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Invited review

Clinical neurophysiology of visual and auditory processing in dyslexia: A review

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ABSTRACT

Neurophysiological studies on children and adults with dyslexia provide a deeper understanding of how visual and auditory processing in dyslexia might relate to reading deficits. The goal of this review is to provide an overview of research findings in the last two decades on motion related and contrast sensitivity visual evoked potentials and on auditory event related potentials to basic tone and speech sound processing in dyslexia. These results are particularly relevant for three important theories about causality in dyslexia: the magnocellular deficit hypothesis, the temporal processing deficit hypothesis and the phonological deficit hypothesis. Support for magnocellular deficits in dyslexia are primarily provided from evidence for altered visual evoked potentials to rapidly moving stimuli presented at low contrasts. Consistently ERP findings revealed altered neurophysiological processes in individuals with dyslexia to speech stimuli, but evidence for deficits processing certain general acoustic information relevant for speech perception, such as frequency changes and temporal patterns, are also apparent.

Contents

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1. Introduction

Dyslexia is a specific developmental disorder in learning to read, which is not the direct result of impairments in general intelligence, gross neurological deficits, uncorrected visual or auditory problems, emotional disturbances or inadequate schooling. (International Classification of Diseases, ICD-10, 2009; Dilling et al., 1991; DSM IV-TR American Psychiatric Association, 2000). Dyslexia accompanies the individual throughout their lifespan and interferes with academic achievement or activities of daily living that require reading skills (Shaywitz et al., 1999). It occurs in all known languages (Lindgren et al., 1985; McBride-Chang et al., 2008) and is one of the most common developmental disorders affecting around 5% of school-aged children (Shaywitz et al., 1990; Katusic et al., 2001). Socioeconomic status and family factors are known to influence the development of reading abilities, but are not causally related to dyslexia (Stevenson and Fredman, 1990; Vellutino et al., 2004).

Since the first description of dyslexic cases a familial aggregation was observed (Morgan, 1896). Family and twin studies clearly point to a genetic basis of this complex disorder and first candidate genes have been found (Smith et al., 1983; Taipale et al., 2003; for reviews see Paracchini et al., 2007; Schumacher et al., 2007). A key function of these genes is their involvement in neuronal migration and axon growth. Imaging studies have clearly demonstrated an altered cortical network in dyslexic subjects that comprises left and right superior temporal cortices, left inferior temporal-occipital cortices and both left and right inferior frontal and posterior temporo-parietal cortices (for review see Schlaggar and McCandliss, 2007).

A number of electrophysiological studies have provided evidence for basic perceptual deficits in dyslexia. Abnormal event-related potentials (ERPs; amplitude, latency, topography) for auditory processing of non-speech and speech sounds were found in dyslexic children and adults. Analogously, altered visual evoked potentials (VEPs) were reported in dyslexic subjects when non-linguistic stimuli were presented.

We conducted a PubMed search spanning two decades of research using *dyslexia* and *reading disorder* as keywords in combination with *event related potentials*, *ERPs, VEPs, motion onset, contrast sensitivity* and *mismatch negativity* and found 74 papers reporting on electrophysiological correlates of dyslexia (studies with unclear group selection criteria or reporting magnetoencephalography (MEG) data were excluded). Forty articles primarily concerned visual processing of non-linguistic material (e.g. graphical material that varied on spatial frequency, contrast and temporal frequency) and linguistic material (e.g. letters, words, lexical, syntactic and semantic aspects of word comprehension). Auditory perception of non-linguistic material (e.g. sinus tones), speech sounds (e.g. speech contrasts like /da/ vs. /ga/) and phonological processing (e.g. rhyme judgements) were reported in 34 articles. For both auditory and visual perception approximately half of the articles investigated ERP correlates elicited by non-speech and non-linguistic stimuli. The goal of this review is to summarize and integrate investigations conducted on the neurophysiological correlates of basic perceptual visual ERPs to motion and contrast sensitivity, as well as auditory ERPs to tones and speech sounds, in dyslexia over the last 20 years. Because the definition of reading disorders can be quite broad, we attempted to only include those studies which explicitly recruited their participants according to below average reading (and in some cases spelling). In exceptional cases (e.g. Kraus et al., 1996) we included studies with less strict definitions of dyslexia, as we considered them to be very important for the review. In these cases, the discrepancy has been pointed out.

Visual and auditory perception deficits in dyslexia have been reported since the beginning of dyslexia research (Hinshelwood, 1895; Morgan, 1896; Borel-Maisonny, 1951). Since then, it has been repeatedly shown that phonological processing is one of the most relevant factors for learning to read and spell and is impaired in dyslexic children, adults and in compensated adults (for reviews see Rack, 1994; Snowling, 2000; Ramus et al., 2003).

Based on observations of aphasic children in the 1970's (Tallal and Piercy, 1973a) a temporal processing theory was formulated in order to explain perceptual deficits that could account for the phonological processing deficits observed in dyslexia (Tallal, 1980a, 2004). Since then numerous studies were conducted to explore the basic auditory deficits in dyslexia by investigating the ability to discriminate non-speech stimuli (Farmer and Klein, 1995; McArthur and Bishop, 2001). The temporal processing theory was extended to the perception of non-linguistic visual stimuli (Stein, 2001). For both areas, neurophysiological studies have made major contributions to the understanding of the neurobiological correlates of dyslexia.

For this review we chose to focus on these two research lines, visual and auditory processing of non-linguistic and sub-lexical stimuli, in dyslexia. The first reason for this selection is that more than 36 empirical papers have been published on these topics. Secondly, several common remediation programs, at least in Europe, are based on the assumption of basic visual or auditory perception deficits in dyslexia. These are often time consuming interventions, for both therapists and clients. If the empirical basis for such interventions is low, the use of these interventions in therapeutic settings should be critically discussed. Thirdly, there continues to be an urgent need to improve the understanding of the aetiology of dyslexia despite more than 100 years of research. Although a phonological deficit is often found in dyslexic individuals (between 30% and 60% depending on the study), its aetiology remains, for the most part poorly understood.

We begin with a summary of the ERP studies on visual processing of non-linguistic stimuli followed by a discussion on the literature covering the basic auditory processing of non-linguistic stimuli. The auditory processing review culminates with speech (sub-lexical) perception and includes studies on early predictors. We have decided to integrate speech perception, which focuses on examining discrimination abilities between consonant–vowel (CV) stimuli and cortical auditory evoked potentials to CV stimuli, because there is accumulating evidence that speech perception is one of the best predictors of reading disability (Guttorm et al., 2005; Lyytinen et al., 2005a, b). Furthermore, the first patho-physiological pathway model, from gene function to speech perception in dyslexia, has recently been described (Roeske et al., 2009). This finding renders ERP correlates of speech promising candidates for understanding the aetiology of dyslexia.

2. Visual perception

Dyslexia was first postulated to be a disorder of the visual system (Hinshelwood, 1895; Kussmaul, 1877). Since then, numerous empirical studies have described visual deficits for movement and contrast perception in dyslexic individuals (for reviews see Laycock and Crewther, 2008; Stein, 2001). Some reports point to deficits only within sub-groups of dyslexia (Borsting et al., 1996; Heim et al. 2008; Reid et al., 2007). For example, Borsting et al. (1996) demonstrated that contrast sensitivity to low spatial frequencies was reduced only in a group of more severely impaired dyslexic individuals, who suffered from both whole word recall and auditory deficits; however, ERP studies have not yet systematically examined visual potentials in these sub-groups.

Visual sensory impairments in dyslexia have been explained by deficient functioning within the fast processing transient

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pathway of the visual system, known as the visual magnocellular pathway (Stein, 2001; Ramus, 2004; Sperling et al., 2006; Laycock and Crewther, 2008). The magnocellular pathway is characterized by large cells widely distributed across the retina and projects, via the ventral lateral geniculate nucleus, to the visual cortex and thereafter largely to the posterior parietal cortex. Magnocells specialize in motion and positional relationships, and preferentially mediate fast temporal resolution, low contrast, and low spatial frequencies (Merigan and Maunsell, 1993). Anatomically, magnocellular deficits might be attributed to cortical anomalies in the visual system (Galaburda et al., 1985, 1994), where neurons in the lateral geniculate nucleus were found to be smaller and less structured (Livingstone et al., 1991). Although promising, these findings should be regarded with some degree of caution as they result from the investigation of brains of poorly defined dyslexics and have not yet been replicated in a more accurately defined groups. Furthermore, possible developmental delays, acquired and genetic illnesses may have influenced brain anatomy in these cases. Nonetheless, these findings are important for the validity of the magnocellular theory (Stein, 2001).

