNs in participants with attention problems was conducted by Winsberg and colleagues (1993) and even in this study the difference might have been caused by peak latency difference. Also our study II showed participants with attention deficit to differ from controls in the latency of the MMN peaks. The latency differences of groups under study may distort the results if ignored. Apart from the latency diffrerences, none of the studies of this thesis using duration changes in continuous sound found reduced MMN amplitudes in children with attention deficit.

The importance of clearly defined participant groups cannot be overemphasized. Attention deficits and reading disorders often co-occur (Dykman & Ackerman, 1991; Knivsberg & Andreassen, 2008; Lyytinen et al., 1998; Willcutt & Pennington, 2000a&b), and participants with comorbid problems may differ from the participants with "pure" attention or reading problem (Bental, & Tirosh, 2007; Toplak, Rucklidge, Hetherington, John, & Tannock, 2003). The present studies show that "pure" groups and comorbid groups should be treated as independent groups in order to identify the unique features of each group.

Pennington (2006) has suggested changing the focus of studies from looking single deficits to probing multiple deficit models allowing several interacting factors in different levels of analysis to affect the outcome of a disorder. The model acknowledges the possibility of several genetic and environmental risk and protective factors, which interact with each other, and none of which might be sufficient to cause all symptoms of a behavioral disorder. At the moment several factors affecting reading development have been well documented and studied, and it is becoming more obvious that no single factor is enough to explain reading problems at any level. Thus, multiple deficit models as described by Pennington (2006) might provide an ambitious goal for the future research.

TIIVISTELMÄ

Kuulonvarainen keston erottelu lapsilla, joilla on lukemisvaikeus, tarkkaavaisuuden ongelma tai molemmat

Kuulonvaraisen prosessoinnin ongelmien on esitetty olevan yksi mahdollinen syy lukemisvaikeuksiin ja niiden yhteydessä tyypillisesti esiintyviin fonologisen prosessoinnin ongelmiin. Tallalin (1980; 2000) mukaan kuulonvaraisen prosessoinnin ongelmat voivat vaikeuttaa puheen prosessointia, mikä puolestaan johtaa vaikeuksiin kielen pienimpien yksiköiden, foneemien, hahmottamisessa ja käsittelyssä. Vaikka useissa tutkimuksissa lukemisvaikeuksien yhteydessä on havaittu kuulonvaraisen prosessoinnin vaikeutta, tulokset vaihtelevat huomattavasti tutkimuksesta toiseen. Syynä ristiriitaisiin tuloksiin ovat todennäköisesti muun muassa huomattavat erot tutkimusmenetelmissä ja otoksissa.

Kuulonvaraisen prosessoinnin tutkimuksessa käytetyt tutkimusmenetelmät ovat vaihdelleet käyttäytymistason tehtävistä psykofysiologisiin tutkimuksiin. Aivojen kuvantamisen tutkimuksissa poikkeavuusnegatiivisuudeksi (mismatch negativity, MMN) nimetty aivosähkökäyrän herätepotentiaali on objektiivinen mittari kuulonvaraisen erottelukyvyn arviointiin. Poikkeavuusnegatiivisuus voidaan havaita poikkeavan ärsykkeen esiintyessä toistettavien ääniärsykkeiden joukossa. Toisin sanoen, poikkeavuusnegatiivisuus havaitaan aivojen reagoidessa muutokseen kuulonvaraisessa informaatiossa.

Kielet eroavat toisistaan niille keskeisten piirteiden osalta. Suomen kielessä kestolla on keskeinen merkitys sanojen merkitystä määrittävänä piirteenä, ja toisinaan kaksi muuten identtistä sanaa eroaakin toisistaan ainoastaan foneemin keston osalta (esim. mato-matto). Poikkeavuusnegatiivisuuden avulla kuulonvaraista prosessointia lukemisvaikeuksisilla koehenkilöillä arvioineet tutkimukset ovat tuottaneet ristiriitaista tietoa keston muutosten havainnoinnista kyseisessä ryhmässä. Englanninkielisillä tutkittavilla ei ole havaittu heikompaa poikkeavuusnegatiivisuutta lukemisvaikeuksien yhteydessä (Baldeweg ym., 1999), kun taas espanjankielisillä, lukemisvaikeuksisilla tutkittavilla poikkeavuusnegatiivisuuden on havaittu olevan heikentynyt (Corbera, Escera, & Artigas, 2006). Espanjan kielessä, kuten suomen kielessäkin, kesto on keskeinen merkitystä määrittävä piirre, mikä saattaa osaltaan vaikuttaa tuloksiin.

Kielen ohella tutkittavat ryhmät voivat erota myös muiden olennaisten muuttujien osalta. Yksi tällainen tekijä ovat lukemisvaikeuksien kanssa samanaikaisesti esiintyvät vaikeudet. Lukemisvaikeuksien yhteydessä havaitaan usein tarkkaavaisuuden ongelmia, jotka voivat vaikuttaa tutkimustuloksiin vääristävästi, mikäli kyseisiä ongelmia ei seulota tutkimusryhmiä määritettäessä. Aiemmissa tutkimuksissa on havaittu, että tutkittavat, joilla on samanaikaisia vaikeuksia sekä lukemisessa että tarkkaavaisuudessa, poikkeavat tutkittavista, joilla esiintyy ainoastaan lukemisen tai tarkkaavaisuuden ongelmia (Bental, & Tirosh, 2007; Toplak, Rucklidge, Hetherington, John, & Tannock, 2003).

Tähän väitöskirjaan kuuluvissa tutkimuksissa oli tarkoitus selvittää keston erottelun ongelmien esiintymistä suomenkielisillä lapsilla, joilla on vaikeuksia lukemisessa, tarkkaavaisuudessa tai molemmissa edellä mainituissa taidoissa. Koska keston erottelulla on keskeinen rooli suomen kielessä, sen tutkiminen on erityisen kiinnostavaa kyseisessä kieliryhmässä. Lukemisvaikeuksien lisäksi tutkimuksissa arvioitiin keston prosessointia tarkkaavaisuusongelmien yhteydessä, millä pyrittiin selvittämään, onko mahdollinen keston prosessoinnin heikkous erityispiirre, joka on havaittavissa ainoastaan lukemisvaikeuksien yhteydessä.

Ensimmäisessä tutkimuksessa normaalisti lukevia lapsia verrattiin lapsiin, joilla oli joko lukemisvaikeus tai tarkkaavaisuusongelma. Tutkimuksessa tuotettiin poikkeavuusnegatiivisuusvaste yhtäjaksoisessa äänessä esiintyvillä keston muutoksilla. Ryhmien välillä ei ollut merkittäviä eroja poikkeavuusnegatiivisuuden ajoituksen tai suuruuden suhteen, mutta normaalisti lukevilla lapsilla poikkeavuusnegatiivisuus oli painottunut oikeaan aivopuoliskoon, kun taas lukemisvaikeuksisilla poikkeavuusnegatiivisuus oli vahvempi vasemmalla aivopuoliskolla. Tarkkaavaisuusongelmaisten lasten poikkeavuusnegatiivisuusvasteet eivät poikenneet normaalisti lukevien vasteista.

Toista tutkimusta varten ryhmävalinnan kriteerejä muutettiin, sillä tavoitteena oli tutkia, ovatko ryhmien päällekkäisyys ja lukemisvaikeuksien aste mahdollisesti vaikuttaneet tuloksiin. Ensimmäisessä tutkimuksessa merkittävällä osalla tarkkaavaisuusongelmaisia lapsia oli havaittavissa myös lukemisvaikeuksia. Ryhmien selkiyttämiseksi tutkittavat jaettiin neljään ryhmään: normaalisti lukevat lapset, lukemisvaikeuksiset lapset, tarkkaavaisuusongelmaiset lapset ja lapset, joilla oli sekä lukemisvaikeuksia että tarkkaavaisuusongelmia. Lisäksi lukemisvaikeuksien määrittelyssä käytettyjä kriteerejä tiukennettiin, minkä vuoksi toisessa tutkimuksessa lukemisvaikeudet olivat asteeltaan vaikeampia kuin ensimmäisessä. Keston muutoksella tuotetun poikkeavuusnegatiivisuuden havaittiin olevan merkitsevästi heikentynyt sekä lapsilla, joilla oli ainoastaan lukemisvaikeuksia että jopa merkitsevämmin heikentynyt lapsilla, joilla oli lukemisvaikeuksien ohella myös tarkkaavaisuuden ongelmia. Tarkkaavaisuusongelmaisten lasten poikkeavuusnegatiivisuus-vasteet puolestaan erosivat niiden esiintymisajankohdan suhteen: normaalisti lukevilla poikkeavuusnegatiivisuus esiintyi merkitsevästi tarkkaavaisuusongelmaisten ryhmää myöhemmin.

Kolmannessa tutkimuksessa pyrittiin arvioimaan keston erottelua poikkeavuusnegatiivisuuden ohella myös käyttäytymistason tehtävän avulla. Käyttäytymistason tehtävässä lasten tuli havaita keston muutos puheärsykkeessä. Koska aiemmat tutkimukset osoittivat, etteivät tarkkaavaisuusongelmaisten lasten poikkeavuusnegatiivisuus-vasteet eroa normaalisti lukevien vasteista, kolmannessa tutkimuksessa vertasimme tarkkaavaisuusongelmaisia lukemisvaikeuksisiin sekä lapsiin, joilla oli sekä tarkkaavaisuuden että lukemisen ongelmia. Tarkkaavaisuusongelmaisten lasten ryhmässä poikkeavuusnegatiivisuus-vasteet olivat merkitsevästi suurempia kuin kahdessa muussa ryhmässä. Lisäksi käyttäytymistason tehtävässä tarkkaavaisuusongelmaisten ryhmä suoriutui kahta muuta ryhmää tarkemmin. Psykofysiologisten muuttujien ja käyttäytymistason muuttujien välillä oli myös havaittavissa merkitseviä korrelaatioita.

Väitöskirjaan kuuluvissa tutkimuksissa havaittiin kuulonvaraisen keston erottelun vaikeus lapsilla, joilla on lukemisvaikeuksia riippumatta samanaikaisesta tarkkaavaisuusongelmasta. Tarkkaavaisuusongelmien yhteydessä kuulonvaraisen keston erottelu ei puolestaan ollut heikentynyt, vaikka erottelun nopeudessa olikin poikkeavuutta. Kuulonvaraisen keston erottelun vaikeus on tutkimusten perusteella lukemisvaikeuksien yhteydessä havaittava erityispiirre, joka ei selity tarkkaavaisuuden ongelmilla.

REFERENCES

Adlard, A. & Hazan, V. (1998). Speech perception in children with specific reading difficulties (dyslexia). The Quarterly Journal of Experimental Psychology, 51, 153-177.

Ahissar, M. (2007). Dyslexia and the anchoring-deficit hypothesis. Trends in Cognitive Sciences, 11, 458-465.

