Auditory and speech processing in Specific Language Impairment (SLI) and dyslexia

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I, *Outi Tuomainen*, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

ABSTRACT

This thesis investigates auditory and speech processing in Specific Language Impairment (SLI) and dyslexia. One influential theory of SLI and dyslexia postulates that both SLI and dyslexia stem from similar underlying sensory deficit that impacts speech perception and phonological development leading to oral language and literacy deficits. Previous studies, however, have shown that these underlying sensory deficits exist in only a subgroup of language impaired individuals, and the exact nature of these deficits is still largely unknown.

The present thesis investigates three aspects of auditory-phonetic interface: 1) The weighting of acoustic cues to phonetic voicing contrast 2) the preattentive and attentive discrimination of speech and non-linguistic stimuli and 3) the formation of auditory memory traces for speech and non-linguistic stimuli in young adults with SLI and dyslexia. This thesis focuses on looking at both individial and group-level data of auditory and speech processing and their relationship with higher-level language measures. The groups of people with SLI and dyslexia who participated were aged between 14 and 25 and their performance was compared to a group of controls matched on chronological age, IQ, gender and handedness.

Investigations revealed a complex pattern of behaviour. The results showed that individuals with SLI or dyslexia are not poor at discriminating sounds (whether speech or non-speech). However, in all experiments, there was more variation and more outliers in the SLI group indicating that auditory deficits may occur in a small subgroup of the SLI population. Moreover, investigations of the exact nature of the input-processing deficit revealed that some individuals with SLI have less categorical representations for speech sounds and that they weight the acoustic cues to phonemic identity differently from controls and dyslexics.

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ABBREVIATIONS

ANOVA analysis of variance

CA chronological age control

CP categorical perception

D' d-prime

DYS dyslexia

EEG electroencephalogram

ERP event-related potential

 $\mathbf{F_1}$, $\mathbf{F_2}$, $\mathbf{F_3}$ 1st, 2nd and 3rd formant frequencies

FL foreign language

MMN mismatch negativity

NL native language

NS not significant

PME perceptual magnet effect

PSE point of subjective equivalence

RATP rapid auditory temporal processing theory

ROI region of interest

SD standard deviation

SE standard error

SLI Specific Language Impairment

SWS sine-wave speech

T₁, T₂, T₃ MMN analysis time-windows

1. Thesis outline

The main objective of this thesis is to investigate auditory-phonetic processing in Specific Language Impairment (SLI) and dyslexia. For this reason, I cover the main aspects of how speech is perceived, how speech perception develops in early infancy and what is known about the relationship between lower-level input-processing and linguistic deficits, such as SLI.

In the theoretical part of the thesis, I start off by describing the nature and characteristics of SLI (Chapter 2) and dyslexia (Chapter 3). I review the main theories of the underlying causes of SLI/dyslexia focusing mainly on auditory processing and its causal role in these impairments. After establishing that, I describe the theoretical framework of speech perception and the development of phonological categories (Chapter 4). Specific goals and objectives of the experimental part of the thesis are provided in Chapter 5.

2. Specific Language Impairment

2.1 Introduction

The vast majority of children acquire language effortlessly and quickly and continue to do so throughout childhood and early adolescence. However, for about 7% of English speaking children this almost automatic task of acquiring a language does not follow the normal course. Specific Language Impairment (SLI) is a developmental disorder where children lag behind their peers in language production and comprehension without any apparent reason (Bishop, 1997). SLI by definition is not secondary to the factors that usually induce language problems, such as focal brain lesion or traumatic brain injury, hearing loss or even environmental factors. Furthermore, in SLI the language problems occur without any other apparent cognitive impairment, that is, despite relatively normal non-verbal intelligence. Therefore, SLI is diagnosed by its *specificity* to language (for a review see Leonard, 1998).

Generally, certain core areas of language, such as syntax, morphology and phonology are impaired in SLI. Furthermore, in addition to these linguistic deficits, many children show persisting word-finding and vocabulary difficulties or even communicative (or pragmatic) impairments. However, these different components of language are not impacted to the same degree in all children, resulting in relatively heterogeneous linguistic profiles in SLI.