Both investigations of psychophysical and VEP responses have provided some evidence for deficits in the magnocellular system in dyslexia by reporting on perceptual measurements of coherent motion (Cornelissen et al., 1998), contrast sensitivity (Borsting et al., 1996), rapid motion (Demb et al., 1998), visible persistence (Winters et al., 1989; Schulte-Körne et al., 2004a) and spatial frequency (Livingstone et al., 1991). The merit of these results coupled with the questionable impact of the anatomical data has been a matter of critical debate (Gross-Glenn et al., 1995; Skottun, 2000; Stuart et al., 2001; Amitay et al., 2002; Skoyles and Skottun, 2004; Skottun and Skoyles, 2007, 2008). For example, Skottun and Skoyles (2008) have questioned whether coherent motion can be used as an appropriate test for understanding and interpreting magnocellular pathway function. Furthermore, the authors have argued that based on similar perceptual deficits found in other patient groups (e.g. schizophrenia and autism) the magnocellular deficit cannot be causally linked to dyslexia. Despite the apparent controversery surrounding the possibility of magnocellular deficits in dyslexia, current knowledge pertaining to visual system function does suggest a number of functionally important roles for magnocellular deficits in dyslexia (Laycock and Crewther, 2008). In a recent review, Laycock and Crewther (2008) explore how magnocellular deficits might impact reading. The authors describe how the magnocellular pathway contributes to the rapid integration of visual information when reading, via spatial, temporal and attentional processes, including the control, direction and organization of saccadic eye-movements.

A second visual processing subsystem known as the parvocellular pathway is thought to be largely intact in dyslexia (however, for exceptions see Farrag et al., 2002; Skottun, 2000). The parvocellular pathway originates in small cells concentrated within the fovea, projects to the visual cortex via the dorsal lateral geniculate nucleus, and culminates in the temporal cortex. It is sensitive to medium and high spatial frequencies, has a moderate temporal resolution (Merigan and Maunsell, 1993) and is important for object discrimination based on colour, form, and texture.

2.1. Motion

Neurophysiological studies investigating the response characteristics of the magnocellular system have made significant contributions on its importance for dyslexia. Based on the high temporal resolution of the VEP, different ERP components were found to correlate to different visual processes, i.e. contrast sensitivity and motion perception (Kuba et al., 2007).

Niedeggen and Wist (1999) differentiated two ERP components of motion onset in a coherent motion paradigm. At occipital sites visual motion onset evoked a positive component between the latency of 100 and 130 ms (P100), and a positivity at about 230 ms (P200) was recorded at central-parietal leads. Hirai et al. (2003) identified a negativity evoked by coherent motion onset occurring between 200 and 280 ms bilaterally over occipito-temporal electrodes. This negativity was nearly independent of luminance contrast and its amplitude varied with stimulus velocity. These neurophysiological correlates of motion perception were also recorded in 8 month old infants suggesting that even at a very young age neurons of the visual systems are sensitive to motion perception (Hirai and Hiraki, 2005).

These VEPs correspond to different functional properties of motion processing neurons and cortical areas which are essential for the analysis and perception of motion. Motion onset is primarily related to local motion detectors which are located in the primary cortex of visual area V1. Coherent motion perception, i.e. the global integration of local motion information, is mainly related to cortical activity outside the primary visual cortex in the extrastriate visual area V5, and the cortical regions bordering the temporal, parietal, and occipital lobes, respectively (Rodman and Albright, 1989; Nowak and Bullier, 1997; Nichols and Newsome, 2002). V5, also known as MT (middle temporal), is involved in perceiving motion, integrating local motion signals into global percepts and guiding some eye movements (Born and Bradley, 2005).

Motion-related VEPs were investigated in dyslexia in accordance with the hypothesis that the magnocellular visual pathway is impaired in these individuals (Table 1). The VEP studies investigating motion onset in dyslexia consistently found prolonged latencies and smaller amplitudes of the typically evoked P100. This finding suggests a reduced velocity in visual processing and contributes to the hypothesis of a selective weakness of the visual transient system.

Interestingly, in the Kubová studies (Kubová et al., 1996; Kuba et al., 2001) a developmental decrease in the N160 latency in dyslexic subjects was reported, and suggested a maturational deficit of the magnocellular system. In 10-year old children with dyslexia N160 latencies were recorded at 236 ms, whereas at age 14 the N160 latency had reduced to 162 ms. In comparison, latencies recorded from children without reading problems at about 10 years of age did not differ from those recorded at 14 years of age (167 ms at 10 years, 158 ms at 14 years). Because 40–60% of subjects with dyslexia were found to have a longer N160 latency for motion perception (Kuba et al., 2001), the motion onset VEP was recommended as part of the diagnostic procedure for dyslexia (Kuba et al., 2007). However, altered motion onset VEPs were also found in other disorders such as optic neuritis (prolongation of motion onset VEPs in 28% of the patients) and multiple sclerosis (17% of the patients) (Kuba et al., 2007).

Thus, the studies reporting on motion VEPs suggest deficits in dyslexia. Prolonged P100 latencies and attenuated amplitudes are altered to rapid movements, but not to static visual perception. The longer N160 latencies in younger children with dyslexia suggest that motion detection might be developmentally delayed, and at adolescence the response is comparable to age-matched controls.

2.2. Contrast sensitivity

A substantial amount of research examining the magnocellular deficit hypothesis comes from psychophysical and neurophysiological studies on contrast sensitivity, a measure of the ability to detect contrasts (both in the spatial and in the temporal realm) at differing thresholds (Lovegrove et al., 1980, 1982; Cornelissen et al., 1998). Based on the assumption that dyslexic subjects are characterized by altered magnocellular function, the expectation

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Table 1
Motion: VEP study summary.

Study	Sample	Motion onset stimuli	Motion VEP/ electrodes	VEP group differences
Kubová et al. (1996)	CG: N = 16 DG: N = 20 Mean age: 10	Motion onset: isolated checks; velocity 10 deg/s; random movement for 200 ms Control condition: stationary phase	N160 peak & latency at 3 occ. electrodes: left, right & Oz	Motion onset: * N160 longer latency in 70% in DG Control condition: n.s.
Kuba et al. (2001)	3 age groups: CG: N = 7 Mean age: 13 DG_1: N = 10 Mean age: 14 DG_2: N = 25 Mean age: 10	Motion onset: Low contrast (10%); 200 ms motion; ISI 1 s 1. isolated checks, linear motion; velocity 10 deg/s; 5 colour modifications (equivalent wavelengths) 2. grey concentric frames; expansionControl condition: Transient pattern reversal, high contrast (96%); black & white checkerboard	N160 peak & latency at 3 electrodes: occ. left, right, & Oz	Motion onset: * N160 longer latencies in DG_2 for condition 1 (20%) & 2 (48%); independent from colour; DG_1 latency shortens from 10–14 years; in both CG & DG_1 amplitude is lowest at 14 years Control condition: n.s.
Schulte-Körne et al. (2004b)	CG: N = 12 DG: N = 10 Mean age: 12	White dots in rectangular patch on black background; 5 deg/s Coherent motion: coherent movement (10%, 20% or 40%); fraction of dots Control condition: random movement; 1000 ms	1. P1 & P2 at O1 & O2 2. P500 at O1, O2, T5, T6, TP7, TP8, P3, P4, CP3 & CP4	Coherent motion: * P500 reduced area in DG Control condition: n.s.
Schulte-Körne et al. (2004c)	CG: N = 8 DG: N = 14 Mean age: 12	Motion onset: Sine wave vertical gratings, 2 cpd visual angle, contrast = 0.8, luminance 12 cpd/m^2; 3 velocities: 2, 8, 16 deg/s; each 600 ms Control condition: stationary phase	P1 at O1, O2, Oz; P2 at O1, O2, Oz, P3, Pz, P4, C3, Cz, C4	Motion onset: * P1 and P2 lower amplitude & longer latency in DG; P2 interaction group & velocity: difference between groups increased with greater velocities Control condition: n.s.

CG = control group; DG = dyslexic group; cpd = cycles per degree; GFP = global field power; deg = degrees; ISI = interstimulus interval; * = significant group differences found; n.s. = not significant; n.a. = not applicable; occ. = occipital.

is that differences in the early VEP components in contrast sensitivity paradigms should be observed under low contrasts and fast presentation rates. Here, non-linguistic low spatial frequency stimuli in the form of sinusoidal gratings and checkerboard pattern-reversals were presented at various frequencies and contrasts.

One of the earliest electrophysiological studies on contrast sensitivity used simple checker-board like pattern-reversal stimuli. Livingstone et al. (1991) reported diminished transient VEP responses (negativity between 20 and 50 msec) in adults with dyslexia for low contrast stimuli presented at high reversal rates (Table 2) and these results were at least partially confirmed in children with dyslexia (May et al., 1991; Lehmkuhle et al., 1993; Romani et al., 2001).

Delayed steady state responses based on longer latencies and reduced amplitudes of an early positivity (P100) and an early negativity (N100) at occipital cortical areas suggested an altered magnocellular function in dyslexia (Lehmkuhle et al., 1993; Livingstone et al., 1991). However, several studies (Victor et al., 1993; Johannes et al., 1996; May et al., 1991; Romani et al., 2001; Farrag et al., 2002; Schulte-Körne et al., 1999a; Brecelj et al., 1997-1998) found inconsistent results, reporting shorter VEP latencies in contrast to the control group. The latency reduction reported by Romani et al. (2001) could be explained by the amplitude reduction observed at this latency and was interpreted as a "paradoxical" latency reduction.

Victor et al. (1993) and Johannes et al. (1996) used a range of contrasts and temporal frequencies that were highly comparable to the stimulus characteristics from the Livingston et al. study and measured transient and steady state VEP responses in children (Victor et al., 1993) and adults (Johannes et al., 1996). Both studies failed to provide supporting evidence for diminished VEP responses. Variability in responses between the dyslexic and control groups could not account for failure to replicate the findings, although subjects with attention deficit-hyperactivity disorder (ADHD) did have significantly more variability (Victor et al., 1993). Thus, one explanation for the lack of replication in these two studies might be that Livingstone et al.'s (1991) findings resulted from a comorbid disorder of dyslexia and ADHD in at least a sub-group of participants. This possibility highlights the importance of careful participant selection criteria. Here, a reduction in contrast sensitivity might be partially be explained by reduced attention focusing in children, which would presumably render the perception of low contrast stimuli more difficult.