Ahissar, M., Lubin, Y., Putter-Katz, H., & Banai, K. (2006). Dyslexia and the failure to for a perceptual anchor. Nature Neuroscience, 9, 1558-1564.

Ahissar, M., Protopapas, A., Reid, M., & Merzenich, M.M. (2000). Auditory processing parallels reading abilities in adults. Proceedings of the National Academy of Sciences of the United States of America (PNAS), 97, 6832-6837.

Alarcón, M. & DeFries, J.C. (1997). Reading performance and general cognitive ability in twins with reading difficulties and control pairs. Personality and Individual Differences, 22, 793-803.

Alho, K. (1995). Cerebral generators of mismatch negativity (MMN) and its magnetic counterparts (MMNm) elicited by sound changes. Ear and Hearing, 16, 38-51.

Alho, K., Sainio, K., Sajaniemi, N., Reinikainen, K., & Näätänen, R. (1990). Event-related brain potentials of human newborns to pitch change of an acoustic stimulus. Electroencephalography and Clinical Neurophysiology, 77, 151-155.

Alho, K., Woods, D.L., & Algazi, A. (1994). Processing of auditory stimuli during auditory and visual attention as revealed by event-related potentials. Psychophysiology, 31, 469-479.

Alonso-Búa, B., Díaz, F., & Ferraces, M.J. (2006). The contribution of AERPs (MMN and LDN) to studying temporal vs. linguistic processing deficits in children with reading difficulties. International Journal of Psychophysiology, 59, 159-167.

American Psychiatric Association (APA). (2000). Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR, 4th ed., text revision). Washington, DC: Author.

Amitay, S., Ahissar, M., & Nelken, I. (2002). Auditory processing deficits in reading disabled adults. JARO, 03, 302-320.

Au, A., & Lovegrove, B. (2001). The role of visual and auditory temporal processing in reading irregular and nonsense words. Perception, 30, 1127-1142.

Baddeley, A. (2003). Working memory and language: an overview. Journal of Communication Disorders, 36, 189-208.

Bakwin, H. (1973). Reading disability in twins. Developmental Medicine and Child Neurology, 15, 184-187.

Baldeweg, T., Richardson, A., Watkins, S., Foale, C., & Gruzelier, J. (1999). Impaired auditory frequency discrimination in dyslexia detected with mismatch evoked potentials. Annals of Neurology, 45, 495-503.

Banai, K. & Ahissar, M. (2006). Auditory processing deficits in dyslexia: Task or stimulus related? Cerebral Cortex, 16, 1718-1728.

Barker, T.A., Torgesen, J.K., & Wagner, R.K. (1992). The role of orthographic processing skills on five different reading tasks. Reading Research Quarterly, 27, 334-345.

Beaulieu, C., Plewes, C., Paulson, L.A., Roy, D., Snook, L., Concha, L., & Phillips, L. (2005). Imaging brain connectivity in children with diverse reading ability. NeuroImage, 25, 1266-1271.

Benasich, A.A., Choudhury, N., Friedman, J.T., Realpe-Bonilla, T., Chojnowska, C., & Gou, Z. (2006). The infant as a prelinguistic model for language learning impairments: Predicting from event-related potentials to behavior. Neuropsychologia, 44, 396-411.

Bental, B. & Tirosh, E. (2007). The relationship between attention, executive functions and reading domain abilities in attention deficit hyperactivity disorder and reading disorder: a comparative study. Journal of Child Psychology and Psychiatry, 48, 455-463.

Ben-Yehudah, G., Sackett, E., Malchi-Ginzberg, L., & Ahissar, M. (2001). Impaired temporal contrast sensitivity in dyslexics is specific to retain-and-compare paradigms. Brain, 124, 1381-1395.

Best, M., & Demb, J. (1999). Normal planum temporale asymmetry in dyslexics with a magnocellular pathway deficit. Neuroreport, 10, 607-612.

Bishop, D.V.M. (2007). Using mismatch negativity to study central auditory processing in developmental language and literacy impairments: Where are we, and where should we be going? Psychological Bulletin, 133, 651-672.

Bishop, D.V.M., Carlyon, R.P., Deeks, J.M., & Bishop, S.J. (1999). Auditory temporal processing impairment. Neither necessary nor sufficient for causing language impairment in children. Journal of Speech, Language, and Hearing Research, 42, 1295-1310.

Blomert, L. & Mitterer, H. (2004). The fragile nature of the speech-perception deficit in dyslexia: Natural vs. synthetic speech. Brain and Language, 89, 21-26.

Boder, E. (1971) Developmental dyslexia: Prevailing diagnostic concepts. In H.R. Mykelbust (Ed), Progress in Learning Disabilities and a New Diagnostic Approach (pp. 293-321). New York: Grune and Stratton.

Boets, B., Wouters, J., van Wieringen, A., & Ghesquiére, P. (2007). Auditory processing, speech perception and phonological ability in pre-school children at high-risk for dyslexia: A longitudinal study of the auditory temporal processing theory. Neuropsychologia, 45, 1608-1620.

Boets, B., Wouters, J., van Wieringen, A., De Smedt, B., & Ghesquiére, P. (2008). Modelling relations between sensory processing, speech perception, orthographic and phonological ability, and literacy achievement. Brain and Language, 106, 29-40.

Borsting, E., Ridder, W.H. 3rd, Dudeck, K., Kelley, C., Matsui, L., & Motoyama, J. (1996). The presence of a magnocellular defect depends on the type of dyslexia. Vision Research, 36, 1047-1063.

Bradley, L., & Bryant, P. (1983). Categorizing sounds and learning to read: A causal connection. Nature, 301, 419-421.

Brady, S. (1991). The role of working memory in reading disability. In Brady, S.A., Shankweiler, D.P. (Eds.), Phonological Processes in Literacy: A Tribute to Isabelle Y. Liberman (pp. 129-151). Hillsdale, NJ: Lawrence Erlbaum Associates.

Brady, S.A. & Shankweiler, D.P. (1991). Phonological processes in literacy: A tribute to Isabelle Y Liberman. Hillsdale, NJ: Lawrence Erlbaum Associates.

Brady, S.A., Shankweiler, D.P., & Mann, V. (1983). Speech perception and memory coding in relation to reading ability. Journal of Experimental Child Psychology, 35, 345-367.

Breier, J.I., Fletcher, J.M., Denton, C., & Gray, L.C. (2004). Categorical perception of speech stimuli in children at risk for reading difficulty. Journal of Experimental Child Psychology, 88, 152-170.

Breier, J.I., Gray, L.C., Fletcher, J.M., Foorman, B., & Klaas, P. (2002). Perception of speech and nonspeech stimuli by children with and without reading disability and attention deficit hyperactivity disorder. Journal of Experimental Child Psychology, 82, 226-250.

Breier, J.I., Simos, P.G., Fletcher, J.M., Castillo, E.M., Zhang, W., & Papanicolaou, A.C. (2003). Abnormal activation of temporoparietal language areas during phonetic analysis in children with dyslexia. Neuropsychology, 17, 610-621.

Brown, W.E., Eliez, S., Menon, V., Rumsey, J.M., White, C.D., & Reiss, A.L. (2001). Preliminary evidence of widespread morphological variations of the brain in dyslexia. Neurology, 56, 781-783.

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.

Campbell, K., Bell, I., & Bastien, C. (1991). Evoked potential measures of information processing during natural sleep. In R. Broughton & R. Ogilvie (Eds.), Sleep, arousal, and performance (pp. 88-116). Cambridge: M.A. Birkhauser.

Cardon, L.R., Smith, S.D., Fulker, D.W., Kimberling, W.J., Pennington, B.F., & DeFries, J.C. (1994). Quantitative trait locus for reading disability on chromosome 6. Science, 266, 276-279.

Ceponiené, R., Cheour, M., & Näätänen, R. (1998). Interstimulus interval and auditory event-related potentials in children: evidence for multiple generators. Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section, 108, 345-354.

Ceponiené, R., Kushnerenko, E., Fellman, V., Renlund, M., Suominen, K., & Näätänen, R. (2002). Event-related potential features indexing central auditory discrimination by newborns. Cognitive Brain Research, 13, 101-113.

Ceponiené, R., Lepistö, T., Soininen, M., Aronen, E., Alku, P., & Näätänen, R. (2004). Event-related potentials associated with sound discrimination versus novelty detection in children. Psychophysiology, 41, 130-141.

Cheour, M., Alho, K., Ceponiené, R., Reinikainen, K., Sainio, K., Pohjavuori, M., Aaltonen, O., & Näätänen, R. (1998a). Maturation of mismatch negativity in infants. International Journal of Psychophysiology, 29, 217-226.

Cheour, M., Ceponiené, R., Lehtokoski, A., Luuk, A., Allik, J., Alho, K., & Näätänen, R. (1998b). Development of language-specific phoneme representations in the infant brain. Nature Neuroscience, 1, 351-353.

Cheour, M., Kusherenko, E., Ceponiené, R., Fellman, F., & Näätänen, R. (2002a). Electric brain responses obtained from newborn infants to changes in duration in complex harmonic tones. Developmental Neuropsychology, 22, 471-479.

Cheour, M., Shestakova, A., Alku, P., Ceponiené, R., & Näätänen, R. (2002b). Mismatch negativity shows that 3-6-year-old children can learn to discriminate non-native speech sounds within two months. Neuroscience Letters, 325, 187-190.

Cheour-Luthanen, M., Alho, K., Sainio, K., Rinne, T., Reinikainen, K., Pohjavuori, M., Renlund, M., Aaltonen, O., Eerola, O., & Näätänen, R. (1996). The ontogenetically earliest discriminative response of the human brain. Psychophysiology, 33, 478-481.

Chiarenza, G.A. (1990). Motor-perceptual function in children with developmental reading disorders: Neuropsychophysiological analysis. Journal of Learning Disabilities, 23, 375-385.

Clark, M.M. & Plante, E. (1998). Morphology of the inferior frontal gyrus in developmentally language-disordered adults. Brain and Language, 61, 288-303.

Cohen, L., Lehéricy, S., Chochon, F., Lemer, C., Rivaud, S., & Dehaene, S. (2002). Language-specific tuning of visual cortex? Functional properties of the visual word form area. Brain, 125, 1054-1069.

Cohen, L., Martinaud, O., Lemer, C., Lehéricy, S., Samson, Y., Obadia, M., Slachevsky, A., & Dehaene, S. (2003). Visual word recognition in the left and right hemispheres: Anatomical and functional correlates of peripheral alexias. Cerebral Cortex, 13, 1313-1333.

Cohen, R.L. & Netley, C. (1981). Short-term memory deficits in reading disabled children, in the absence of opportunity for rehearsal strategies. Intelligence, 5, 69-76.

Corbera, S., Escera, C., & Artigas, J. (2006) Impaired duration mismatch negativity in developmental dyslexia. Neuroreport, 17, 1051-1055.