Outside this linguistic heterogeneity, some SLI children also show co-occurring deficits in areas of functioning that seem to require little or no language ability. These non-linguistic weaknesses include deficits in auditory perception, cognitive functions and even in motor abilities (Bishop, 1997; Leonard, 1998). In auditory perception, impaired processing of the incoming acoustic signal can be identified in some SLI children. However, not all children with SLI show this deficit (van der Lely, Rosen & Adlard, 2004; McArthur & Bishop, 2005). In addition to the co-occurring auditory deficit, many SLI children are notoriously poor in non-word repetition and sentence recall which could indicate problems in phonological short-term memory (PSTM) (Gathercole & Baddeley, 1990) and they also show weaknesses in mental representation and symbolic play (Leonard, 1998).

However, the behavioural and electrophysiological data on these co-occurring deficits is inconclusive. There is an increasing body of evidence that these deficits do not systematically appear in conjunction with SLI (Bishop, Carlyon, Deeks, & Bishop, 1999; Rosen, 2003). In order to investigate the underlying causes of SLI we need to understand linguistic and non-linguistic heterogeneity. Therefore, first, we need a through description of the characteristics of SLI to investigate how these linguistic and non-linguistic deficits relate to each other.

2.2 Characteristics of SLI

Children with SLI generally acquire their first words later than typically developing children. They produce fewer utterances and occasionally even errors that are uncommon in children with normal language. However, the linguistic problems in children with SLI are particularly prominent in certain components of grammar, that is, in syntax, morphology and phonology.

In the following Sections, I review the linguistic profile in SLI with respect to these three components and describe the non-linguistic deficits found in SLI.

2.2.1 Syntax

Children with SLI have difficulties in understanding of complex grammar. This deficit is apparent in situations where they have to rely solely on the syntactic information, that is, in situations where semantic and pragmatic knowledge cannot guide them. Comprehension and production of complex grammar have been widely investigated by van der Lely and her colleagues (van der Lely & Stollwerk, 1997; van der Lely, 2005; Marshall & van der Lely, 2006). Van der Lely argues that children with SLI (or a subgroup of children with SLI, see below Section 2.4.2) have particular difficulties with structural relations involving non-local syntactic dependencies. Van der Lely (2005) has argued that the syntactic deficit is characterized by impairment in structures that involve "movement" (e.g., reversible passives) and/or "binding" (pronominal sentences). These structural difficulties manifest as impaired tense marking, assignment of thematic roles in passive sentences, wh- questions, and relative clauses. Furthermore, SLI children also show weaknesses in marking subject-verb agreement and in pronominal and anaphoric reference assignment (Bishop, 1979; Leonard, 1995; van der Lely & Stollwerck, 1997; van der Lely & Ullman, 2001; van der Lely & Battell, 2003; Montgomery & Evans, 2009). For example, in a sentence Mowgli says Baloo bear is tickling him/himself, SLI children have been reported to show difficulties determining who him/himself refers to (Mowgli or Baloo bear) and in a sentence The boy was pushed by the girl, SLI children often fail in interpreting of "who does what to whom".

These syntactic deficits are observed not only in English but in other languages such as in French, Greek, Hebrew and Italian (Jakubowicz, Nash, Rigaut, & Gérard, 1998; Friedmann & Novogrodsky, 2004; Adani, Guasti, & van der Lely, 2006; see Leonard, 1998 for a review).

2.2.2 Morphology

In addition to these marked syntactic difficulties, children with SLI have weaknesses in inflectional morphology resulting in the omission of regular past tense suffix -ed and 3^{rd} person singular suffix -s in obligatory contexts, (Rice & Wexler, 1996; Marshall & Lely, 2006) or they use these suffixes in inappropriate contexts (van der Lely & Battell, 2003).

There are two opposing views concerning the mechanisms that underlie regular and irregular English past tense formation. On one account, regular suffixed forms are stored and processed in the same way as irregular past tense forms, within a single associative system, that is, through stored full-form representations (Joanisse & Seidenberg, 1999). In contrast, the dual mechanism model (Pinker, 1991) claims that regular and irregular past tense formation is governed by two distinct cognitive mechanisms. According to the dual mechanism model, regular past tense inflection is achieved by applying combinatory rules (add - ed) that decompose inflected word forms into morphological constituents, whereas irregular forms (e.g., sing - sang) are processed associatively (Pinker & Ullman, 2002).