In comparison, Brecelj et al. (1997-1998) found group differences (prolongation of the P100 latency) for high contrast stimuli (100%) in children with dyslexia compared to controls, whereas Farrag et al. (2002) reported shortened P100 latencies. Unfortunately, such high contrasts are not suited to investigate contrast sensitivity as processed by the magnocellular system. Thus, the findings of these studies can be interpreted as evidence for a parvocellular deficit in dyslexic subjects.

Finally, Schulte-Korne et al. (1991a) examined VEPs in German adults with spelling disorder using sine wave vertical gratings of varying contrasts (0.2, 0.4, 0.6, 0.8) and found no group differences. However, laterality differences were observed. VEP responses recorded from controls were greater over right occipital leads, whereas the individuals with spelling disorder showed a bilateral distribution. Other studies have used reading as their main criteria for study inclusion and therefore the inclusion of subjects based on spelling disorder may be one reason for the non-replication of previous results. This might have led to identification of subjects that are different from the typical reading disabled subjects in the other studies.

3. Summary

We have outlined the ERP research exploring visual processing in dyslexia. In the foreground of the research were studies exploring the functional role of the magnocellular system.

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Table 2
Contrast: VEP study summary.

Study	Sample	Stimuli	Contrast sensitivity VEP/ electrodes	Relts
Livingston et al. (1991)	CG: *N* = 7 Mean age: 25.8 DG: *N* = 5 Mean age: 27.4	Checkerboard, vertical .0.16 cpd; horizontal 0.12 cpd Transient pattern reversal VEP: Contrast 0.2–0.02 at reversal rate 0.5 Hz Steady state VEP: various contrast reversals at varying frequencies	Early negativity (C1), P1 at CZ & Oz	Transient VEP: *DG missing or late early negative component at 50 ms at .02 contrast *P1 delayed in DG for low contrast condition at .02 contrast n.s. 0.2 contrast Steady state VEP: *amplitude reduction in DG for low contrast conditions (.01, .02) at 15 Hz n.s. high contrasts
May et al. (1991)	CG/DG: *N* = 10 Mean age: 12.4	Sinusoidal gratings: pattern onset-offset-paradigm with spatial frequencies (0.5, 1, 2, 4, and 8 cpd) at 1.6 Hz	N1, P1, N2, P2 at Oz	*Reduced N1 and P1 amplitude of the offset response at low spatial frequency (0.5 cpd) in DG *Latency reduction of the N2 and P2 in DG at low spatial frequency (1 cpd)
Lehmkuhle et al. (1993)	CG: *N* = 13 DG: *N* = 8 Mean age: 10.6	Sinusoidal gratings Pattern-onset VEP; spatial frequencies 0.5 & 4.5 cpd at 0.1 contrast Two background conditions: Steady State & Uniform Field-Flicker background	One occipital electrode	Steady state: *P1/N2 lower amplitude and delayed latency at 0.5 cpd Uniform Field Flicker: n.s.
Victor et al., 1993	CG: *N* = 11 Mean age: 12 DG: *N* = 10 Mean age: 13.5	Checkerboard, stimulus contrast between 2% and 20%, reversal rate 4, 8 and 16 Hz	P1 at Cz and Oz	n.s.
Johannes et al. (1996)	CG/DG: N = 6 Mean age: 21	Checkerboard, 7 × 5 checks, sides = 4° of visual angle Transient pattern reversal VEP: Contrasts: 0.01, 0.02, 0.15, 0.2, 0.5 at reversal rate of 1 Hz Steady state VEP: Contrasts: 0.01, 0.02, 0.15, 0.2, 0.5 at reversal rates of 10, 20, 30 Hz	C1, P1 and N1 at 13 frontal, central, temporal, parietal and occipital electrodes	n.s.
Brecelj et al. (1998)	CG/DG: *N* = 12 Mean age: 12	Checkerbaords (24′, 49′ &180′) with 3 varying contrasts (5%, 42%, 100%); stimulation rate 2 Hz	P50, N95, P1 at 3 occipital electrodes	*P1 DG longer latencies for 24′ at 100% contrast n.s. P50, N95
Schulte-Körne et al. (1999a)	CG: *N* = 19 Mean age: 22.3 DG: *N* = 15 Mean age: 25.9	Sinusoidal gratings, spatial frequencies: 2, 11.33 cpd; contrasts: 0.2, 0.4, 0.6, 0.8	P1 and P2 at occipital electrodes	n.s. according to differences in contrasts and spatial frequencies But *laterality group effect. CG was right lateralized for conditions. This lateralization difference increased with low frequencies. DG did not show lateralization effects.
Romani et al. (2001)	CD/DG: *N* = 9 Age: 10–17	Checkerboards, 0.5 and 2 cpd Transient pattern reversal VEP: 1.05 Hz Steady state VEP: 4 Hz	Transient VEP: N70, P1 at Oz Steady State VEP: Fast Fourier Transform of the amplitudes and phases of the harmonics at 8, 16 and 24 Hz	Transient VEP: *N70 lower amplitude and shorter latency in DG for low spatial frequency (0.5 cpd) n.s. P1 Steady state VEP: *lower amplitudes in DG
Farrag et al. (2002)	CG: N = 41 DG: *N* = 52 Mean age: children, age not given	Checkerboards 1: low (50%) & high (100%) contrast at 3 Hz; size 16 2: high contrast (100%) at 1 & 8 Hz; size 16 3: high contrast (100%) at 3 Hz; spatial frequencies low (8 Hz, large size) & high (64 Hz, small size)	One electrode at Oz; P1	1: high contrasts: *shorter latency in DG; low contrasts: n.s. between groups, but in DG N1-P1 amplitude reduced when comparing low contrast to high contrast. Finding not observed in CG 2. both conditions n.s. 3. 8 Hz condtion: *shorter P1 latency in DG; 64 Hz condition: *tendency for P1 prolongation in DG; *within DG latencies between conditions significantly different. In CG no differences

CG = control group; DG = dyslexic group; cpd = cycles per degree; GFP = global field power; deg = degrees; ISI = interstimulus interval; * = significant group differences found; n.s. = not significant.

The VEPs recorded over occipital cortical brain areas at a latency of 100–200 ms were prolonged mainly when rapidly moving stimuli were presented at low contrasts. This finding strongly suggests an altered magnocellular system; although, this interpretation has been controversially discussed (Skottun and Skoyles, 2006; Schulte-Körne et al., 2004d), the VEP findings on motion have consistently reported delayed and altered processing of neurons activated by rapidly moving non-linguistic stimuli.

However, for contrast sensitivity the results were less consistent. This is unexpected if the magnocellular deficit theory is valid because low contrast stimuli presented at high temporal frequencies should specifically activate neurons of the magnocellular system (Skottun, 2000).

Some aspects that might explain the contradictory findings are subject age, differences in experimental design across studies and comorbid disorders like ADHD.

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Furthermore, a substantial variability across experimental methods exists, rendering between study comparisons difficult, as can be seen in Tables 1 and 2. For example, contrast levels range from very low (0.01) to very high (1) as do spatial frequencies (low 0.15 to high 8 cpd).

The age range between 10 and 46 years and the small sample sizes (e.g. below 10 dyslexic subjects in Livingston et al., 1991; Johannes et al., 1996; Romani et al., 2001) further give evidence for methodological drawbacks of the VEP studies in dyslexia.

Another critical aspect of the reviewed studies is the possibility of comorbid ADHD amongst dyslexic subjects that was not reported in the majority of studies. A possible objection against the influence of ADHD on the ERP findings might be the use of a parvocellular control condition in most studies reported. The influence of comorbidity with ADHD would also be expected to impact the parvocellular pathway, which was not the case. None-the-less, attention remains a relevant aspect for understanding and interpreting these ERP findings. Both visual attention and visuospatial attention are associated with the magnocellular system and are likely related to reading problems in dyslexia (Boden and Giaschi, 2007). The magnocellular system culminates in the posterior parietal cortex, which is well known to be involved in a range of attentional operations (Constantinidis, 2006; Nachev and Husain, 2006). In dyslexic subjects impaired visual attention has been repeatedly found (Facoetti et al., 2000a,b; Heiervang and Hugdahl, 2003; Kinsey et al., 2004) Also, ERP research reports evidence for a spatial selective attention deficit in dyslexia (Wijers et al., 2005) and for top-down impaired attention modulating processes in a coherent motion perception task (Schulte-Körne et al., 2004b). These processes were suggested to mediate processing in the magnocellular stream (Vidyasagar, 2005).

How a visual attention deficit contributes to impaired word reading has been intensively discussed by Boden and Giaschi (2007) and empirically evaluated in behavioural studies (e.g. Solan et al., 2007). In summary, attention is required to filter out letters that do not belong to the word (spatial attention) and to direct attention to the locus of fixation. From behavioural studies there is evidence that strengthens the role of visual attention in word reading in dyslexic subjects (for review see Boden and Giaschi, 2007). For ERP research in particular, more adequate study designs are necessary to investigate the influence of visual attention on basic visual perception in dyslexia.