Cornelissen, P.L., Hansen, P.C., Gilchrist, I., Cormack, F., Essex, J., & Frankish, C. (1998a). Coherent motion detection and letter position encoding. Vision Research, 38, 2181-2191.

Cornelissen, P.L., Hansen, P.C., Hutton, J.L., Evangelinou, V., & Stein, J.F. (1998b). Magnocellular visual function and children's single word reading. Vision Research, 38, 471-482.

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.

DeFries, J.C. & Alarcón, M. (1998). Genetics of specific reading disability. Mental Retardation and Developmental Disabilities Research Reviews, 2, 39-47.

DeFries, J.C., Fulker, D.W., & Labuda, M.C. (1987). Evidence for a genetic aetiology in reading disability twins. Nature, 329, 537-539.

DeFries, J.C., Singer, S.M., Foch, T.T., & Lewitter, F.I. (1978). Familial nature of reading disability. The British Journal of Psychiatry, 132, 361-367.

De Gelder, B. & Vroomen, J. (1998). Impaired speech perception in poor readers: Evidence from hearing and speech reading. Brain and Language, 64, 269-281.

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.

Dehaene, S., Naccache, L., Cohen, L., Le Bihan, D., Mangin, J.-F., Poline, J.-B., & Riviére, D. (2001). Cerebral mechanisms of word masking and unconscious repetition priming. Nature Neuroscience, 4, 752-758.

De Jong, P.F. (1998). Working memory deficits of reading disabled children. Journal of Experimental Child Psychology, 70, 75-96.

Demb, J.B., Boynton, G.M., Best, M., & Heeger, D.J. (1998). Psychophysical evidence for a magnocellular pathway deficit in dyslexia. Vision Research, 38, 1555-1559.

Demb, J.G., Boynton, G.M., & Heeger, D.J. (1998). Functional magnetic resonance imaging of early visual pathways in dyslexia. The Journal of Neuroscience, 18, 6939-6951.

Démonet, J.F., Fiez, J.A., Paulesu, E., Petersen, S.E., & Zatorre, R.J. (1996). PET studies of phonological processing: A critical reply to Poeppel. Brain and Language, 55, 352-379.

Deutsch, G.K., Dougherty, R.F., Bammer, R., Siok, W.T., Gabrieli, J.D.E., & Wandell, B. (2005). Children's reading performance is correlated with white matter structure measured by diffusion tensor imaging. Cortex, 41, 354-363.

De Weirdt, W. (1988). Speech perception and frequency discrimination in good and poor readers. Applied psycholinguistics, 9, 163-183.

Dhar, M., Been, P.H., Minderaa, R.B., & Althaus, M. (2008). Distinct information processing characteristics in dyslexia and ADHD during a covert orienting task: An event-related potential study. Clinical Neurophysiology, doi:10.1016/j.clinph.2008.05.027.

Dougherty, R.F., Ben-Shachar, M., Deutsch, G.K., Hernandez, A., Fox, G.R., & Wandell, B.A. (2007). Temporal-callosal pathway diffusivity predicts phonological skills in children. Proceedings of the National Academy of Sciences of the United States of America (PNAS), 104, 8556-8561.

Duara, R., Kushch, A., Gross-Glenn, K., Barker, W.W., Jallad, B., Pascal, S., Loewenstein, D.A., Sheldon, J., Rabin, M., Levin, B., & Lubs, H. (1991). Neuroanatomic differences between dyslexic and normal readers on magnetic resonance imaging scans. Archives of Neurology, 48, 410-416.

Dykman, R.A. & Ackerman, P.T. (1991). Attention deficit disorder and specific reading disability: Separate but often overlapping disorders. Journal of Learning Disabilities, 24, 96-103.

Eckert, M.A., Leonard, C.M., Richards, T.L., Aylward, E.H., Thomson, J., & Berninger, V.W. (2003). Anatomical correlates of dyslexia: frontal and cerebellar findings. Brain, 126, 482-494.

Eden, G.F., VanMeter, J.W., Rumsey, J.M., Maisog, J.M., Woods, R.P., & Zeffiro, T.A. (1996). Abnormal processing of visual motion in dyslexia revealed by functional brain imaging. Nature, 382, 66-69.

Eden, G.F., VanMeter, J.W., Rumsey, J.M., & Zeffiro, T.A. (1996). The visual deficit theory of developmental dyslexia. Neuroimage, 4, S108-S117.

Eden, G.F., Stein, J.F., Wood, H.M., & Wood, F.B. (1994). Differences in eye movements and reading problems in dyslexic and normal children. Vision Research, 34, 1345-1348.

Eliez, S., Rumsey, J.M., Giedd, J.N., Schmit, J.E., Patwardhan, A.J., & Reiss, A.L. (2000). Morphological alteration of temporal lobe gray matter in dyslexia: An MRI study. The Journal of Child Psychology and Psychiatry and Allied Disciplines, 41, 637-644.

Fagerheim, T., Raeymaekers, P., Tønnessen, F.E., Pedersen, M., Tranebjaerg, L., & Lubs, H.A. (1999). A new gene (DYX3) for dyslexia is located on chromosome 2. Journal of Medical Genetics, 36, 664-669.

Falzi, G., Perrone, P., & Vignolo, L.A. (1982). Right-left asymmetry in anterior speech region. Archives of Neurology, 39, 239-240.

Faraone, S.V., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The worldwide prevalence of ADHD: is it an American condition? World Psychiatry, 2, 104-113.

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., & Dean, P. (1996). Impaired performance of children with dyslexia on a range of cerebellar tasks. Annals of Dyslexia, 46, 259-283.

Felmingham, K.L. & Jakobson, L.S. (1995). Visual and visuomotor performance in dyslexic children. Experimental Brain Research, 106, 467-474.

Felton, R.H., Naylor, C.E., & Wood, F.B. (1990). Neuropsychological profile of adult dyslexics, Brain and Language, 39, 485-497.

Finch, A.J., Nicolson, R.I., & Fawcett, A.J. (2002). Evidence for a neuroanatomical difference within the olivo-cerebellar pathway of adults with dyslexia. Cortex, 38. 529-539.

Fischer, B. & Weber, H. (1990). Saccadic reaction times of dyslexic and age-matched normal subjects. Perception, 19, 805-818.

Fisher, A.E., Barnes, G., Hillebrand, A., Holliday, I.E., Witton, C., & Richards, I.L. (2006). Abnormality of mismatch negativity in response to tone omission in dyslexic adults. Brain Research, 1077, 90-98.

Fisher, S.E. & DeFries, J.C. (2002). Developmental dyslexia: Genetic dissection of a complex cognitive trait. Nature reviews, 3, 767-780.

Fisher, S.E., Francks, C., & Marlow, A.J., MacPhie, I.L., Newbury, D.F., Cardon, L.R., Ishikawa-Brush, Y., Richardson, A.J., Talcott, J.B., Gayán, J., Olson, R.K., Pennington, B.F., Smith, S.D., DeFries, J.C., Stein, J.F., & Monaco, A.P. (2002). Independent genome-wide scans identify a chromosome 18 quantitative trait locus influencing dyslexia. Nature Genetics, 30, 86-91.

Fisher, S.E., Marlow, A.J., Lamb, J., Maestrini, E., Williams, D.F., Richardson, A.J., Weeks, D.E., Stein, J.F., & Monaco, A.P. (1999). A quantitative-trait locus on chromosome 6p influences different aspects of developmental dyslexia. The American Journal of Human Genetics (AJHG), 64, 146-156.

Friederici, A.D., Friedrich, M., & Weber, C. (2002). Neural manifestation of cognitive and precognitive mismatch detection in early infancy. Neuroreport, 13, 1251-1254.

Frumkin, B. & Rapin, I. (1980). Perception of vowels and consonant-vowels of varying duration in language impaired children. Neuropsychologia, 18, 443-454.

Gaab, N., Gabrieli, J.D., Deutsch, G.K., Tallal, P., & Temple, E. (2007). Neural correlates of rapid auditory processing are disrupted in children with developmental dyslexia and ameliorated with training: an fMRI study. Restorative Neurology and Neuroscience, 25, 295-310.

Galaburda, A.M. (1989). Ordinary and extraordinary brain development: Anatomical variation in developmental dyslexia. Annals of Dyslexia, 39, 65-80.

Galaburda, A.M. (1995). Developmental dyslexia and animal studies: at the interface between cognition and neurology. In Mehler, J., & Franck, S. (Eds.), Cognition on cognition (pp. 31-47). Paris: Mit Press.

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., Menard, M.T., & Rosen, G.D. (1994). Evidence of aberrant auditory anatomy in developmental dyslexia. Proceedings of the National Academy of Sciences of the United States of America, 91, 8010-8013.

Galaburda, A.M., Sherman, G.F., Rosen, G.D., Aboitiz, F., & Geschwind, N. (1985). Developmental dyslexia: Four consecutive patients with cortical anomalies. Annals of Neurology, 18, 222-233.

Gayán, J., & Olson, R.K. (2001). Genetic and environmental influences on orthographic and phonological skills in children with reading disabilities. Developmental Neuropsychology, 20, 483-507.

Gayán, J., Willcutt, E.G., Fisher, S.E., Francks, C., Cardon, L.R., Olson, R.K., Pennington, B.F., Smith, S.D., Monaco, A.P., & DeFries, J.C. (2005). Bivariate linkage scan for reading disability and attention-deficit/hyperactivity disorder localizes pleiotropic loci. Journal of Child Psychology and Psychiatry, 46, 1045-1056.

Georgiewa, P., Rzanny, R., Gaser, C., Gerhard, U.-J., Vieweg, U., Freesmeyer, D., Mentzel, H.-J., Kaiser, W.A., & Blanz, B. (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., & Blanz, B. (1999). FMRI during word processing in dyslexic and normal reading children. Brain Imaging, 10, 3459-3465.

Geschwind, N., & Behan, P. (1982). Left-handedness: Association with immune disease, migraine, and developmental learning disorder. Proceedings of the National Academy of Sciences of the United States of America (PNAS), 79, 5097-5100.

Geschwind, N., & Galaburda, A.M. (1985). Cerebral lateralization: Biological mechanisms, associations, and pathology: I. A hypothesis and a program for research, Archives of Neurology, 42, 428-459.

Geschwind, N., & Levitsky, W. (1968). Human brain: Left-right asymmetries in temporal speech region. Science, 161, 186-187.

Giard, M.H., Perrin, F., Pernier, J., & Bouchet, P. (1990). Brain generators implicated in the processing of auditory stimulus deviance: A topographic event-related potential study. Psychophysiology, 27, 627-640.

Giard, M.H., Perrin, F., Echallier, J.F., Thevenet, M., Froment, J.C., & Pernier, J. (1994). Dissociation of temporal and frontal components in the human auditory N1 wave a scalp current density and dipole model analysis. Electroencephalography and Clinical Neurophysiology, 92, 238-252.