If the production of irregular past tense is dependent on associative lexical memory, and the production of regular forms is not, then one would expect lexical factors (such as item frequency) to affect irregular but not regular forms (van der Lely & Ullman, 2001). Because the majority of English-speaking SLI children produce a substantial number of bare stem forms in obligatory past-tense contexts (e.g., *Yesterday I walk.), it has been suggested that there is an impairment in the suffixation rule in SLI (Pinker, 1991, van

der Lely & Ullman, 2001). To test this hypothesis, van der Lely and Ullman (2001) investigated past tense production in children a subgroup of SLI children, namely in the Grammatical (G)-SLI, and in three groups of language-matched controls. They found two surprising patterns in the data. Firstly, the G-SLI group failed to show the advantage for regular verbs over irregulars that control children showed. Secondly, for the G-SLI group, item frequency affected the regular past tense formation —a pattern not observed in the controls.

On the basis of these findings, van der Lely and Ullman (2001) concluded that regular past tense formation is rule-governed and that this suffixation rule is impaired in SLI. Furthermore, they claimed that SLI children rely on storing regular past tense forms in lexical memory, hence the frequency effect for regular verbs.

Further evidence for the claim that SLI children store both regular and irregular forms is provided by van der Lely and Christian (2000) and Marshall and van der Lely (2006). Van der Lely and Christian (2000) investigated compound-formation in SLI and reported that G-SLI children used both regular and irregular plurals inside compounds (*rats-eater, mice-eater), indicating that both irregular and regular forms are stored similarly and thus available for same morphological processes. This pattern of regular plural inside compounds is very rarely observed in typically developing children.

Moreover, Marshall and van der Lely (2006) investigated the role of phonotactics in past tense formation. They argued that if children apply a morphological rule in forming the regular past tense then the phonotactic properties of a word (e.g., cluster frequency) should not affect their performance. However, if children store these past tense forms, phonotactic properties should have an effect on the use of past tense forms. Marshall and van der Lely (2006) found that phonotactic properties of the verb can affect the mechanisms of regular past tense formation in G-SLI. They noticed that G-SLI children were more likely to omit past tense forms containing phonotactically illegal clusters (and hence less frequent), an effect, again, not observed in typically developing children.

2.2.3 Phonology

Children with SLI children are generally delayed in acquiring the segments of the language (i.e., their native language phonemes) and end up with proportionally smaller phonological inventories (Fee, 1995; Rescorla & Ratner, 1996). However, despite the smaller phoneme inventories, the general order in which these native language phonemes are acquired usually tend to follow the same developmental path as in typically developing children (Rescorla & Ratner, 1996; Leonard, 1998). One possibility is that these phonological limitations arise from deficits in the sub-segmental level, for example, from impaired or inaccurate production of distinctive features (Chomsky & Halle, 1968). Several studies adopted this approach but failed to find any systematic error patterns in the SLI data (Leonard, 1973; Schwartz, Leonard, Folger, & Wilcox, 1980) (See more on distinctive features in Chapter 4).

In aspects of phonological processing, many children with SLI are also notoriously poor at repeating novel sequences of sounds, i.e. in non-word repetition (Gathercole & Baddeley, 1990; Bishop, North, & Donlan, 1996). Accurate repetition of non-words requires the ability to retain a phonological representation of the non-word in phonological short-term memory (PSTM) because representations of these novel forms are unlikely to be memorized. PSTM is characterized as a temporary store of phonological information that is essential for the formation of phonological representations and play a role in vocabulary development. A usual pattern is that the repetition accuracy deteriorates when the number of syllables in non-words increases, and this hits the SLI children especially hard (Gathercole & Baddeley, 1990; Bishop et al., 1996). These findings lead to a conclusion that children with SLI have limited PSTM capacity, and that this deficit in PSTM restricts lexical learning and can impact grammar in SLI (Gathercole & Baddeley, 1990). However, the relationship between memory and language ability may not be that straightforward and many researchers agree that nonword repetition may involve multiple processes. Moreover, the non-word repetition performance is affected by several test-related factors such as wordlikeness of the nonwords, non-word length and articulatory complexity (see Estes, Evans, Else-Quest, & Nicole, 2007).

Some researchers (van der Lely & Howard, 1993) have claimed the causal relationship between memory and language impairment could actually be in the opposite direction i.e. that, a linguistic deficit causes the phonological short-term memory (PSTM) deficit, rather than vice versa. In this fashion, Gallon, Harris, & van der Lely (2007) investigated the effect of prosodic complexity on the non-word repetition scores in SLI by manipulating the properties of three parameters within a non-word: the onset (simple vs. complex), rhyme (open vs. closed) and word ending (vocalic vs. consonantal). They reported that increasing the complexity caused a systematic increase in errors in SLI, observed even in short non-words suggesting that a deficit in phonology that is independent of a short-term memory deficit can exist.