4. General acoustic processing deficits

The analysis of general acoustic information, and speech signals in particular, requires successful interpretation of both temporal and spectral sound features. Tallal first suggested that poor language skills in dyslexia might arise from a general deficit in processing rapidly occurring temporal information (Tallal, 1975, 1980b; Tallal and Piercy, 1973b). She and her coworkers could show that individuals with dyslexia performed worse when discriminating between both rapid speech and non-speech stimuli. When the stimuli were presented at slower rates, perception improved. Farmer and Klein (1995) extended Tallal's claim to all sensory modalities, for example suggesting temporal processing disorders in visual perception as well as in auditory perception. ERP research investigating general auditory processing has examined not only temporal, but also spectral aspects of acoustic processing in dyslexia. Most notably, investigations have been conducted in these areas with respect to discrimination abilities for pitch discrimination (e.g. Baldeweg et al., 1999; Kujala et al., 2003), stimulus duration (e.g. Baldeweg et al., 1999), frequency modulation (e.g. Stoodley et al., 2006), gap detection (e.g. Kujala et al., 2006; Moisescu-Yiflach and Pratt, 2005), and temporal order judgements (Kujala et al., 2000; Schulte-Körne et al., 1999b). The majority of investigations examine the mismatch negativity component (MMN; Näätänen et al., 1978; Bishop, 2007; Näätänan, 2007; Garrido et al., 2009).

4.1. Stimuli durations

The relevance of duration features for speech perception varies from language to language. In English for example, vowel duration can indicate a number of linguistic cues such as distinctions between long and short vowels, vowel stress, and information regarding within syllable voiced or voiceless consonants (Klatt, 1976). In some languages, such as Swedish, Finnish and Japanese vowel duration differences carry semantic relevance (Lidestam, 2009). German is for example, a language less reliant on vowel durations for detecting word meaning, and it could be shown that German speakers are less sensitive to duration changes (Kirmse et al., 2008). Relevant durations for speech stimuli are highly variable, for example the difference between /ba/-/da/ CV stimuli is characterized by a 40 ms duration transition (Mody et al., 1997), whereas a longer duration of 178 ms distinguishes the vowel/æ/ in the English word *back* vs. the /æ/ in *bag* (Ko, 2009).

In order to examine duration processing devoid of linguistic information, Baldeweg et al. (1999) used both passive (e.g. participants should ignore all stimuli) and active (e.g. participants should react to deviant stimuli) oddball paradigms to compare discrimination between 1000 Hz tones of differing durations in 10 adults diagnosed with dyslexia against 10 age-matched controls. The dyslexic adults were characterized by poor reading skills (in comparison to controls), poor working memory and poor phonological skills, and dyslexia was defined by a large discrepancy between their general abilities and written language abilities. Four deviant stimuli of varying durations (160, 120, 80, 40 ms) were presented together with a standard duration stimulus of 200 ms. MMN analysis revealed normal duration processing in dyslexia for both MMN peak latency and amplitude irrespective of attention. Similarly, Kujala et al. (2006) reported no group differences in adults with dyslexia for standard durations of 50 and 100 ms to duration deviants of 33 and 65 ms, respectively.

The above studies describe results pertaining to adults. Huttunen et al. (2007) examined children (8.8–14.2 years) with and without a reading disorder in a continuous sound paradigm where tones of 600 and 800 Hz (both 100 ms in duration) continually alternated (i.e. no gap). Deviant stimuli were decreased durations in length (30 or 50 ms) for the 600 Hz stimulus (see Fig. 1). The authors postulated that continuous sound would be a more sensitive measure as it avoids possible MMN analysis confounds that may arise due to the N1 elicited by stimulus onset (Pihko et al., 1995). MMN was elicited by both groups to both deviant durations, and no group differences were reported. Slight lateralization differences were found suggesting more activation over the left hemisphere in the reading disabled group. It is surprising

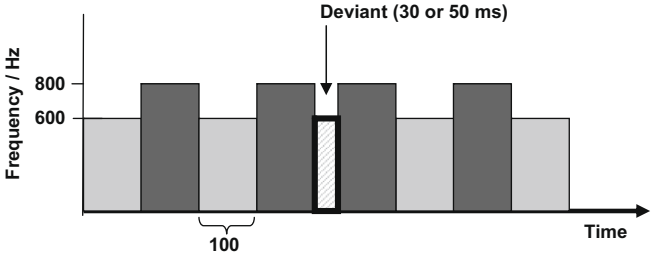

Fig. 1. Depiction of the continuous stimulus train used by Huttunen et al. (2007). Deviant stimuli were shortened durations of the 600 Hz stimulus.

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that neither amplitude nor latency deficits were reported in the reading disabled group, as duration plays an important role in the speech deficits in Finnish speaking populations (e.g. Lyytinen et al., 1995). A potential drawback to the continuous sound paradigm is that it restricts analysis to a very short window (300 ms), potentially eliminating relevant late components.

In conclusion, the ERP data show that dyslexic individuals do not suffer from a deficit in processing stimuli of differing durations. The durations used in the above studies are relevant for speech perception, ranging from 30 to 200 ms. Furthermore, even in populations where duration information is important for speech perception, no deficits were reported.

4.2. Frequency discrimination

Paramount to acoustic and speech signals are the frequencies that comprise them. In speech, these frequencies are referred to as formants. Formants are meaningful components of sound that provide distinguishing information for vowels and consonants. Vowels are usually made up of four to six formants, where the first two formants generally provide enough information to render the vowel distinct. Consonants change the vowel formant structure in varying ways. For example, bilabial sounds like 'b' in ball and 'p' in map cause the formants to lower, whereas plosives modify the position of formants of surrounding vowels (Ladefoged, 2001). It follows that if dyslexia is characterized by deficits perceiving and extracting frequency information, then these deficits would most likely impact their ability to interpret rapid frequency changes important for speech perception.

Investigations of frequency (e.g. pitch: the psychological correlate of frequency) discrimination suggest a deficit in dyslexia only between stimuli that differ with less than 100 Hz, in a graded fashion. An overview of the studies described below is summarized in Table 3.

Baldeweg et al. (1999) reported delayed and reduced MMN responses in a graded fashion in dyslexic adults for tone frequency deviants of 1015, 1030 and 1060 Hz, but not for 1090 Hz to a standard stimulus of 1000 Hz. Significant correlations between MMN latencies and errors in both word ($r = .52$, $p < .05$) and non-word reading ($r = .71$, $p < .01$) were found. Further, the N1 latencies to deviants were normal in the dyslexic group, indicating a well-functioning transient detector system (e.g. normal neural encoding of stimulus onset and offset). The attenuated MMN response on the other hand suggests abnormal decoding of the physical characteristics of the stimuli into sensory memory (Näätänen, 1992). In comparison, Kujala et al. (2006) using a 5-deviant paradigm also found an attenuated MMN for pitch contrasts in dyslexic adults. Here, however, discrimination deficits in their dyslexic group were also found for the 90 Hz pitch difference. The 5-deviant experimental paradigm is substantially different than traditional oddball paradigms. To illustrate, deviant stimuli for duration, location, intensity and gap were presented together with deviant pitch stimuli. Standard stimuli occurred only 50% of the time, thus not building a comparable memory trace to other studies, which normally use about 85% standard stimuli vs. 15% deviant stimuli. It is therefore unclear if the attenuated MMN to deviant pitch stimuli can only be attributed to pitch discrimination, or if multiple experimental factors might confound and thereby contribute to the attenuation, for example, due to probable constraints on short-term memory capacity in the dyslexic group. Finally, studies using large pitch differences (e.g. 200 Hz and greater) between tones did not report any abnormalities in adults with dyslexia (Schulte-Körne et al., 2001; Kujala et al., 2003), with the exception of Sebastian and Yasin (2008), suggesting a deficit for shorter spectral changes only.

A similar pattern of results can be observed in studies with children and adolescents with dyslexia. Maurer et al. (2003) found an attenuated MMN in kindergarten children who were at risk for dyslexia to pitch differences of 30 and 60 Hz. Children were considered at risk based on their genetic predisposition, therefore at least one first grade relative had been previously diagnosed with dyslexia. Meng et al. (2005) did not report any differences between children 8 and 13 years old with and without dyslexia to relatively large pitch differences (150 Hz). However, both Shankarnarayan and Maruthy (2007) and Schulte-Körne et al. (1998) reported no differences in MMN response between smaller pitch differences of 100 and 50 Hz in pre-adolescents, respectively. One reason for the lack of MMN group differences in Schulte-Körne's study might be attributed to the clinical group who were recruited based on their spelling disability; whereas other groups were recruited based on reading deficits (although the participants in Schulte-Körne's study (1998) had poorer word reading skills than controls).

In accordance with this finding, Lachmann et al. (2005) postulated that a drawback of previous studies was the neglect of analyzing differences in potential sub-groups. To this end, the authors defined two groups of children with dyslexia. The first consisted of children with deficits in reading frequent words only; whereas the second group had deficits in reading either non-words only or deficits reading both non-words and frequent words. An attenuated MMN in the first sub-group characterized by isolated frequent word reading deficits was found for tone differences of

Table 3
An overview of findings regarding frequency (pitch) discrimination as measured by MMN in dyslexia.