Glass, E., Sachse, S., & von Suchodoletz, W. (2008). Development of auditory sensory memory from 2 to 6 years: an MMN study. Journal of Neural Transmission, 115, 1221-1229.

Gomes, H., Molholm, S., Ritter, W., Kurtzberg, D., Cowan, N., & Vaughan Jr., H.G. (2000). Mismatch negativity in children and adults, and effects of an attended task. Psychophysiology, 37, 807-816.

Gomot, M., Giard, M.-H., Roux, S., Barthelemy, C., & Bruneau, N. (2000). Maturation of frontal and temporal components of mismatch negativity (MMN) in children. Neuroreport, 11, 3109-3112.

Goswami, U. (2002). Phonology, reading development, and dyslexia: A cross-linguistic perspective. Annals of Dyslexia, 52, 141-163.

Goswami, U., Thomson, J., Richardson, U., Stainthorp, R., Hughes, D., Rosen, S., & Scott, S.K. (2002). Amplitude envelope onsets and developmental dyslexia: A new hypothesis. Proceedings of the National Academy of Sciences of the United States of America (PNAS), 99, 10911-10916.

Grigorenko, E.L. (2001). Developmental dyslexia: An update on genes, brains, and environments. Journal of Child Psychology and Psychiatry, 42, 91-125.

Grigorenko, E.L., Wood, F.B., Meyer, M.S., Hart, L.A., Speed, W.C., Shuster, A., & Pauls, D.L. (1997). Susceptibility loci for distinct components of developmental dyslexia on chromosomes 6 and 15. American Journal of Human Genetics, 60, 27-39.

Grigorenko, E.L., Wood, F.B., Meyer, M.S., & Pauls, D.L. (2000) Chromosome 6p influences on different dyslexia-related cognitive processes: further confirmation. American Journal of Human Genetics, 66, 715-723.

Grigorenko, E.L., Wood, F.B., Meyer, M.S., Pauls, J.E.D., Hart, L.A., & Pauls, D.L. (2001). Linkage studies suggest a possible locus for developmental dyslexia on chromosome 1p. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 105, 120-129.

Guttorm, T., Leppänen, P., Richardson, U., & Lyytinen, H. (2001). Event-related potentials and consonant differentiation in newborns with familial risk for dyslexia. Journal of Learning Disabilities, 34, 534-544.

Hallgren, B. (1950). Specific dyslexia: A clinical and genetic study. Acta Psychiatrica et Neurologica Scandinavia, 65, 1-287.

Hari, R., & Kiesilä, P. (1996). Deficit of temporal auditory processing in dyslexic adults. Neuroscience Letters, 205, 138-140.

Hari, R., Valta, M., & Uutela, K. (1999). Prolonged attentional dwell time in dyslexic adults. Neuroscience Letters, 271, 202-204.

Haslam, R.H., Dalby, J.T., Johns, R.D., & Rademaker, A.W. (1981). Cerebral asymmetry in developmental dyslexia, 38, 679-682.

Hatcher, P.J. & Hulme, C. (1999). Phonemes, rhymes, and intelligence as predictors of children’s responsiveness to remedial reading instruction: Evidence from a longitudinal intervention study. Journal of Experimental Child Psychology, 72, 130-153.

Hayduk, S., Bruck, M., & Cavanagh, P. (1996). Low level visual processing skills of adults and children with dyslexia. Cognitive Neuropsychology, 13, 975-1016.

He, C., Hotson, L., & Trainor, L.J. (2007). Mismatch responses to pitch changes in early infancy. Journal of Cognitive Neuroscience, 19, 878-892.

Heath, S.M., Hogben, J.H., & Clark, C.D. (1999). Auditory temporal processing in disabled readers with and without oral language delay. Journal of Child Psychology and Psychiatry, 40, 637-647.

Heiervang, E., Hughdahl, K., Steinmetz, H., Smievoll, A.I., Stevenson, J., Lund, A., Ersland, L., & Lundervold, A. (2000). Planum temporale, planum parietale and dichotic listening in dyslexia. Neuropsychologia, 38, 1704-1713.

Heiervang, E., Stevenson, J., & Hughdahl, K. (2002). Auditory processing in children with dyslexia. Journal of Child Psychology and Psychiatry, 43, 931-938.

Heim, S., Tschierse, J., Amunts, K., Wilms, M., Vossel, S., Willmes, K., Grabowska, A., & Huber, W. (2008). Cognitive subtypes of dyslexia. Acta Neurobiologicae Experimentalis, 68, 73-82.

Helenius, P., Tarkiainen, A., Cornelissen, P., Hansen, P.C., & Salmelin, R. (1999). Dissociation of normal feature analysis and deficient processing of letter-strings in dyslexic adults. Cerebral Cortex, 9, 476-483.

Herman, A.E., Galaburda, A.M., Fitch, R.H., Carter, A.R., & Rosen, G.D. (1997). Cerebral microgyria, thalamic cell size and auditory temporal processing in male and female rats. Cerebral Cortex, 7, 453-464.

Hier, D.B., LeMay, M., Rosenberger, P.B., & Perlo, V.P. (1978). Developmental dyslexia. Evidence for a subgroup with a reversal of cerebral asymmetry. Archives of Neurology, 35, 90-92.

Holopainen, I.E., Korpilahti, P., Juottonen, K., Lang, H., & Sillanpää, M. (1998). Abnormal frequency mismatch negativity in mentally retarded children and in children with developmental dysphasia. Journal of Child Neurology, 13, 178-183.

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, 21, 8939-8944.

Hugdahl, K., Heiervang, E., Nordby, H., Smievoll, A.I., Steinmetz, H., Stevenson, J., & Lund, A. (1998). Central auditory processing, MRI morphometry and brain laterality: applications to dyslexia. Scandinavian Audiology, 27 (suppl. 49), 26-34.

Humphreys, P., Kaufmann, W.E., & Galaburda, A.M. (1990). Developmental dyslexia in women: Neuropathological findings in three patients. Annals of Neurology, 28, 727-738.

Huttunen, T., Halonen, A., Kaartinen, J., & Lyytinen, H. (2007). Does mismatch negativity show differences in reading-disabled children compared to normal children and children with attention deficit? Developmental Neuropsychology, 31, 453-470.

Huttunen-Scott, T., Kaartinen, J., Tolvanen, A., & Lyytinen, H. (2008). Mismatch negativity (MMN) elicited by duration deviations in children with reading disorder, attention deficit or both. International Journal of Psychophysiology, 69, 69-77.

Huttunen-Scott, T., Kaartinen, J., Tolvanen, A., Richardson, U., & Lyytinen, H. (submitted). A study of electrophysiological and behavioral aspects of auditory duration discrimination in children with reading disorder, attention deficit or both.

Hynd, G.W., Hall, J., Novey, E.S., Eliopulos, D., Black, K., Gonzalez, J.J., Edmonds, J.E., Riccio, C., & Cohen, M. (1995). Dyslexia and corpus callosum morphology, 52, 32-38.

Hynd, G.W., Semrud-Clikeman, M., Lorys, A.R., Novey, E.S., & Eliopulos, D. (1990). Brain morphology in developmental dyslexia and attention deficit disorder/hyperactivity. Archives of Neurology, 47, 919-926.

Jenner, A.R., Rosen, G.D., Galaburda, A.M. (1999). Neuronal asymmetries in primary visual cortex of dyslexic and nondyslexic brains. Annals of Neurology, 46, 189-196.

Johnston, A., Bruno, A., Watanabe, J., Quansah, B., Patel, N., Dakin, S., & Nishida, S. (2008). Visually-based temporal distortion in dyslexia. Vision research, doi:10.1016/j.visres.2008.04.029.

Jääskeläinen, I.P., Ahveninen, J., Bonmassar, G., Dale, A.M., Ilmoniemi, R.J., Levänen, S., Lin, F.-H., May, P., Melcher, J., Stufflebeam, S., Tiitinen, H., & Belliveau, J.W. (2004). Human posterior auditory cortex gates novel sounds to consciousness. PNAS, 101, 6809-6814.

Kemner, C., Verbaten, M.N., Koelega, H.S., Buitelaar, J.K., van der Gaag, R.J., Camfferman, G., & van Engeland, H. (1996). Event-related brain potentials in children with attention-deficit and hyperactivity disorder: Effects of stimulus deviancy and task relevance in the visual and auditory modality. Biological Psychiatry, 40, 522-534.

Kilpeläinen, R., Partanen, J., & Karhu, J. (1999). Reduced mismatch negativity (MMN) suggests deficits in preattentive auditory processing in distractible children. Neuroreport, 10, 3341-3345.

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.

Klicpera, C., Wolff, P.H., & Drake, C. (1981). Bimanual co-ordination in adolescent boys with reading retardation. Developmental Medicine and Child Neurology, 23, 617-625.

Klingberg, T., Hedehus, M., Temple, E., Salz, T., Gabrieli, J.D.E., Moseley, M.E., & Poldrack, R.A. (2000). Microstructure of temporo-parietal white matter as a basis for reading ability: Evidence from diffusion tensor magnetic resonance imaging. Neuron, 25, 493-500.

Knivsberg, A.-M. & Andreassen, A.B. (2008). Behaviour, attention and cognition in severe dyslexia. Nordic Journal of Psychiatry, 62, 59-65.

Knopik, V.S., Smith, S.D., Cardon, L., Pennington, B., Gayan, J., Olson, R.K., & DeFries, J.C. (2002). Differential genetic etiology of reading component processes as a function of IQ. Behavior Genetics, 32, 181-198.

Korpilahti, P. (1995). Auditory discrimination and memory functions in SLI children: A comprehensive study with neurophysiological and behavioural methods. Logopedics Phoniatrics Vocology, 20, 131-139.

Korpilahti, P., Krause, C.M., Holopainen, I., & Lang, H. (2001). Early and late mismatch negativity elicited by words and speech-like stimuli in children. Brain and Language, 76, 332-339.

Korpilahti, P. & Lang, H.A. (1994). Auditory ERP components and mismatch negativity in dysphasic children. Electroencephalography and Clinical Neurophysiology, 91, 256-264.

Korzyukov, O., Alho, K., Kujala, A., Gumenyuk, V., Ilmoniemi, R.J., Virtanen, J., Kropotov, J., & Näätänen, R. (1999). Electromagnetic responses of the human auditory cortex generated by sensory-memory based processing of tone-frequency changes. Neuroscience Letters, 276, 169-172.

Kraus, N., Koch, D.B., McGee, T.J., Nicol, T.G., & Cunningham, J. (1999). Speech-sound discrimination in school-age children. Journal of Speech, Language, and Hearing Research, 42, 1042-1060.

Kraus, N., McGee, T., Carrell, T., Sharma, A., Micco, A., & Nicol, T. (1993). Speech-evoked cortical potentials in children. Journal of American Academy of Audiology, 4, 238-248.