2.2.4 Auditory processing

When learning the mother tongue, an infant must first acquire the phonemic categories of the ambient language. The acquisition of native language phonemes takes place rapidly during the first 12 months of life, during which infants learn to gradually tune into the relevant features of their mother tongue (Werker & Tees, 1984; Kuhl, 2000). This acquisition process generally requires relatively accurate perceptual capacity and most children show excellent speech discrimination from a very early age (Aslin, Pisoni, Hennessy, & Perey, 1981; Kuhl & Miller, 1982; Werker & Tees, 1984). Successful spoken language comprehension also involves processing of rapid sequential information encoded in the fast-fading auditory signal. Failures in this task may indicate problems in language learning.

To investigate the relationship between perceptual accuracy and higher-level language impairments, Tallal and colleagues (Tallal & Piercy, 1973; Tallal & Piercy, 1975; Tallal & Stark, 1981) looked at children's ability to distinguish different speech and non-speech contrasts. Two types of tasks were used in language impaired and typically

developing children: they were asked to discriminate between sounds that were presented in pairs ("same-different" judgement task) or to identify the order in which the sounds were presented (with varying inter-stimulus-intervals, ISIs). These studies showed that children with SLI were sensitive to two factors: the length of the stimulus and the ISI as defined by higher number of errors and longer reaction times. Tallal concluded that there is evidence that some SLI children fail to distinguish between sounds that are either presented rapidly, are brief in duration or contain rapid transitions in frequency (such as formant transitions distinguishing place of articulation, $[b_{\Lambda}]$ – $[d_{\Lambda}]$). Tallal and her colleagues proposed that this impaired input-processing is the primary factor causing the language learning problems in SLI (Tallal & Piercy, 1975; Tallal, Stark, Kallman, & Mellits, 1980), see review of the theory in Section 2.4).

Sussman (1993) studied the discrimination and identification ability of English [bʌ] – [dʌ] contrast in 5-6 years old children with SLI and their age and language matched controls. In contrast to Tallal, Sussman (1993) found that the SLI children showed relatively normal discrimination ability in the CV contrast but they seemed to be less accurate than the controls in the identification task. The poor identification performance was particularly prominent in the most prototypical sounds (i.e. in the category representatives, see Chapter 4), as manifested by generally shallower identification slopes. Sussman (1993) concluded that the main problem in SLI is not a failure to appropriately discriminate sounds but in forming phonological representations and actually linking acoustic information to these representations. However, Coady et al. (2005) failed to find similar pattern of results in their identification experiment and concluded that poor performance on speech perception tasks in SLI could be attributable to factors such as memory load and task demands.

To account for these contradictory findings, Leonard (1998:276) speculated that on the basis of the vast amount of data on the input-processing abilities in SLI, the processing

limitations might develop because children with SLI are initially unable to focus on the relevant or "ideal" acoustic cues as they begin to tune into their native language.

By using another paradigm, Wright and colleagues (1997) found that children with SLI were significantly impaired in backward masking where a brief tone (target) is followed by a masking noise. On the basis of their results, Wright et al. (1997) concluded that this auditory deficit could degrade the perception of the rapidly occurring elements of speech. This backward masking problem could potentially explain why contrasts such as $[b_{\Lambda}] - [d_{\Lambda}]$, where the consonant is followed (and masked) by the following vowel, are particularly difficult for SLI children (however, see Rosen, 2003 for discussion). Rosen and colleagues (2009) investigated auditory processing in the grammar specific subgroup of SLI children that should not, by definition, demonstrate auditory problems. Rosen et al. (2009) reported that, as a group, the G-SLI children had higher thresholds in both simultaneous and backward masking condition when compared to their agematched controls. This group-level difference, however, was due to 6 out 14 SLIs performing poorly on either or both of these tasks. Moreover, the auditory and language skills did not correlate in either SLI or control groups. On the basis of these data, Rosen et al. (2009) argued that auditory deficits are more likely only to be associated with SLI but not cause it. Furthermore, also Bishop and colleagues (1999) found no systematic difference in auditory tasks between LI twins and age and IQ matched control twins.