Article	Age controls	Age dyslexic	Frequency standard	Frequency deviant	SD in Hz	MMN group differences	ISI (ms)	Stimuli duration (ms)
Schulte-Körne et al. (1998)	12.5	12.6	1000	1050	50	n.s.	590	90
Baldeweg et al. (1999)	Adults	Adults	1000	1015	15	*	500	50
				1030	30	*		
				1060	60	*		
				1090	90	n.s.		
Schulte-Körne et al., 2001	Adults	Adults	2200	2640	440	n.s.	500	90
Kujala et al. (2003)	Adults	Adults	500	750	250	n.s.	560	40
Maurer et al. (2003)	6.6	6.5	1000	1030	30	*	283	100
				1060	60	*		
Lachmann et al. (2005)	9.3	9.3	700	770	70	[*]	515	385
Bitz et al. (2007)	6.9	6.9	500	750	250	n.s.	600	250
Shankarnarayan and Maruthy (2007)	7–12	7–12	1000	1100	100	*	SOA 526	250
Sebastian and Yasin (2008)	Adults	Adults	1000	1200	200	*[a]	638	362

Age indicates average age, or a given range. S = standard; D = deviant; SD = the absolute difference between standards and deviants; dur = duration; ISI = interstimulus interval; SOA = stimulus onset asynchrony; * = significant differences found between controls and dyslexics; [*] = significant differences found only for a sub-group characterized by poor frequent word reading; n.s. = non-significant differences.

[a] Dichotic listening paradigm.

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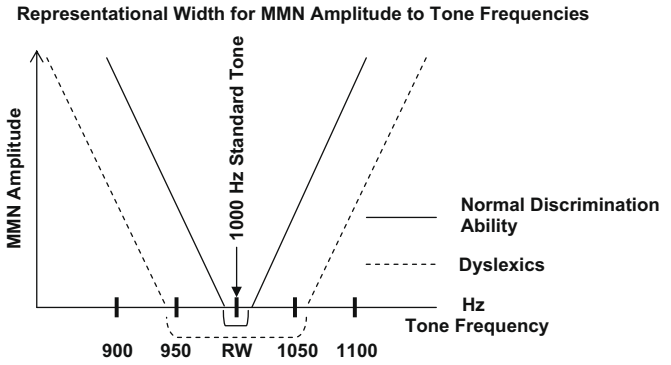

Fig. 2. Schematic representation (adapted from Näätänen and Alho, 1997) of the concept of representational width (RW) for tone frequencies. RW shows the range of sound around the standard stimulus that will not be detected as a deviant stimulus and will not elicit an MMN. To illustrate, the RW might be widened in dyslexia (dashed line), resulting in poorer discrimination of tones near the standard, but normal discrimination of tones further away from the standard. According to this figure, a standard stimulus of 1000 Hz would elicit MMN in dyslexics with deviants of 900 and 1100 Hz, but not for deviants of 950 or 1050 Hz. For non-dyslexics MMN would be elicited in both cases.

70 Hz, whereas the group with non-word reading deficits or a combination of non-word and frequent word reading deficits had a comparable MMN to control children. It is unclear if this group might show diminished MMN were it homogenous for non-word and frequent word reading disabilities. Furthermore, it is unclear why frequent word reading problems lead to pitch discrimination deficits in tones and non-word reading problems do not.

In general, a number of reports point to the existence of sub-groups in dyslexia (e.g. Manis et al., 1996; Lovett et al., 2000; Wolf and Bowers, 1999, 2000) where distinct neurological deficits might account for dyslexic sub-groups. In the case of the Lachmann et al. study, frequent word reading problems might be attributed to a visual, or whole-word dyslexia sometimes referred to as dyseidetic dyslexia. The group with combined deficits might be characterized by auditory deficits, also described as dysphonetic dyslexia (Boder, 1970, 1973). Therefore, understanding how these groups differ at the behavioural and neurophysiological levels is relevant to understanding the aetiology and the heterogeneity of dyslexia.

As reviewed here, a deficit in perceiving differences in pitch between two sounds seems to characterize dyslexia. This deficit might reflect a widened *representational width* (RW) in sound perception. RW is a concept proposed by Näätänen and Alho (1997) to describe an individual's discrimination accuracy, which is dependent on their particular ability to perceive differences in sound (Fig. 2). The narrower the width is, the better the discrimination ability. The data reported here might reflect a widening of this construct in dyslexia, thus allowing them to accurately discriminate in general, but to a less precise extent.

4.3. Tone pattern manipulations

Tone patterns reflect experiments with multiple sinus tones presented in a rhythmic pattern with a combination of durations and frequencies between tones. Deviations in the pattern therefore reflect a combination of stimulus duration and stimulus frequency. These more complicated acoustic signals provide a more valid analogy to speech than the presentation of tones in isolation, reflecting the necessity to perceive rapid sequences of incoming information such as formant transitions and voice onset timing.

Kujala et al. (2000) used patterns composed of four or two 50 Hz tones (30 ms duration) and varied the duration of intervals between the onset of tones within the patterns in order to examine discrimination abilities among adults with dyslexia. In the four tone pattern the intervals between tones in the standard stimulus were 200, 150 and 50 ms (see Fig. 3a). In the deviant condition the 2nd (150 ms) and the 3rd (50 ms) interval durations were switched. In essence, this resulted in two deviant conditions: (1) the early onset of the 3rd tone; and (2) the absence of the 3rd tone in its expected position (see Fig. 2a).

Controls elicited two MMN responses corresponding to each deviant condition; however the individuals with dyslexia showed only the second MMN response to the absence of the 3rd tone in its expected position. In controls this second response was lateralized to the right hemisphere, whereas the group with dyslexia showed bilateral activity. In a control condition, MMN was similar between the two groups to a single interval deviant (Fig. 2b).

The presence of deficient MMN to an additional deviant condition in adults listening to tone patterns was further substantiated by Schulte-Körne et al. (1999) and Kujala et al. (2003). Increased backward masking effects were suggested as a potential contributor to the discrimination deficit. Finally, in a study with Chinese children (8–13 years old) Meng et al. (2005) were able to demonstrate that children with dyslexia also perceive tone patterns irregularly. The children exhibited attenuated MMN in a large time window from 150 to 500 ms (determined by averaging 20 ms MMN time windows).

These results suggest that dyslexia is characterized by an inability to process short rhythmic patterns, and that this deficit is related to the physical properties of the stimuli. Accurate perception of rhythmic patterns requires the ability to integrate these incoming sounds temporally. In healthy subjects, the integration of incoming auditory features into neural representations in sensory memory is thought to occur within a sliding temporal window of about 150–200 ms referred to as the temporal window of integration (TWI; Näätänen, 1990). The length of this window has been established in a number of MMN studies (Schröger, 1997; Tervaniemi et al., 1994; Wang et al., 2005; Winkler and Näätänen, 1992, 1994; Yabe et al., 1997, 1998. Within 150–200 ms incoming acoustic information is integrated into a single auditory percept. Information occurring in the subsequent

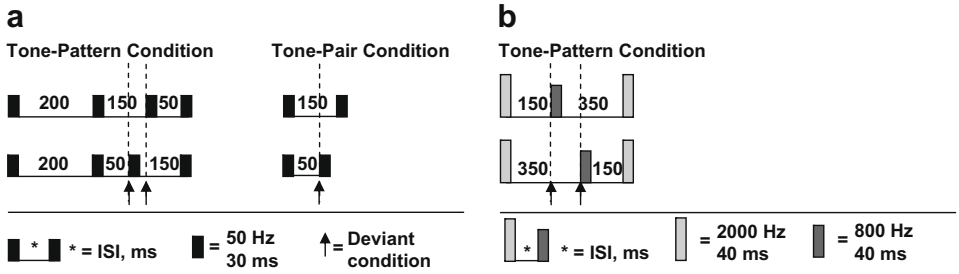

Fig. 3. (a) Depiction of stimuli used by Kujala et al. (2000) for a four tone pattern condition and a tone pair condition with adult subjects. (b) Standard and deviant patterns used with children. Both pattern deviations led to irregular MMN in dyslexics. Arrows indicate the onset of the deviant patterns. In Tone-Pattern conditions both the onset of a tone in an unexpected position and the absence of thetone in its expected position are deviations from the standard pattern.

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150–200 ms is thought to be integrated into a second auditory percept, thus the TWI continuously slides forward in time. In support of this, Winkler et al. (1998) could show that only one MMN was elicited if two deviations from a standard stimulus occurred within 150 ms, in other words, the two events were treated as one deviant event. If, on the other hand, the same two deviant events occurred 250 ms apart two MMNs were elicited. Thus, it is plausible that the absence of a MMN observed in the subjects with dyslexia in the studies described above, might in fact reflect a wider TWI in dyslexia. If this were the case, the deviations occurring in the tone patterns might have been integrated into a single temporal unit instead of separate units as in the healthy controls, thus signifying abnormal temporal integration into auditory short-term sensory memory.

4.4. Frequency modulation

In order to investigate whether auditory perception is influenced by cognition in individuals with dyslexia, Stoodley et al. (2006) sampled high-functioning university students who had been diagnosed with dyslexia in their childhood, and compared these subjects to non-impaired readers with comparable cognitive capabilities. Despite their high achievement, reading skills continued to differentiate the individuals with dyslexia from their peers. Assuming that university students with dyslexia must have developed strategies to compensate for their difficulties in reading in order to achieve success in academia, the study was designed to tap auditory deficits that could not be influenced by top-down cognitive strategies.