Kraus, N., McGee, T., Carrell, T.D., King, C., Tremblay, K., & Nicol, T. (1995). Central auditory system plasticity associated with speech discrimination training. Journal of Cognitive Neuroscience, 16, 25-32.

Kraus, N., McGee, T.J., Carrell, T.D., Zecker, S.G., Nicol, T.G., & Koch, D.B. (1996). Auditory neurophysiologic responses and discrimination deficits in children with learning problems. Science, 273, 971-973.

Kronbichler, M., Hutzler, F., Staffen, W., Mair, A., Ladurner, G., & Wimmer, H. (2006). Evidence for a dysfunction of left posterior reading areas in German dyslexic readers. Neuropsychologia, 44, 1822-1832.

Kujala, T., Belitz, S., Tervaniemi, M., & Näätänen, R. (2003). Auditory sensory memory disorder in dyslexic adults as indexed by the mismatch negativity. European Journal of Neuroscience, 17, 1323-1327.

Kujala, T., Halmetoja, J., Näätänen, R., Alku, P., Lyytinen, H., & Sussman, E. (2006). Speech- and sound-segmentation in dyslexia: evidence for a multiple-level cortical impairment. European Journal of Neuroscience, 24, 2420-242.

Kujala, T., Karma, K., Ceponiene, R., Belitz, S., Turkkila, P., Tervaniemi, M., & Näätänen, R. (2001). Plastic neural changes and reading improvement caused by audiovisual training in reading-impaired children. Proceedings of the National Academy of Sciences of the United States of America, 28, 10509-10514.

Kujala, T., Lovio, R., Lepistö, T., Laasonen, M., & Näätänen, R. (2006). Evaluation of multi-attribute auditory discrimination in dyslexia with the mismatch negativity. Clinical Neurophysiology, 117, 885-893.

Kujala, T., Myllyviita, K., Tervaniemi, M., Alho, K., Kallio, J., & Näätänen, R. (2000). Basic auditory dysfunction in dyslexia as demonstrated by brain activity measurements. Psychophysiology, 37, 262-266.

Kushnerenko, E., Ceponiené, R., Balan, P, Fellman, V., & Näätänen, R. (2002). Maturation of the auditory change detection response in infants: a longitudinal ERP study. Neuroreport, 13, 1843-1848.

Lachmann, T., Berti, S., Kujala, T., & Schröger, E. (2005). Diagnostic subgroups of developmental dyslexia have different deficits in neural processing of tones and phonemes. International Journal of Psychophysiology, 56, 105-120.

Lambert, N.M., Sandoval, J., & Sassone. D. (1978). Prevalence of hyperactivity in elementary school children as a function of social system definers. American Journal of Orthopsychiatry, 48, 446-463.

Landerl, K. (1997). Word recognition in English and German dyslexics: a direct comparison. In Leong, C.K., Joshi, R.M. (Eds.), Cross-language Studies of Learning to Read and Spell: Phonologic and Orthographic Processing (pp. 121-137). Dordrecth, The Netherlands: Kluwer Academic Publishers.

Larsen, J.P., Høien, T., Lundberg, I., & Odegaard, H. (1990). MRI evaluation of the size and symmetry of the planum temporale in adolescents with developmental dyslexia. Brain and Language, 39, 289-301.

Larsen, J.P., Høien, T., & Ødegaard, H. (1992). Magnetic resonance imaging of the corpus callosum in developmental dyslexia. Cognitive Neuropsychology, 9, 123-134.

Laycock, R., & Crewther, S.G. (2008). Towards an understanding of the role of the 'magnocellular advantage' in fluent reading. Neuroscience and Biobehavioral Reviews, doi:10.1016/j.neubiorev.2008.06.002.

Leonard, C.M. (2001). Imaging brain structure in children: differentiating language disability and reading disability. Learning Disability Quarterly, 24, 158-176.

Leonard, C.M., Eckert, M.A., Lombardino, L.J., Oakland, T., Kranzler, J., Mohr, C.M., King, W.M., & Freeman, A. (2001). Anatomical risk factors for phonological dyslexia. Cerebral Cortex, 11, 148-157.

Leonard, C.M., Lombardino, L.J., Walsh, K., Eckert, M.A., Mockler, J.L., Rowe, L.A., Williams, S., & DeBose, C.B. (2002). Anatomical risk factors that distinguish dyslexia from SLI predict reading skill in normal children. Journal of Communication Disorders, 35, 501-531.

Leonard, C.M., Voeller, K.K., Lombardino, L.J., Morris, M.K., Hynd, G.W., Alexander, A.W., Andersen, H.G., Garofalakis, M., Honeyman, J.C., Mao, J., Agee, O.F., & Staab, E.V. (1993). Anomalous cerebral structure in dyslexia revealed with magnetic resonance imaging. Archives of Neurology, 50, 461-469.

Leppänen, P.H.T., Eklund, K.M., & Lyytinen, H. (1997). Event-related brain potentials to change in rapidly presented acoustic stimuli in newborns. Developmental Neuropsychology, 13, 175-204.

Leppänen, P.H.T., Guttorm, T.K., Pihko, E., Takkinen, S., Eklund, K.M., & Lyytinen, H. (2004). Maturational effects on newborn ERPs measured in the mismatch negativity paradigm. Experimental Neurology, 190, 91-101.

Leppänen, P.H.T., Pihko, E., Eklund, K.M., & Lyytinen, H. (1999) Cortical responses of infants with and without a genetic risk for dyslexia: II. Group effects. Neuroreport, 10, 969-973.

Leppänen, P.H.T., Richardson, U., Pihko, E., Eklund, K.M., Guttorm, T.K., Aro, M., & Lyytinen, H. (2002). Brain responses to changes in speech sound durations differ between infants with and without familial risk for dyslexia. Developmental Neuropsychology, 22, 407-422.

Licht, R., & Horsley, T.M. (1998). Mismatch negativity as a tool in studying auditory/phonological processing in reading disability. In R. Licht, A. Bouma, W. Slot, & W. Koobs (Eds.), Child neuropsychology, reading disability and more (pp. 85-105). Delft, The Netherlands: Uitgeverij Eburon.

Livingstone, M.S., & Hubel, D.H. (1987). Psychophysical evidence for separate channels for the perception of form, color, movement, and depth. The Journal of Neuroscience, 7, 3416-3468.

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.

Lovegrove, W.J., Bowling, A., Badcock, D., & Blackwood, M. (1980). Specific reading disability: differences in contrast sensitivity as a function of spatial frequency. Science, 210, 439-440.

Lovegrove, W., Martin, F., Bowling, A., Blackwood, M., Badcock, D., & Paxton, S. (1982). Contrast sensitivity functions and specific reading disability. Neuropsychologia, 20, 309-315.

Lyon, G.R., Shaywitz, S.E., Shaywitz, B.A. (2003). A definition of dyslexia. Annals of Dyslexia, 53, 1-14.

Lyytinen, H., Ahonen, T., Aro, M., Aro, T., Närhi, V., & Räsänen, P., 1998. Learning disabilities: A view of developmental neuropsychology. In Licht, R., Bouma, A., Slot, W. & Koobs, W. (Eds.), Child Neuropsychology. Reading Disability and more…(pp. 85-105). Delft: Uitgeverij Eburon.

Lyytinen, H., Guttorm, T.K., Huttunen, T., Hämäläinen, J., Leppänen, P.H.T., & Vesterinen, M. (2005). Psychophysiology of developmental dyslexia: a review of findings including studies of children at risk for dyslexia. Journal of Neurolinguistics, 18, 167-195.

Lyytinen, G.R., Leinonen, S., Nikula, M., Aro, M., & Leiwo, M. (1995). In search of the core features of dyslexia: observations concerning dyslexia in the highly orthographically regular Finnish language. In Berninger, V.W. (Ed.), The Varieties of Ortographic Knowledge II: Relationships to Phonology, Reading and Writing (pp. 177-204). Dordrecht, The Netherlands: Kluver Academic Publishers.

Martin, F. & Lovegrove, W. (1984). The effects of field size and luminance on contrast sensitivity differences between specifically reading disabled and normal children. Neuropsychologia, 22, 72-77.

Martin, F. & Lovegrove, W. (1987). Flicker contrast sensitivity in normal and specifically disabled readers. Perpeption, 16, 215-221.

Martin, B.A., Shafer, V.L., Morr, M.L., Kreuzer, J.A., & Kurtzberg, D. (2003). Maturation of mismatch negativity: A scalp current density analysis. Ear & Hearing, 24, 463-471.

Martos, F.J. & Vila, J. (1990). Differences in eye movements control among dyslexic, retarded and normal readers in the Spanish population, Reading and Writing, 2, 175-188.

Martynova, O., Kirjavainen, J., & Cheour, M. (2003). Mismatch negativity and late discriminative negativity in sleeping human newborns. Neuroscience Letters, 340, 75-78.

Maurer, U., Bucher, K., Brem, S., & Brandeis, D. (2003). Altered responses to tne and phoneme mismatch in kindergartners at familial dyslexia risk. Neuroreport, 14, 2245-2250.

May, J.G., Lovegrove, W.J., Martin, F., & Nelson, P. (1991). Pattern elicited visual evoked potentials in good and poor readers. Clinical Vision Science, 6, 131-136.

McArthur, G.M. & Bishop, D.V.M. (2004). Which people with specific language impairment have auditory processing deficits. Cognitive Neuropsychology, 21, 79-94.

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.

McCrory, E.J., Mechelli, A., Frith, U., & Price, C.J. (2005). More than words: a common neural basis for reading and naming deficits in developmental dyslexia? Brain, 128, 261-267.

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.

Meyler, A., Keller, T.A., Cherkassky, V.L., Lee, D., Hoeft, F., Whitfield-Gabrieli, S., Gabrieli, J.D.E., & Just, M.A. (2007). Brain activation during sentence comprehension among good and poor readers. Cerebral Cortex, 17, 2780-2787.

Mody, M. (2003). Phonological basis in reading disability: A review and analysis of the evidence. Reading and Writing, 16, 21-39.

Mody, M., Studdert-Kennedy, M., & Brady, S. (1997). Speech perception deficits in poor readers: Auditory processing or phonological coding? Journal of Experimental Child Psychology, 64, 199-231.

Moore, C.J., & Price, C.J. (1999). Three distinct ventral occipitotemporal regions for reading and object naming. NeuroImage, 10, 181-192.

Morris, D.W., Robinson, L., Turic, D., Duke, M., Webb, V., Milham, C., Hopkin, E., Pound, K., Fernando, S., Easton, M., Hamshere, M., Williams, N., McGuffin, P., Stevenson, J., Krawczak, M., Owen, M.J., O'Donovan, M.C., Williams, J. (2000). Family-based association mapping provides evidence for a gene for reading disability on chromosome 15q. Human Molecular Genetics, 9, 843-848.