On the whole, the behavioural data on auditory processing in SLI is highly controversial and there seems to be no systematic evidence that auditory deficits are a necessary or sufficient cause of language impairments. In fact, on the basis of a large body of data, it is very likely that these auditory deficits only occur in association with language impairments –not as an underlying deficit.

However, it has been proposed that decision making, attention and compensation could potentially affect the behavioural measurements and decrease their sensitivity to detect

subtle perceptual deficits. To account for this, some recent studies have combined the traditional behavioural measures with electrophysiological techniques (ERPs).

Kraus and her colleagues (1996) studied the relationship between discrimination ability and learning difficulties in a large group (n=91) of language impaired (LI) children and normal children (n=90). In this study, Kraus et al. (1996) combined the behavioural discrimination measures with its neurophysiological correlate that can be elicited preattentively (i.e., the mismatch negativity, MMN, component of auditory ERPs, see Section 4.5) They investigated the discrimination of rapid spectrotemporal changes, as in contrast [da] vs. [ga] where the primary change is in onset frequency of F2 transition, and in contrast [b_{\Lambda}] vs. [w_{\Lambda}] where the difference is in the duration of the formant transition. They found that, firstly, that the discrimination of $[d\Lambda] - [g\Lambda]$ contrast was poorer than the discrimination of $[b\Lambda] - [w\Lambda]$ in both groups, and, secondly, that the behavioural discrimination accuracy was correlated with the electrophysiological measures. However, the language impaired group showed a particular difficulty in discriminating the spectral contrast [da] - [ga] suggesting that the perception of all acoustic cues may not be impaired to same extent but processing of some aspects of the signal, such as frequency, may be more profoundly affected. Kraus et al. (1996) concluded that the underlying cause of some language problems may be a central discrimination deficit that occurs before conscious perception and therefore best detected with electrophysiological measures. However, in the study of Kraus and colleagues (1996) the children with "learning disability" group actually consisted of children with diagnosis of learning disability, attention deficit disorder (ADD) or both, making generalisations to SLI population fairly difficult.

Neville and colleagues (1993) compared SLI children and their age-matched controls in three tasks: on auditory tone-detection task, a visual target-detection task and a lexical processing task. They found that in the auditory tone-detection task (standard 1000 Hz, target 2000 Hz) the SLI children as a group did not differ from their controls. However,

when children with SLI were re-grouped on the basis of their performance in behavioural rapid processing task (Tallal & Piercy, 1973), the poor performers also showed reduced processing capacity, as indicated by reduction in amplitude, in the detection task. In the visual task, the SLI children had lower amplitudes in the early visual components. Lastly, some of the SLI children had atypical voltage distribution of the N400 component that reflects lexical processing, suggesting abnormal hemispheric specialization. Neville and colleagues (1993) suggested that there are neurophysiological correlates for auditory processing accuracy -but in the SLI population the auditory deficit does not necessarily co-occur with the linguistic problems. Recently, using similar auditory monitoring task Fonteneau & van der Lely (2008) showed that adolescents with (G-)SLI did not differ from their typically developing controls in auditory pure-tone discrimination task and in semantic processing. Fonteneau & van der Lely (2008) argued that these grammatically impaired children showed a selective impairment in automatic grammatical processing as measured with Early Left Anterior Negativity (ELAN) that according to them indicates that not all linguistic deficits are necessarily caused by underlying input-processing deficits. However, the auditory task used in this experiment consisted of similar stimuli used by Neville and colleagues, i.e., the difference between standard and deviant sound was of a magnitude of an octave. Therefore, it is not surprising that no group effects were detected with these stimuli in the auditory task. Korpilahti & Lang (1994) examined the frequency and duration discrimination in SLI with the mismatch negativity (MMN) component of auditory event-related potentials. They found that the dysphasic (SLI) group had attenuated amplitudes to frequency change (500/553 Hz) and a significant difference in duration change with extreme contrasts (50/110 ms or 50/500 ms). Furthermore, they reported differences in hemispheric symmetry of MMN response for tonal stimuli between groups. Similarly, Holopainen and colleagues (Holopainen, Korpilahti, Juottonen, Lang, & Sillanpaa, 1997; Holopainen, Korpilahti, Juottonen, Lang, & Sillanpaa, 1998) studied pure tone discrimination in children with developmental dysphasia (SLI) by using the same 53 Hz difference in simple tones as Korpilahti & Lang (1994). They found that young SLI children exhibited attenuated ERP responses (e.g., reduced amplitudes) to simple tones but normal latencies.