Stoodley et al. (2006) employed a pure standard tone (1000 Hz) and deviant tones (1000 Hz frequency-modulated with 5, 20 or 240 Hz) in 3 blocks (17% deviants) to test auditory perception in a passive oddball paradigm. MMN was attenuated in the 20 Hz condition in both early (150–300 ms) and late (300–500 ms) windows in the group of students with dyslexia. The magnitude of the MMN correlated to measures of literacy: smaller MMN was related to a larger discrepancy between cognitive and literacy abilities, suggesting a deficit in frequency modulation related to literacy. The reason why a correlation with the 20 Hz frequency modulation was found, but not for frequency modulation of 5 Hz and 50 Hz might be explained by the findings that the 20 Hz time frame is important for distinguishing stop consonants (see Clark and Yallop, 1995). Interestingly, in a separate test session, examining psychophysical thresholds the group with dyslexia performed as well as controls in actively detecting the frequency modulated changes, possibly demonstrating top-down strategies for enhancing performance. Therefore, MMN seems to be a more sensitive indicator of perceptual deficits in dyslexia.

4.5. Gap detection

Gap detection is a temporal processing task which measures the minimum ISI required to perceive an interruption (gap) in a constant train of stimuli (Farmer and Klein, 1995). Moisescu-Yiflach and Pratt (2005) investigated the ERP correlates to standard stimuli (85%; duration 280 ms) of white noise in comparison to deviant stimuli (15%; duration 280 ms) of white noise with gaps of 20 ms in both active and passive paradigms. No group differences were found at the behavioural level. However, both N1 and P3 latencies were significantly prolonged in the dyslexic group, whereas N1 magnitude was greater in comparison to controls. N1 effects were observed for both the active and the passive paradigms, whereas P3 was present in the active paradigm reflecting the allocation of attentional resources (e.g. Picton, 1988). The N1 results further point to deficits in sensory processing, whereas P3 has been associated with a variety of deficits, including attention deficits. The increased magnitude of the N1 in dyslexics has been reported in other studies (Georgiewa et al., 2002; Helenius et al., 2002) and might be related to general allocation of pre-attentional resources. However, in high-functioning dyslexic participants it might also reflect compensatory mechanisms serving to increase arousal and readiness; in turn the enhanced N1 in the dyslexic group might reflect an early investment in stimulus feature analysis in order to counteract perceptual impairments. In favour of this assumption is the lack of group differences found for task performance. Thus, this study further strengthens the view that (at least) adults with dyslexia are characterized by auditory processing deficits when processing non-speech stimuli, and highlights a further dimension that these deficits can manifest. The ERP data show that this perceptual impairment occurs within 100 ms from stimulus onset.

5. Summary

Deficits in general auditory processing in dyslexia are prevalent for stimuli of increasing complexity and/or similarity. Detecting differences of frequencies between simple sinus tones reveal MMN irregularities only when tones differed by about 100 Hz or less. This finding might be explained by a widened RW in dyslexia and is relevant for speech perception. A deficit perceiving stimulus duration differences alone cannot explain speech perception deficits. Interestingly, more complex patterns of tones with various durations and frequencies consistently revealed diminished MMN in both children and adults. This finding is important for speech perception because tone patterns characterize the rapid and dynamic transmission of natural speech sounds and might reflect a widened TWI. Furthermore, individuals with dyslexia may also have difficulties with frequency modulation and gap detection, although replications of these preliminary findings would be required for any strong conclusions. Importantly, in some cases (Moisescu-Yiflach and Pratt, 2005; Stoodley et al., 2006) the results reveal how ERP studies more sensitively detect persistent deficits associated with dyslexia than psychophysical studies, where presumably high-functioning adults with dyslexia have compensated for their difficulties in reading and writing in a number of ways and developed cognitive strategies to enhance their performance in these tasks.

6. Speech specific auditory processing deficits

A second area of investigation in dyslexia has focused on examining perception specific to the acoustic processing of speech stimuli. A speech specific deficit conjures with research suggesting that the core deficit in dyslexia is phonologically based (Shaywitz, 1996; Ramus et al., 2003; Bishop and Snowling, 2004). This hypothesis underlines deficits in phoneme awareness, or the explicit knowledge about the sound structure of speech. In some cases, it has been argued that auditory deficits in dyslexia are specific to speech, and therefore cannot be attributed to general acoustic processing deficits (Mody et al., 1997; Schulte-Körne et al., 1998, 2001; Bishop and Snowling, 2004; Bitz et al., 2007).

Speech perception involves the mapping of basic auditory information onto phonological units. The relationship or boundary between acoustic sound features and phonemic processing is not well defined. In spoken language, a single phonemic sound's production is dependent on the surrounding phonemes, resulting in no fixed pattern for any phoneme (Lieberman and Blumstein, 1988; Tunmer et al., 1984). Therefore, phonemic units alone are abstract, but meaningful acoustic representations of speech parts. Not surprisingly, an auditory speech-processing deficit was determined in dyslexia in a number of ERP studies (see Table 4). The majority of studies have focused on MMN, as an index of successful

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Table 4
An overview of findings regarding consonant–vowel (CV) discrimination in both children and adults with dyslexia, including MMN and auditory evoked potential studies.

Article	Age controls	Age dyslexic	CV S	CV D	ERP group differences	ISI (ms)	Stimuli dur (ms)
Kraus et al. (1996)	6–15	6–15	/da/	/ga/	∗(MMN)	?	100
			/ba/	/wa/	n.s.		
Schulte-Körne et al. (1998)	12.6	12.5	/da/	/ba/	∗(MMN)	590	110
Cunningham et al. (2001)	10–13	10–13	/da/ in quiet	n.a	n.s.	550	40
			/da/ in noise	n.a	∗(P2,N2)(labelled P1́,N1́)		
			/da/ in cue-enhanced noise	n.a	n.s.		
Schulte-Körne et al. (2001)	Adults	Adults	/da/	/ga/	∗(MMN)	500	110
Maurer et al., 2003	6.6 Mean	6.5 Mean	/ba/	/ta/ & /da/	∗(MMR)	283	100
Warrier et al. (2004)	8–13	8–13	/da/ in quiet	n.a.	n.s.	590	40
			/da/ with background noise	n.a.	[∗] (N2)		
Giraud et al. (2005)	Adults	Adults	/ba/	n.a.	Sub-group 1: [∗] (extra components) Sub-group 2: [∗] (missing N240)	1030	380
			/pa/	n.a.	Sub-group 1: [∗] (extra components) Sub-group 2:[n.s.]		270
Lachmann et al. (2005)	8–11	8–11	/ba/ +	/da/ +	[∗](MMN), ∗(N250)	515	385
Meng et al. (2005)	8–13	8–13	/ba/	/ga/	∗(MMN)	700	40
			/dan/	/dai/	∗(MMN)		
			/ba1/	/ba2/	n.s.		
Moisescu-Yiflach and Pratt (2005)	Adults	Adults	/ta/	/pa/	∗(N1,N2, P2,P3)	1900	?
			/ba/	/pa/	∗(N1,N2, P2,P3)		
Cohen-Mimran (2006)	10–13	10–13	/pa/	/ba/	∗(P3)	2000	230
Bitz et al. (2007)	6–7	6–7	/ga/	/ka/	∗(MMN)	600	250
Shankarnarayan and Maruthy (2007)	7–12	7–12	/tΣa/	/dZa/[a] & /sa/[b]	[∗] (MMN)	SOA 526	250
			/da/	/ð8a/[c] & /da$_s$/[d]	[∗] (MMN)	SOA 526	250 & 175
Sebastian and Yasin (2008)	Adults	Adults	/ta/	/ka/	n.s.	638	362
			/ba/	/da/	n.s.		

Age indicates average age, or a given range. S = standard; D = deviant; dur = duration; ISI = interstimulus interval; SOA = stimulus onset asynchrony; ∗ = significant differences found between controls and dyslexics; [∗] = significant differences found for a sub-group only; n.s. = non-significant differences; From the Kannada language in South India. Stimuli differ in.

[a] Voicing.
[b] Manner of articulation.
[c] Place of articulatio.
[d] Vowel duration; n.a. = not applicable.

discrimination between formant transitions (FT) (spectral changes; e.g. /da/ vs. /ga/), with some research examining voice onset timing (VOT) transitions (temporal changes; e.g. /ba/ vs. /pa/).