Nagarajan, S., Mahncke, H., Salz, T., Tallal P., Roberts, T., & Merzenich, M.M. (1999). Cortical auditory signal processing in poor readers. Proceedings of the National Academy of Sciences of the United States of America, 96, 6483-6488.

Nashida, T., Yabe, H., Sato, Y., Hiruma, T., Sutoh, T., Shinozaki, N., Kaneko, S. (2000). Automatic auditory information processing in sleep. Sleep, 15, 821-828.

Nation, K., Adams, J.W., Bowyer-Crane, C.A., & Snowling, M. (1999). Working memory deficits in poor comprehenders reflect underlying language impairments. Journal of Experimental Child Psychology, 73, 139-158.

Nicolson, R.I. & Fawcett, A.J. (1994). Comparison of deficits in cognitive and motor skills among children with dyslexia. Annals of Dyslexia, 44, 147-164.

Nicolson, R.I., Fawcett, A.J., & Dean, P. (2001). Developmental dyslexia: the cerebellar deficit hypothesis. Trends in Neurosciences, 24, 508-511.

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. The Lancet, 353, 1662-1667.

Nopola-Hemmi, J., Myllyluoma, B., Haltia, T., Taipale, M., Ollikainen, V., Ahonen, T., Voutilainen, A., Kere, J., & Widén, E. (2001). A dominant gene for developmental dyslexia on chromosome 3. Journal of Medical Genetics, 38, 658-664.

Novitski, N., Tervaniemi, M., Huotilainen, M., & Näätänen, R. (2004). Frequency discrimination at different frequency levels as indexed by electrophysiological and behavioral measures. Cognitive Brain Research, 20, 26-36.

Näätänen, R. (1992). Attention and brain function. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Näätänen, R., Gaillard, A.W.K., & Mäntysalo, S. (1978). Early selective attention effect on evoked potential reinterpreted. Acta Psychologica, 42, 313-329.

Näätänen, R., Jacobsen, T., & Winkler, I. (2005). Memory-based or afferent processes in mismatch negativity (MMN): A review of the evidence, Psychophysiology, 42, 25-32.

Näätänen, R., Lehtokoski, A., Lennes, M., Cheour, M., Huotilainen, M., Ilvonen, A., Vainio, M., Alku, P., Ilmoniemi, R., Luuk, A., Allik, J., Sinkkonen, J., & Alho, K. (1997). Language-specific phoneme representations revealed by electric and magnetic brain responses. Letters to Nature, 385, 432-434.

Näätänen, R. & Michie, P.T. (1979). Early selective attention effects on the evoked potential: a critical review and reinterpretation. Biological Psychology, 8, 81-136.

Näätänen, R., Paavilainen, P., & Reinikainen, K. (1989). Do event-related potentials to infrequent decrements in duration of auditory stimuli demonstrate a memory trace in man? Neuroscience Letters, 15, 347-352.

Näätänen, R., Paavilainen, P., Tiitinen, H., Jiang, D., & Alho, K. (1993). Attention and mismatch negativity. Psychophysiology, 30, 436-450.

Näätänen, R., Schröger, E., Karakas, S., Tervaniemi, M., & Paavilainen, P. (1993). Development of a memory trace for a complex sound in the human brain. Neuroreport, 4, 503-506.

Nöthen, M.M., Schulte-Körne, G., Grimm, T., Cichon, S., Vogt, I.R., Müller-Myhsok, B., Propping, P., & Remschmidt, H. (1999). Genetic linkage analysis with dyslexia: Evidence for linkage of spelling disability to chromosome 15. European Child & Adolescent Psychiatry, 8, 56-59.

Oades, R.D., Dittmann-Balcar, A., Schepker, R., Eggers, C., & Zerbin, D. (1996). Auditory event-related potentials (ERPs) and mismatch negativity (MMN) in healthy children and those with attention-deficit or tourette/tic symptoms. Biological Psychology, 43, 163-185.

Oceák, A., Winkler, I., Sussman, E., & Alho, K. (2006). Loudness summation and the mismatch negativity event-related brain potential in humans. Psychophysiology, 43, 13-20.

Olson, R.K. & Datta, H. (2002). Visual-temporal processing in reading-disabled and normal twins. Reading and Writing, 15, 127-149.

Olson, R.K., Datta, H., Gayan, J., & DeFries, J.C. (1999). A behavioral-genetic analysis of reading disabilities and component processes. In R.M. Klein & P.A. McMullen (Eds.), Converging Methods for Understanding Reading and Dyslexia (pp. 133-151). Cambridge, MA: MIT Press.

Omtzigt, D., Hendriks, A.W., & Kolk, H.H.J. (2002). Evidence for magnocellular involvement in the identification of flanked letters. Neuropsychologia, 40, 1881-1890.

Opitz, B., Schröger, E., & von Cramon, Y. (2005). Sensory and cognitive mechanisms for preattentive detection in auditory cortex. European Journal of Neuroscience, 21, 531-535.

Orton, S. T. (1925). "Word-blindness" in school children. Archives of Neurology and Psychiatry. 14, 5, 581-615.

Paavilainen, P., Arajärvi, P., & Takegata, R. (2007). Preattentive detection of nonsalient contingencies between auditory features. Neuroreport, 18, 159-163.

Pakarinen, S., Takegata, R., Rinne, T., Huotilainen, M., & Näätänen, R. (2007). Measurement of extensive auditory discrimination profiles using the mismatch negativity (MMN) of the auditory event-related potential (ERP). Clinical Neurophysiology, 118, 177-185.

Pang, E.W., Edmonds, G.E., Desjardins, R., Khan, S.C., Trainor, L.J., & Taylor, M.J. (1998). Mismatch negativity to speech stimuli in 8-month-old infants and adults. International Journal of Psychophysiology, 29, 227-236.

Paul, I., Bott, C., Heim, S., Wienbruch, C., & Elbert, T.R. (2006). Phonological but not auditory discrimination is impaired in dyslexia. European Journal of Neuroscience, 24, 2945-2953.

Paulesu, E., Frith, U., Snowling, M., Gallagher, A., Morton, J., Frackowiak, R.S., Frith, C.D. (1996). Is developmental dyslexia a connection syndrome? Evidence from PET scanning. Brain, 119, 143-157.

Paulesu, E., Demonet, J.F., Fazio, F., McCrory, E., Chanoine, V., Brunswick, N., Cappa, S.F., Cossu, G., Habib, M., Frith, C.D., & Frith, U. (2001). Dyslexia: cultural diversity and biological unity. Science, 291, 2165-2167.

Pennington, B.F. (1990). Annotation: the genetics of dyslexia. Journal of Child Psychology and Psychiatry, 31, 193-201.

Pennington, B.F. (2006). From single to multiple deficit models of developmental disorders. Cognition, 101, 385-413.

Pennington, B.F., Gilger, J.W., Pauls, D., Smith, S.A., Smith, S.D., & DeFries, J.C. (1991). Evidence for major gene transmission of developmental dyslexia. The Journal of the American Medical Association (JAMA), 266, 1527-1534.

Penolazzi, B., Spinorelli, C., & Angrilli, A. (2008). Delta EEG activity as a marker of dysfunctional linguistic processing in developmental dyslexia. Psychophysiology, doi:10.1111/j.1469-8986.2008.00709.x.

Petryshen, T.L., Kaplan, B.J., Hughes, M.L., Tzenova, J., & Field, L.L. (2002). Supportive evidence for the DYX3 dyslexia susceptibility gene in Canadian families. Journal of Medical Genetics, 39, 125-126.

Picton, T.W., Alain, C., Otten, L., Ritter, W., & Achim, A. (2000). Mismatch negativity: Different water in the same river. Audiology & Neurotology, 5, 111-139.

Pihko, E;, Leppänen, P.H.T., Eklund, K., Cheour, M., Guttorm, T., & Lyytinen, H. Cortical responses of infants with and without a genetic risk for dyslexia: I. Age effects. Neuroreport, 10, 901-905.

Plante, E., Swisher, L., Vance, R., & Rapcsak, S. (1991). MRI findings in boys with specific language impairment. Brain and Language, 41, 52-66.

Polanczyk, G., de Lima, M.S., Horta, B.L., Biederman, J., & Rohde, L.A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. American Journal of Psychiatry, 164, 942-948.

Pugh, K.R., Shaywitz, B.A., Shaywitz, S.E., Constable, R.T., Skudlarski, P., Fulbright, R.K., Bronen, R.A., Shankweiler, D.P., Katz, L., Fletcher, J.M., & Gore, J.C. (1996). Cerebral organization of component processes in reading. Brain, 119, 1221-1238.

Pugh, K.R., Mencl, W.E., Shaywitz, B.A., Shaywitz, S.E., Fulbright, R.K., Constable, R.T., Skudlarski, P., Marchione, K.E., Jenner, A.R., Fletcher, J.M., Liberman, A.M., Purvis, K.L. & Tannock, R. (1997). Language abilities in children with attention deficit hyperactivity disorder, reading disabilities, and normal controls. Journal of Abnormal Child Psychology, 25, 133-144.

Purvis, K.L. & Tannock, R. (2000). Phonological processing, not inhibitory control, differentiates ADHD and reading disability. Journal of the American Academy of Child and Adolescent Psychiatry, 39, 485-494.

Rabin, M., Wen, X.L., Hepburn, M., Lubs, H.A., Feldman, E., & Duara, R. (1993). Suggestive linkage of developmental dyslexia to chromosome 1p34-p36. Lancet, 342, 178-179.

Ramus, F., Rosen, S., Dakin, S. C., Day, B.L., Castellote, J.M., White, S., & Frith, U. (2003). Theories of developmental dyslexia: Insights form a multiple case study of dyslexic adults. Brain, 126, 841-865.

Reed, M.A. (1989). Speech perception and the discrimination of brief auditory cues in reading-disabled children. Journal of Experimental Child Psychology, 48, 270-292.

Richards, T.L., Corina, D., Serafini, S., Steury, K., Echelard, D.R., Dager, S.R., Marro, K., Abbott, R.D., Maravilla, K.R., & Berninger, V.W. (2000). Effects of phonologically driven treatment for dyslexia on lactate levels measured by proton MR spectroscopic imaging. American Journal of Neuroradiology, 21, 916-922.

Richardson, U. (1998). Familial dyslexia and sound duration in the quantity distinctions of Finnish infants and adults (Doctoral dissertation). Studia Philological Jyväskyläensia, University of Jyväskylä.

Richardson, U., Leppänen, P.H.T., Leiwo, M., & Lyytinen, H. (2003). Speech perception of infants with familial risk for dyslexia differ at the age of 6 months. Developmental Neuropsychology, 23, 385-397.

Ridder, W.H.3rd, Borsting, E., Cooper, M., McNeel, B., & Huang, E. (1997). Not all dyslexics are created equal. Optometry and Vision Science, 74, 99-104.