More recently, Uwer, Albrecht, & von Suchodoletz (2002) studied both frequency and duration discrimination in children of 5-10 years with SLI by using simple tones of 1000 Hz vs. 1200 Hz and 175 ms. vs. 100 ms. and synthesized CV syllables differing in place of articulation /bʌ/, /dʌ/, /gʌ/. Language impaired children showed attenuated ERP responses to speech sounds but not to tones. SLI children also made more errors in behavioural discrimination task in both conditions (speech and non-speech) but these did not correlate with the MMN amplitudes. Uwer et al. (2002) concluded that the processing deficit in SLI seems to be specific to speech. This would imply that the auditory impairment is part of the language system rather than general input-processing deficit. In similar fashion, Ors et al. (2002) found normal early sensory ERP components (N1/P2) for tones in SLI but abnormalities in later-stage auditory perceptual processing as indexed by the P3 component that reflects broad recognition and memory-updating processes.

Weber, Hahne, Friedrich, & Friederici (2005) studied 5-month-old German infants atrisk for language impairment. These children who were later (at 12-24 months) found to have lower word production scores showed significantly lower MMN amplitudes for trochaic stress patterns typical for German in CVCV pseudowords, such as /ba:ba/. Friedrich, Weber, & Friederici (2004) in turn, found that even 2-moth-old at-risk for SLI children show delayed auditory ERP responses (MMN) to changes in duration in vowels in CV syllables. They concluded that already infants that are at-risk for language impairments show delays in processing of auditory stimulus change providing support for the view that SLI may be a consequence of abnormal perceptual learning (Tallal et al., 1996).

Shafer, Morr, Datta, Kurtzberg, & Schwartz (2005) reported deficient speech perception abilities as indexed by absent MMN and poor behavioural identification but relatively

good behavioural discrimination of [bIt] vs. [bɛt] syllables in children with SLI. They concluded that SLI children fail to correctly weight the relevant acoustic cues resulting in poorer categorization performance. They also proposed that that the relationship between decision making and early pre-attentive cortical discrimination is not as straight-forward as previously assumed as they failed to find significant correlations between MMN features and behavioural discrimination performance. While discussing their findings, Shafer et al. (2005) contrasted the present data with their earlier studies on late learners of English pointing out that SLI children showed similar pattern as non-native speakers, suggesting that both groups have incorrectly weighted phonological representations.

McArthur & Bishop (2005) reported that poor auditory processing in SLI (as measured both by the early sensory N1-P2-N2 components and by behavioural discrimination tasks) is associated with inability to discriminate different frequencies, and not rapid processing as such. They found that younger children with SLI had poorer discrimination threshold for vowels and tones in addition with abnormal early sensory ERP components. However, when studying older children, McArthur and Bishop (2005) found intact behavioural discrimination in SLI accompanied with abnormal auditory ERPs. To account for these findings, McArthur & Bishop (2005) proposed that in the older group their behavioural measures had hit the ceiling level and they suggested that the validity of behavioural measures of auditory function is questionable after certain age.

Lastly, Rinker et al. (2007) found differences in hemispheric activity for frequency change in simple tones (700/750 Hz) and overall reduction of MMN amplitudes (especially in the later time-windows) in SLI children. Furthermore, to consolidate the differential results reported by many groups, they argued that detecting an auditory input-processing deficit in SLI children could be dependent both on the frequency range in question and on the amount of acoustic deviance between standard and the deviant stimuli.

There is an enlarging body of evidence suggesting that there may be a lower-level inputprocessing deficit in some children with SLI and that the different behavioural methods may not have been sensitive enough to spot this deficit.

However, several inconsistencies remain because some studies report an auditory deficit that is specific to speech whereas other studies claim that the deficit is a general inputprocessing deficit, affecting speech and non-speech processing. Whereas Tallal & Piercy (1973) claim that the primary deficit in SLI is in discriminating rapidly occurring or brief sounds, Shafer et al. (2005) and Sussman (1993) propose that the core problem is in forming phonological representations and allocating attention to relevant aspects of the speech stimuli. Moreover, when looking at the data, some studies report intact early sensory components (N1, P2, N2) but attenuated, delayed or even absent later components (Korpilahti & Lang, 1994; Holopainen et al. 1997; 1998; Uwer et al. 2002) whereas others report only abnormal early sensory processing (Neville et al. 1993; Tonnquist-Uhlen, 1996; McArthur & Bishop, 2005). Furthermore, it is not necessarily straightforward which children, or if all, should show these deficits and how they correlate with different language measures (see Bishop 2007 for a review). If auditory deficits are the core impairment in SLI, we could expect to find strong correlations between auditory tasks and language measures in all groups of children. However, many studies fail to establish such correlation (van der Lely et al., 2004; Rosen et al. 2009; see Rosen, 2003 for a review).