6.1. Studies in children

Kraus et al. (1996) first reported attenuated MMN to FT in a group of learning disabled children and adolescents (aged 6–15). The children in the experimental group were defined by either a diagnosis of reading disorder, attention deficit disorder or both. Despite the heterogeneous make-up of the group, the children's performance on measures of reading was poorer than that of the age-matched controls. Participants were first tested on their ability to discriminate between two CV syllables, /da/ vs. /ga/ (spectral change) and /ba/ vs. /wa/ (temporal change). All learning disabled children were much poorer in both tasks, however some could discriminate /ba/-/wa/ contrasts relatively well. Therefore, in passive oddball paradigms comparing both CV pairs, learning disabled children who could sufficiently discriminate /ba/-/wa/, but remained poor /da/-/ga/ perceivers, were compared to age-matched controls, who performed well in both discrimination measures. Significant MMN (200–500 ms) to /da/-/ga/ was found for control children, but the amplitude of the MMN was diminished in the learning disabled group. Similar MMN was present for both groups to /ba/-/wa/ stimuli. MMN duration and area to /da/-/ga/ correlated moderately with /da/-/ga/ discrimination accuracy ($r = -.40$, $r = -.42$, respectively). Therefore, these findings suggest links between neurophysiological mechanisms as assessed by a MMN paradigm (i.e. independent of attention and response and actual discrimination ability at the behavioural level; prior to conscious perception). Furthermore, the discrimination deficits were highly specific, where deficits for contrasts differing in spectral content (/da/-/ga/), but not for contrasts differing in their temporal content (/ba/-/wa/), were found.

Deficits discriminating between CV changes with spectral information were reported in a number of other studies with children. Schulte-Körne et al. (1998) found an attenuated MMN (176–302 ms) in children with dyslexia (average 12.5 years) to /da/-/ba/ contrasts. In younger children (6–7 years) with a familial risk for dyslexia, Maurer et al. (2003) reported earlier attenuation (109–140 ms) of a component similar to MMN, labeled MMR (mismatch response), to both /ba/-/ta/ and -/da/ contrasts. MMR is described as a component that emerges in place of MMN in very young children when differences between stimuli are small and ISIs are very short (in this experiment 283 ms). Short ISIs on the other hand do not allow for analysis of later components, which are dependent on longer processing times. Therefore, with longer ISIs these children may have also revealed deficits in later processing stages. MMN deficits were further reported in Chinese children (8–13 years) to da/-/ga/ stimuli in a very early, atypical time window (0–100 ms) (Meng et al., 2005). Finally, Lachmann et al. (2005) examined the discrimination of /ba/-/da/ contrasts in children (8–11 years) with dyslexia and age-matched controls. The children with reading problems were analyzed according to sub-groups. The first group was poor in fluent word reading only, whereas the second was marked by impairments in either non-word reading only or both non-word and fluent word reading. Surprisingly, only those children marked by isolated frequent word reading deficits had an attenuated MMN from 98–198 ms. A later time window was not examined. This result is unexpected, as those

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children who are marked by phonological impairments would be expected to do most poorly on CV discrimination tasks. The reading of non-words requires good phoneme awareness in order to correctly apply grapheme–phoneme correspondence rules. Therefore, children who performed most poorly in the phonological task might be expected to discriminate the CV stimuli more poorly. These data suggest that grapheme–phoneme correspondence failure, as indexed by non-word fluency, is not a result of early phonemic discrimination processes. Interestingly, the analysis of exogenous ERPs to deviant stimuli revealed an impaired N250 in both sub-groups. N250 is thought to index general aspects of audition and sound reception (Shafer et al., 2000; Ceponiene et al., 2001); therefore abnormal N250 is suggestive of a more general impairment to sound reception in dyslexia. In accordance with Lachmann et al.'s results, Warrier et al. (2004) also reported abnormal latencies in the N250 range to /da/ stimuli presented with background noise in a subset of children with learning problems, but not in others. It is however unclear how many or if any of the children in this study had dyslexia. Reduced amplitudes to /da/ in noise within this time window have been reported elsewhere (Cunningham et al., 2001). Similar to these results, Shankarnarayan and Maruthy (2007) reported individual results for both spectral and temporal stimuli. The authors found that about 2/3 of the children with dyslexia tested ($n = 15$) exhibited prolonged or absent MMN latencies, whereas 1/3 revealed comparable MMN to controls. These children were not however further classified into sub-groups based on behavioural measures.

Despite normal MMN to temporal information (/ba/-/wa/) in Kraus et al.'s study, deviant MMN to VOT was found in two studies with children. Bitz et al. (2007) examined /ga/-/ka/ contrasts in 6–7 year olds at risk for dyslexia, defined by poor phonological decoding skills, compared to age-matched controls. The authors reported two significant late MMN windows in the control children (300–450 ms and 450–600 ms). Later MMN-like activity is also referred to as late discriminative negativity (LDN; Cheour et al., 2001). LDN might be less sensitive to stimulus specific characteristics than MMN (Ceponiene et al., 2002) and is believed to reflect further processing of a deviant stimulus beyond sensory sound discrimination. LDN is, for example, enhanced to words in comparison to non-words matched on all levels of acoustic complexity, thus suggesting some association to the cognitive meaning of these stimuli (Korpilahti et al., 2001). However, the exact nature of this late discriminative processing is not well understood. Bitz et al.'s findings revealed attenuation in both MMN windows for the children with phonological deficits. Furthermore, similar to the findings from Kraus et al. (1996) moderate correlations were found between MMN amplitude (at electrodes FC3/FC4) and phonological decoding abilities ($r = .42$). Here, increasing MMN amplitude indicated enhanced behavioural performance, suggesting a potential link between pre-attentive discrimination at the neural level and phonological skills. Finally, in an active paradigm Cohen-Mimran (2006) found prolonged P3 latencies but normal P3 amplitudes to /pa/ vs. /ba/ stimuli in 10–13 year old Hebrew children diagnosed with a reading disorder. This was coupled with reduced accuracy and longer reaction times. Correlations between later P3 latencies at Cz and poorer phonological awareness skills ($r = .-69$, $p < .001$) were also reported.

6.2. Studies in adults

Schulte-Körne et al. (2001) reported a diminished LDN to /da/-/ga/ contrasts (490–620 ms) in adults with dyslexia who were recruited based on poor spelling achievement, and who were diagnosed with dyslexia (poor reading and spelling) as children. MMN in an earlier time window (270–320 ms) was not significantly attenuated.

Reporting on auditory evoked responses Giraud et al. (2005) found evidence for two distinct response patterns to voiced (/ba/) and voiceless (/pa/) CV stimuli in adult participants with persistent reading deficits. In healthy subjects both stimuli elicited a P1/N2 complex, and additionally /ba/ stimuli evoked an N240. N240 indexes the onset of voicing by /ba/, but is not present for voiceless /pa/ stimuli. Half of the participants with reading deficits elicited an extra, earlier component at 50 ms (P50) to both stimuli and had delayed off-responses. Despite these differences, these participants processed /ba/ stimuli in a similar manner as controls, as indexed by the presence of N240 to /ba/. P50 was absent in the other half of the participants to both stimuli, who also lacked N240 to /ba/, thus ERP morphology was similar for both /ba/ and /pa/. These results suggest diversity in acoustic processing deficits in dyslexia, despite similar reading abilities. The first pattern described here is characterized by additional acoustic processing and slowness in acoustic processing, yet a sustained ability to process voiced /ba/. The second pattern is characterized by an inability to perceive and code important speech cues, such as those differentiating voiced and voiceless CVs.

Moisescu-Yiflach and Pratt (2005) examined both temporal and spectral acoustic qualities of speech stimuli using /ta/-/pa/ (spectral cue) and /ba/-/pa/ (temporal cue) in a group of high-functioning adults with dyslexia. For both CV contrasts, active and passive oddball paradigms were employed to rule out potential confounds resulting from attention, response strategy or motivation on the neurophysiological response. Psychophysical results revealed equally good discrimination in the active condition across groups. However, at the neurophysiological level a number of differences were found. Regardless of whether CV contrasts involved spectral or temporal cues, passive listening revealed longer N1 latencies in dyslexia and active participation resulted in prolonged N1, P2 and P3 latencies. Furthermore, higher N1 amplitudes were found within the dyslexic group in the active conditions.

The N1 is the most prominent exogenous component in response to acoustic input in adults and is thought to index basic encoding of acoustic information at the moment it enters primary auditory cortex (e.g. Näätänen, 1990; Nagarajan et al., 1999). However, the N1 response is complicated by a number of sub-processes that contribute to its morphology (Key et al., 2005; Näätänen and Picton, 1987; Wood, 1995). Although it is not believed to be the same component as the N250 in children (e.g. see Lachmann et al., 2005), these two components are thought to be correlates of similar functions (Ceponiene et al., 2001). In this study (Moisescu-Yiflach and Pratt, 2005), the N1 was found to be irregular in subjects with dyslexia for both spectral and temporal information, suggesting that the speech-processing deficit is general.

Finally, Sebastian and Yasin (2008) studied spectral and temporal contrasts in university students diagnosed with dyslexia as children. As in Stoodley et al.'s (2006) study, these individuals were considered to have compensated for their reading deficits in some manner in order to have achieved an academic standing required for university entry. Sebastian and Yasin (2008) reported no group differences in MMN amplitudes and latencies. This is the only study to report a lack of speech-processing deficit.