Robichon, F. & Habib, M. (1998). Abnormal callosal morphology in male adult dyslexics: relationships to handedness and phonological abilities. Brain and Language, 62, 127-146.

Robichon, F., Levrier, O., Farnarier, P., & Habib, M. (2000). Developmental dyslexia: atypical cortical asymmetries and functional significance. European Journal of Neurology, 7, 35-46.

Rosenberger, P.B., & Hier, D.B. (1980). Cerebral asymmetry and verbal intellectual deficits. Annals of Neurology, 8, 300-304.

Rumsey, J.M., Andreason, P., Zametkin, A.J., Aquino, T., King, A.C., Hamburger, S.D., Pikus, A., Rapoport, J.L., & Cohen, R.M. (1992). Failure to activate the left temporoparietal cortex in dyslexia. An oxygen 15 positron emission tomographic study. Archives of Neurology, 49, 527-534.

Rumsey, J.M., Casanova, M., Mannheim, G.B., Patronas, N., DeVaughn, N., Hamburger, S.D., & Aquino, T. (1996). Corpus callosum morphology, as measured with MRI, in dyslexic men. Biological Psychiatry, 39, 769-775.

Rumsey, J.M., Donohue, B.C., Brady, D.R., Nace, K., Giedd, J.N., & Andreason, P. (1997a). A magnetic resonance imaging study of planum temporale asymmetry in men with developmental dyslexia. Archives of Neurology, 54, 1481-1489.

Rumsey, J.M.,, Nace, K., Donohue, B., Wise, D., Maisog, J.M., & Andreason, P. (1997b). A positron emission tomographic study of impaired word recognition and phonological processing in dyslexic men. Archives of Neurology, 54, 562-573.

Sabri, M., Liebenthal, E., Waldron, E.J., Medler, D.A., & Binder, J.R. (2006). Attentional modulation in the detection of irrelevant deviance: a simultaneous ERP/fMRI study. Journal of Cognitive Neuroscience, 18, 689-700.

Sallinen, M., Kaartinen, J., & Lyytinen, H. (1994). Is the appearance of mismatch negativity during stage 2 sleep related to elicitation of K-complex? Electroencephalography and Clinical Neurophysiology, 91, 140-148.

Sams, M., Hari, R., Rif, J., & Knuutila, J. (1993). The human auditory sensory memory trace persists about 10 sec: Neuromagnetic evidence. Journal of Cognitive Neuroscience, 5, 363-370.

Scarborough, H.S. (1989). Prediction of reading disability from familial and individual differences . Journal of Educational Psychology, 81, 101-108.

Schulte-Körne, G., Deimel, W., Bartling, J., & Remschmidt, H. (1998). Auditory processing and dyslexia: evidence for a specific speech processing deficit. Neuroreport, 9, 337-340.

Schulte-Körne, G., Deimel, W., Bartling, J., & Remschmidt, H. (1999). Pre-attentive processing of auditory patterns in dyslexic human subjects. Neuroscience Letters, 276, 41-44.

Schulte-Körne, G., Deimel, W., Bartling, J, & Remschmidt, H. (2001). Speech perception deficit in dyslexic adults as measured by mismatch negativity (MMN). International Journal of Psychophysiology, 40, 77-87.

Schultz, R.T., Cho, N.K., Staib, L.H., Kier, L.E., Fletcher, J.M., Shaywitz, S.E., Shankweiler, D.P., Katz, L., Gore, J.C., Duncan, J.S., & Shaywitz, B.A. (1994). Brain morphology in normal and dyslexic children: The influence of sex and age. Annals of Neurology, 35, 732-742.

Schulz, E., Maurer, U., van der Mark, S., Bucher, K., Brem, S., Martin, E., & Brandeis, D. (2008). Impaired semantic processing during sentence reading in children with dyslexia: Combined fMRI and ERP evidence. NeuroImage, doi:10.1016/j. neuroimage.2008.02.012.

Schumacher, J., König, I.R., Schröder, T., Duell, M., Plume, E., Propping, P., Warnke, A., Libertus, C., Ziegler, A., Müller-Myhsok, B., Schulte-Körne, G., Nöthen, M.M. (2008). Further evidence for a susceptibility locus contributing to reading disability on chromosome 15q15-q21. Psychiatric Genetics, 18, 137-142.

Serrano, F. & Defior, S. (2008). Dyslexia speed problems in a transparent orthography. Annals of Dyslexia, 58, 81-95.

Shafer, V.L., Morr, M.L., Datta, H., Kurtzberg, D., & Schwartz, R.G. (2005). Neurophysiological indexes of speech processing deficits in children with specific language impairment. Journal of Cognitive Neuroscience, 17, 1168-1180.

Shafer, V.L., Morr, M.L., Kreuzer, J.A., & Kurtzberg, D. (2000). Maturation of mismatch negativity in school-age children. Ear and Hearing, 21, 242-251.

Shanahan, M.A., Pennington, B.F., Yerys, B.E., Scott, A., Boada, R., Willcutt, E.G., Olson, R.K., & DeFries, J.C. (2006). Processing speed deficits in attention deficit/hyperacitivity disorder and reading disability. Journal of Abnormal Child Psychology, 34, 584-601.

Sharma, M., Purdy, S.C., Newall, P., Wheldall, K., Beaman, R., & Dillon, H. (2006). Electrophysiological and behavioral evidence of auditory processing deficits in children with reading disorder. Clinical Neurophysiology, 117, 1130-1144.

Shaywitz, S.E., Shaywitz, B.A., Fletcher, J.M., & Escobar, M.D. (1990). Prevalence of reading disability in boys and girls. Reults of the Connecticut longitudinal study. JAMA, 264, 998-1002.

Shaywitz, S.E., Shaywitz, B.A., Pugh, K.R., Fulbright, R.K., Constable, R.T., Mencl, W.E., Shankweiler, D.P., Liberman, A.M., Skudlarski, P., Fletcher, J.M., Katz, L., Marchione, K.E., Lacadie, C., Gatenby, C., & Gore, J.C. (1998). Functional disruption in the organization of the brain for reading in dyslexia. Proceedings of the National Academy of Sciences (PNAS), 3, 2636-2641.

Shaywitz, B.A., Shaywitz, S.E., Pugh, K.R., Mencl, W.E., Fulbright, R.K., Skudlarski, P., Constable, R.T., Marchione, K.E., Fletcher, J.M., Lyon, G.R., & Gore, J.C. (2002). Disruption of posterior brain systems for reading in children with developmental dyslexia. Biological Psychiatry, 52, 101-110.

Simos, P.G., Breier, J.I., Fletcher, J.M., Foorman, B.R., Bergman, E., Fishbeck, K., & Papanicolaou, A.C. (2000). Brain activation profiles in dyslexic children during non-word reading: a magnetic source imaging study. Neuroscience Letters, 290, 61-65.

Simos, P.G., Breier, J.I., Fletcher, J.M., Foorman, B.R., Castillo, E.M., & Papanicolaou, A.C. (2002). Brain mechanisms for reading words and pseudowords: an integrated approach. Cerebral Cortex, 12, 297-305.

Skottun, B.C. & Skoyles, J.R. (2008). Coherent motion, magnocellular sensitivity and the causation of dyslexia. The International Journal of Neuroscience, 118, 185-190.

Slaghuis, W.L. & Ryan, J.F. (1999). Spatio-temporal contrast sensitivity, coherent motion, and visible persistence in developmental dyslexia. Vision Research, 39, 651-668.

Smith, S.D., Kimberling, W.J., Pennington, B.F., & Lubs, H.A. (1983). Specific reading disability: Identification of an inherited form through linkage analysis. Science, 219, 1345-1347.

Smith, A., Taylor, E., Rogers, J.W., Newman, S., & Rubia, K. (2002). Evidence for a pure time perception deficit in children with ADHD. Journal of Child Psychology & Psychiatry and Allied Disciplines, 43, 529-542.

Spinelli, D., Angelelli, P., De Luca, M., Di Pace, E., Judica, A., Zoccolotti, P. (1997). Developmental surface dyslexia is not associated with deficits in the transient visual system. Neuroreport, 8, 1807-1812.

Stack, J.W. & Dalebout, S.D. (1998). The effect of perceptual training on the identification of a non-native phoneme contrast and its relationship to the MMN auditory evoked potential. The Journal of the Acoustical Society of America, 104, 1854.

Steffens, M.L., Eilers, R.E., Gross-Glenn, K., & Jallad, B. (1992). Speech perception in adult subjects with familial dyslexia. Journal of speech and hearing research, 35, 192-200.

Stein, J. & Walsh, V. (1997). To see but not to read; the magnocellular theory of dyslexia. Trends in Neurosciences, 20, 147-152.

Steinbrink, C., Vogt, K., Kastrup, A., Müller, H.-P., Juengling, F.D., Kassubek, J., & Riecker, A. (2008). The contribution of white and gray matter differences to developmental dyslexia: Insights from DTI and VBM at 3.0 T. Neuropsychologia, 46, 13, 3170-3178.

Studdert-Kennedy, M. (2002). Deficits in phoneme awareness do not arise from failures in rapid auditory processing. Reading and Writing, 15, 5-14.

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.

Szymanski, M.D., Yund, E.W., & Woods, D.L. (1999). Phonemes, intensity and attention: differential effects on the mismatch negativity (MMN). Journal of the Acoustical Society of America, 106, 3492-3505.

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.

Talcott, J.B., Witton, C., McLean, M.F., Hansen, P.C., Rees, A., Green, G.G.R., & Stein, J.F. (2000). Dynamic sensory sensitivity and children's word decoding skills. Proceedings of the National Acadamy of Sciences of the United States of America (PNAS), 97, 2952-2957.

Tallal, P. (1980). Auditory temporal perception, phonics and reading disabilities in children. Brain and Language, 9, 182-198.

Tallal, P. (1984). Temporal or phonetic processing deficit in dyslexia? Applied Psycholinguistics, 5, 167-169.

Tallal, P. (2000). Experimental studies of language learning impairments: From research to remediation. In D.V.M. Bishop & L.B. Leonard (Eds.), Speech and Language Impairments in Children: Causes, Characteristics, Intervention and Outcome (pp. 131-156). East Sussex, UK: Psychology Press.

Tallal, P. & Benasich, A.A. (2002). Developmental language learning impairments. Development and Psychopathology, 14, 559-579.

Tallal, P., Miller, S.L., Bedi, G., Byma, G., Wang, X., Nagarajan, S.S., Schreiner, C., Jenkins, W.M., & Merzenich, M.M. (1996). Language comprehension in language-learning impaired children improved with acoustically modified speech. Science, 271, 81-84.

Tallal, P. & Piercy, M. (1974). Developmental aphasia: rate of auditory processing and selective impairment of consonant perception. Neuropsychologia, 12, 83-93.