7. Speech perception as an early predictor

Recording ERPs elicited by passive listening to speech sounds at birth seems to be a promising method for identifying early predictors of later reading disorders. Molfese (2000) applied a passive listening experiment to 186 full-term babies within 36 h of birth. Synthetic speech stimuli /gi/, /bi/ and /di/ were presented during sleep. Discriminate analysis at 8 years of age was conducted in order to classify the children according to speech ERPs at birth. The

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children were either diagnosed with dyslexia ($n = 17$), were poor readers (low IQ and reading ability; $n = 7$), or were normal readers (control children; $n = 24$). Eighty-one percent of a subset ($n = 48$) of children could be correctly classified into their reading categories based on three ERP components (N1, P2 and N2) at birth and reading and IQ scores. The ERPs at birth could successfully discriminate 76.5% of children with dyslexia, 100% of poor readers and 79% of control children at 8 years of age (Molfese, 2000). Furthermore, correlations between the amplitude and latency of N1, measured between ages 1 and 4, and word reading at age 8 were significant (Espy et al., 2004). In an additional analysis the ERP components, home environment (e.g. academic stimulation, stimulation of communicative competence), socioeconomic status (e.g. parental education and occupation), preschool language score and short-term memory were integrated into a regression analysis (Molfese et al., 2001). Up to 70% of the variance of word reading was explained by these factors. The ERP components, as well as preschool speech perception, made the strongest contribution (half or more of the variance) to reading. It can therefore be concluded that speech perception influences reading abilities at a very early age. Interestingly, the neurophysiological correlates made a stronger prediction than the home environment and the SES.

In a second longitudinal study (Jyväskylä Longitudinal Study of Dyslexia, (Lyytinen et al., 2005a, b)) ERPs to speech stimuli were registered in one hundred babies at risk (defined as one first grade relative reporting dyslexia) and compared with ERPs from 100 babies from control families. Both synthetic and natural spoken speech stimuli were presented (Leppanen et al., 1999; Pihko et al., 1999; Guttorm et al., 2005). Even at birth, distribution differences were found for the standard and deviant response to the natural speech sound /ka/. In the at-risk population an ERP component between 540 and 630 ms at birth elicited by synthetic speech stimuli had a larger and prolonged response in the right hemisphere due to a slower polarity shift from the major positive to the negative deflection. In the control group the differences between standard and deviant stimuli were largest in the left hemisphere, whereas in the at risk group the ERP difference was largest in the right hemisphere (Guttorm et al., 2005; Leppanen et al., 1999). The anomalous ERP activity in the right hemisphere in the at-risk children correlated with lower word and non-word reading accuracy in the first grade of school (Lyytinen et al., 2005a, b) poorer language skills at 2.5 years; verbal memory at 5 years (Guttorm et al., 2005) and reduced phonological skills, slower lexical access and less knowledge of letters at 6.5 years (Guttorm et al., 2009). These findings suggest that already at birth the speech processing in at-risk children differs from controls and that the neurophysiological correlate of this functional difference is located over the right hemisphere. It is not yet clear how well the ERPs will also predict an actual dyslexia diagnosis. However, the poorer language, memory and phonological skills found at ages 2.5, 5 and 6.5 in the at-risk children are often found in older children and adults who have a diagnosis of dyslexia, thus it would not be surprising if a significant portion of the at-risk children will eventually develop dyslexia.

8. Summary

Speech specific processing deficits to CV stimuli have been shown consistently in both children and adults, for both spectral and temporal transitions, and in active and passive paradigms. Early MMN deficits suggest difficulties discriminating between two stimuli, whereas late MMN abnormalities might be indicative of faulty long term memory traces (Näätänen, 2001). Finally, auditory ERPs at birth can be predictive for later reading skills and short-term verbal memory in children at risk for dyslexia.

A number of authors, including our own group, have argued for or against a speech specific vs. a general auditory processing deficit in dyslexia based on isolated studies (e.g. Bitz et al., 2007; Schulte-Körne et al., 1998, 2001). Unfortunately to date no study of dyslexia has addressed this question using equally comparable speech and non-speech stimuli (e.g. such as those of Tampas et al., 2005), and tone conditions were often sinus tone comparisons with differences greater than 100 Hz. Given the above review, it is clear that certain non-speech deficits are apparent in dyslexia, and these are attributed to discrimination of small frequency differences and to detection of differences in complex tone patterns, both of which are relevant to speech discrimination.

9. Conclusion and perspectives

In this review we cover ERP research on basic auditory and visual processing in dyslexia. Higher cognitive processes, such as phonological processing, word recognition and orthographic processing are also impaired in dyslexia, but were not addressed. A review on the ERP literature pertaining to these areas would also be helpful to conclude the current understanding of the electrophysiology of dyslexia.

Throughout this review we have highlighted the neurophysiological literature pertaining to altered visual and auditory sensory processing in dyslexia. The literature touches on infancy, childhood, adolescence and adulthood, demonstrating the persistence of these information processing deficits throughout the lifespan, yet does not present evidence for causality. For example, the identification of the phase locked processes in auditory and visual processing points to sensory processes that might be important for the underlying development of reading, but these findings can not be strongly linked to reading and writing deficits.

Given the heterogeneity of the research reported thus far, it is important to consider the methodologies employed across studies. The following concerns are important to take into consideration for future research.

First, the careful selection of individuals with dyslexia for empirical investigation is of great importance. With the evidence for sub-groups of dyslexia mounting and the high comorbidity rates reported, it is apparent that different selection procedures certainly influence the ERP results. Thus, we emphasize the importance of developing stringent inclusion criterion to be applied across labs. As mentioned, the comorbidity between dyslexia and ADHD is very high, but dyslexia is also comorbid with other disorders including anxiety disorder (Caroll & Iles, 2006), ADHD (Stevenson et al., 2005), antisocial behavior (Maughan et al., 1996), and depression (Willcutt and Pennington, 2000). Most of the ERP studies reviewed did not screen for the existence of ADHD symptoms in their sample of individuals with dyslexia. Assuming that 25% of dyslexic cases are also characterized by comorbid ADHD, it is reasonable to assume that in the ERP studies discussed many children with dyslexia also would be characterized by ADHD. Due to the fact that ADHD strongly influences visual and auditory ERP responses (for review see Banaschewski and Brandeis, 2007); this unexposed comorbidity might be a simple explanation of many of the heterogeneous findings.

Secondly, the diagnostic criteria widely differ between studies. Whereas lower word reading is one of the most common criteria for defining dyslexia, the usage of a discrepancy criterion (e.g. age, grade or IQ discrepancy) is quite different, additionally the magnitude of discrepancy (e.g. 1, 1, 1.5 or 2 standard deviations below the norm) varies greatly. These differences might lead to the identification of different sub-groups from the large population suffering from dyslexia and to different grades of severity of affectedness. Since it has been recently shown that genetic effects on

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dyslexia is for some candidate genes higher in severely affected individuals only (e.g. Schumacher et al., 2006), this aspect of selection bias could also influence results of neurophysiological studies.

Furthermore, due to unspecified criteria for dyslexia diagnosis, plus uncontrolled possible comorbidities of other developmental disorders like ADHD or specific language disorder (developmental dysphasias), neurophysiological studies in this field should be interpreted with caution rather than as established scientific knowledge. In addition, there is a need for clinical control groups in future studies.

Turning toward the future we see two emerging and promising research areas. First, the early identification of children who are at risk for dyslexia is appealing. This perspective will lead to the possibility of successfully preventing reading problems before children enter the school system and before behavioural problems in primary and secondary school have a chance to develop. Currently, accurate tools to diagnosis dyslexia exist for older children only, generally first available in the 3rd or 4th grades, which is relatively late. The consequence of diagnosing dyslexia so late, as well as the late identification of reading problems, is that children have already accumulated negative experiences in school, such as failure to perform well in reading exercises despite adequate instruction. This often leads to detrimental psychological and social problems. However, the only available ERP study predicting reading development at age 8, based on ERP measures at age 3, showed that only in combination with behavioural measures of language and measures of home environment could ERPs significantly predict reading ability (explaining 60–70% of reading ability variance) (Molfese et al. 2001).

From a clinical perspective we believe there is only minimal evidence for both the application of non-linguistic visual or auditory stimuli measurements for diagnosing dyslexia, as well as for the development and usage of basic perceptual interventions, such as temporal processing trainings. Although the ERP research presented generally demonstrates deficits in early perceptual components, it is not possible to conclude whether these findings are a result of altered top down regulation processes or bottom-up perceptual processes. Attention might be an important candidate for investigations on top-down control as increasing evidence shows its role in modulating both visual and auditory sensory processing.

From a research perspective, besides the methodological aspects that were discussed above, we encourage the introduction of ERP research into molecular genetic studies. The heritability of different ERP components (P300, MMN) have been shown to be high: approximately 60% of the variance of these components can be explained by genetic factors (Hall et al., 2006). Linkage studies that aimed to identify susceptibility genes for an ERP trait, i.e. component, were able to find suggestive evidence for loci at chromosome 10 for a slow wave correlated with working memory (Hansell et al., 2006). In dyslexia, the speech MMN has proven to be an excellent candidate trait marker. Roeske et al. (2009) identified a relationship between MMN and a trans-regulation effect on *SLC2A3*, a gene on chromosome 12, which is a predominant facilitative glucose transporter in neurons in children. The attenuated MMN found for the subjects with dyslexia was related to the risk haplotype, which is thought to lead to a reduced expression of the *SLC2A3* gene. Thus, this reduction in glucose levels within the brain during childhood might impact brain development and function. To our knowledge this is the first study that shows a patho-physiological pathway from gene expression to altered brain functions (i.e. reduced speech MMN) in dyslexia, and therefore we find that this is a promising and powerful research strategy which might lead to stronger conclusions about causality, and which goes beyond study designs of group comparisons based on small samples of affected and non-affected subjects.

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