Tallal, P., Sainburg, R.L., & Jernigan, T. (1991). The neuropathology of developmental dysphasia: Behavioral, morphological, and physiological evidence for a pervasive temporal processing disorder. Reading & Writing, 3, 363-377.

Tallal, P. & Stark, R.E. (1981). Speech acoustic-cue discrimination abilities of normally developing and language-impaired children. The Journal of Acoustical Society of America, 69, 568-574.

Tallal, P. & Stark, R.E. (1982). Perceptual/motor profiles of reading impaired children with and without concomitant oral language deficits. Annals of Dyslexia, 32, 163-176.

Tallal, P., Stark, R.E., Kallman, C., & Mellits, D. (1980). Developmental dysphasia: relation between acoustic processing deficits and verbal processing. Neuropsychologia, 18, 273-284.

Temple, E., Deutsch, G.K., Poldrack, R.A., Miller, S.L., Tallal, P., Merzenich, M.M., & Gabrieli, J.D.E. (2003). Neural deficits in children with dyslexia ameliorated by behavioral remediation: Evidence from functional MRI. Proceedings of the National Academy of Sciences (PNAS), 100, 2860-2865.

Temple, E., Poldrack, R.A., Protopapas, A., Nagarajan, S., Salz, T., Tallal, P., Merzenich, M.M., & Gabrieli, J.D.E. (2000). Disruption of the neural response to rapid acoustic stimuli in dyslexia: Evidence from functional MRI. Proceedings of the National Academy of Sciences (PNAS), 97, 13907-13912.

Temple, E., Poldrack, R., Salidis, J., Deutsch, G., Tallal, P., Merzenich, M.M., & Gabrieli, J.D.E (2001). Disrupted neural responses to phonological and orthographic processing in dyslexic children: an fMRI study. Neuroreport, 12, 299-307.

Tervaniemi, M., Jacobsen, T., Röttger, S., Kujala, T., Widmann, A., Vainio, M., Näätänen, R., & Schröger, E. (2006). Selective tuning of cortical sound-feature processing by language experience. European Journal of Neuroscience, 23, 2538-2541.

Tervaniemi, M., Maury, S., & Näätänen, R. (1994). Neural representations of abstract stimulus features in the human brain as reflected by the mismatch negativity. Neuroreport, 5, 844-846.

Thomson, J.M., Fryer, B., Maltby, J., & Goswami, U. (2006). Auditory and motor rhythm awareness in adults with dyslexia. Journal of Research in Reading, 29, 334-348.

Toplak, M.E., Rucklidge, J.J., Hetherington, R., John, S.C.F., & Tannock, R. (2003). Time perception deficits in attention-deficit/hyperactivity disorder and comorbid reading difficulties in child and adolescent samples. Journal of Child Psychology and Psychiatry, 44, 888-903.

Toplak, M.E. & Tannock, R. (2005). Time perception: Modality and duration effects in attention-deficit/hyperactivity disorder (ADHD). Journal of Abnormal Child Psychology, 33, 639-654.

Torgesen, J.K., Wagner, R.K., Rashotte, C.A., Rose, E., Lindamood, P., Conway, T., & Garvan, C. (1999). Preventing reading failure in young children with phonological processing disabilities: Group and individual responses to instruction. Journal of Educational Psychology, 91, 579-593.

Trainor, L.J., Samuel, S.S., Desjardins, R.N., & Sonnadara, R.R. (2001). Measuring temporal resolution in infants using mismatch negativity. Neuroreport, 12, 2443-2448.

Tremblay, K., Kraus, N., Carrell, T.D., & McGee, T. (1997). Central auditory system plasticity: Generalization to novel stimuli following listening training. The Journal of the Acoustical Society of America, 102, 3762-3773.

Tzenova, J., Kaplan, B.J., Petryshen, T.L., & Field, L.L. (2004). Confirmation of a dyslexia susceptibility locus on chromosome 1p34-p36 in a set of 100 Canadian families. American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 1278, 117-124.

Uwer, R., Albrecht, R., & von Suchodoletz, W. (2002). Automatic processing of tones and speech stimuli in children with specific language impairment. Developmental Medicine and Child Neurology, 44, 527-532.

Uwer, R. & von Suchodoletz, W. (2000). Stability of mismatch negativities in children. Clinical Neurophysiology, 111, 45-52.

van Leeuwen, T., Been, P., Kuijpers, C., Zwarts, F., Maassen, B., & van der Leij, A. (2006). Mismatch response is absent n 2-month-old infants at risk for dyslexia. Neuroreport, 17, 351-355.

Vanni, S., Uusitalo, M.A., Kiesilä, P., & Hari, R. (1997). Visual motion activates V5 in dyslexics. Neuroreport, 8, 1939-1942.

Vogler, G.P., DeFries, J.C., & Decker, S.N. (1985). Family history as an indicator of risk for reading disability. Journal of Reading Disabilities, 18, 419-421.

von Plessen, K., Lundervold, A., Duta, N., Heiervang, E., Klauschen, F., Smievoll, A.I., Ersland, L., & Hughdahl, K. (2002). Less developed corpus callosum in dyslexic subjects – a structural MRI study. Neuropsychologia, 40, 1035-1044.

Wadsworth, S.J., Olson, R.K., Pennington, B.F., & DeFries, J.C. (2000). Differential genetic etiology of reading disability as a function of IQ. Journal of Learning Disabilities, 33, 192-199.

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.

Walker, K.M.M., Hall, S.E., Klein, R.M., & Phillips, D.P. (2006). Development of perceptual correlates of reading performance. Brain Research, 1124, 126-141.

Walther-Müller, P.U. (1995). Is there a deficit of early vision in dyslexia? Perception, 24, 919-936.

Watson, B.U. (1992). Auditory temporal acuity in normally achieving and learning-disabled college students. Journal of Speech and Hearing Research, 35, 148-156.

Weinberger, D.R., Luchins, D.J., Morihisa, J., & Wyatt, R.J. (1982). Asymmetrical volumes of the right and left frontal and occipital regions of the human brain. Annals of Neurology, 11, 97-100.

White, S., Frith, U., Milne, E., Rosen, S., Swettenham, J., & Ramus, F. (2006a). A double dissociation between sensorimotor impairments and reading disability: A comparison of autistic and dyslexic children. Cognitive Neuropsychology, 23, 748-761.

White, S., Milne, E., Rosen, S., Hansen, P., Swettenham, J., Frith, U., & Ramus, F. (2006b). The role of sensorimotor impairments in dyslexia: a multiple case study of dyslexic children. Developmental Science, 9, 237-255.

Willcutt, E.G. & Pennington, B.F. (2000a). Comorbidity of reading disability and attention-deficit/hyperactivity disorder: differences by gender and subtype. Journal of Learning Disabilities, 33, 179-191.

Willcutt, E.G. & Pennington, B.F. (2000b). Psychiatric comorbidity in children and adolescents with reading disability. Journal of Child Psychology and Psychiatry, 41, 1039-1048.

Willcutt, E.G., Pennington, B.F., Boada, R., Ogline, J.S., Tunick, R.A., Chhabildas, N.A., & Olson, R.K. (2001). A comparison of the cognitive deficits in reading disability and attention-deficit/hyperactivity disorder. Journal of Abnormal Psychology, 110, 157-172.

Willcutt, E.G., Pennington, B.F., Olson, R.K., Chhabildas, N., & Hulslander, J. (2005). Neuropsychological analyses of comorbidity between reading disability and attention deficit hyperactivity disorder: In search of the common deficit. Developmental Neuropsychology, 27, 35-78.

Willcutt, E.G., Pennington, B.F., Smith, S.D., Cardon, L.R., Gayán, J., Knopik, V.S., Olson, R.K., & DeFries, J.C. (2002). Quantitative trait locus for reading disability on chromosome 6p is pleiotropic for attention-deficit/hyperacitivity disorder. American Journal of medical Genetics, 114, 260-268.

Willis, W.G., & Weiler, M.D. (2005). Neural substrates of childhood attention-deficit/hyperactivity disorder: electroencephalographic and magnetic resonance imaging evidence. Developmental Neuropsychology, 27, 135-182.

Winkler, I., Kujala, T., Tiitinen, H., Sivonen, P., Alku, P., Lehtokoski, A., Czigler, I., Csépe, V., Ilmoniemi, R.J., & Näätänen, R. (1999). Brain responses reveal the learning of foreign language phonemes. Psychophysiology, 36, 638-642.

Winkler, I., Tervaniemi, M., & Näätänen, R. (1997). Two separate codes for missing-fundamental pitch in the human auditory cortex. The Journal of the Acoustical Society of America, 102,1072-1082.

Winsberg, B.G., Javitt, D.C., & Shanahan/Silipo, G. (1997). Electrophysiological indices of information processing in methylphenidate responders. Biological Psychiatry, 42, 434-445.

Winsberg, B.G., Javitt, D.C., Silipo, G.S., & Doneshka, P. (1993). Mismatch negativity in hyperactive children: effects of methylphenidate. Psychopharmacological Bulletin, 29, 229-233.

Witton, C., Stein, J.F., Stoodley, C.J., Rosner, B.S., & Talcott, J.B. (2002). Separate influences of acoustic AM and FM sensitivity on the phonological decoding skills of impaired and normal readers. Journal of Cognitive Neuroscience, 14, 866-874.

Witton, C., Talcott, J.B., Hansen, P.C., Richardson, A.J., Griffiths, T.D., Rees, A., Stein, J.F., & Green, G.G.R. (1998). Sensitivity to dynamic auditory and visual stimuli predicts nonword reading ability in both dyslexic and normal readers. Current Biology, 8, 791-797.

Woldorff, M.G., Hackley, S.A., & Hillyard, S.A. (1991). The effects of channel-selective attention on the mismatch negativity wave elicited by deviant tones. Psychophysiology, 28, 30-42.

World Health Organization (WHO). (1990). International Classification of Diseases 10th revision (ICD-10). Geneva, Switzerland: WHO.

Wright, B.A., Lombardino, L.J., King, W.M., Puranik, C.S., Leonard, C.M., & Merzenich, M.M. (1997). Deficits in auditory temporal and spectral resolution in language-impaired children. Nature, 387, 176-178.

Wright, B.A. & Zecker, S.G. (2004). Proceedings of the National Acadamy of Sciences of the United States of America (PNAS), 29, 9942-9946.

Yabe, H., Tervaniemi, M., Sinkkonen, J., Huotilainen, M., Ilmoniemi, R., & Näätänen, R. (1998). Temporal window of integration of auditory information in the human brain. Psychophysiology, 35, 615-619.

Ylinen, S., Shestakova, A., Huotilainen, M., Alku, P., & Näätänen, R. (2006). Mismatch negativity (MMN) elicited by changes in phoneme length: A cross-linguistic study. Brain Research, 1072, 175-185.

Zeffiro, T., & Eden, G. (2000). The neural basis of developmental dyslexia. Annals of dyslexia, 50, 3-30.