In sum, the linguistic manifestation of SLI is very heterogeneous and not all SLI children show weaknesses in the same areas of language. Some children show particular weaknesses in different components of grammar whereas some children primarily show persistent vocabulary difficulties. Moreover, in some children these deficits are accompanied by impaired auditory processing or short-term memory problems. Awareness of this variation has lead some researchers to attempt to identify a set of subgroups that show uniform linguistic profiles to enable us to better understand the

nature and causes of SLI. In the next Section, I introduce a few proposed subgroups for SLI.

2.3 Sub-grouping SLI

Because the behavioural manifestation of SLI is relatively heterogeneous more homogeneous subgroups for SLI have been put forward. (Rapin & Allen, 1987; Conti-Ramsden, Crutchley, & Botting, 1997; van der Lely, 2005; Novogrodsky & Friedmann, 2006). Heterogeneity among SLI children can be induced by differences in severity of the impairment and by the degree in which production and/or comprehension are affected. The primary aim of sub-grouping SLI is to identify children with similar problems of language performance. In general, there have been three approaches in grouping children with language impairments: clinical, psychometric and linguistic approach.

Based on clinical assessments of child's phonological, syntactic, semantic and pragmatic skills in spontaneous language, Rapin and Allen (1987) suggest a taxonomy of six different subgroups for language disorders: verbal dyspraxia, verbal auditory agnosia (word deafness), phonological programming deficit syndrome, phonologic-syntactic deficit syndrome, lexical-syntactic deficit syndrome and semantic-pragmatic deficit syndrome, of which two or three, namely phonologic-syntactic deficit syndrome, lexical-syntactic deficit syndrome, and semantic-pragmatic deficit are considered to represent SLI given the criteria for SLI provided earlier. Children with phonologic-syntactic deficit syndrome show severe deficits in the production of morpho-syntax and phonology whereas children with lexical-syntactic deficit syndrome have weaknesses mainly in word-finding and immature syntax. Children with semantic-pragmatic disorder, on the other hand, have intact phonology and grammar but abnormal use of language (e.g., difficulties in responding to questions).

From the linguistic perspective, other subgroups have also been put forward, namely the Grammatical (G-) SLI or the Syntactic (S-) SLI studied extensively by van der Lely and

colleagues and Friedemann and colleagues (van der Lely, 1996; van der Lely & Stollwerck, 1997; van der Lely et al. 1998; Friedemann & Novogrodsky, 2004; Marshall & van der Lely, 2007). The G/S-SLI subgroup is a relatively pure domain-specific deficit in grammatical components of language (syntax, morphology and phonology with, arguably, secondary problems in vocabulary). The incidence of G-SLI is around 10-20 % of the SLI children over the age of 9 with normal IQ and no articulatory impairments (van der Lely, 2005). Moreover, since this grammatical subtype seems to be persistent in nature, their language profile remains relatively similar also later in life making it easier to identify G-SLI at a later age. This also allows ruling out the "late talkers" that may look relatively similar at earlier stages of development (van der Lely, 2005).

Children identified with this special grammatical form of SLI are proposed to be impaired in computations underlying hierarchical forms in components of grammar. G-SLI children are reported to have problems in marking syntactic dependencies, whmovement, reversible passives, and pronominal and anaphoric reference and these deficits occur both in perception and production (van der Lely, 1998; van der Lely & Ullman, 2001). Moreover, these deficits are claimed to be independent of memory limitations (van der Lely, 2005; however, see also Montgomery & Evans, 2009).

The G-SLI subtype is identified in children who have already received an SLI diagnosis or alternatively in children who are referred to research groups by educational authorities or by parents. In the latter case, the child's non-verbal intelligence and language scores are assessed. If the child generally fulfils the criteria for SLI, a series of expressive and receptive tests that are specifically designed to identify G-SLI are then administered (see the description of different tests for identifying the G-SLI children in Chapter 5). These tests are designed to probe core aspects of morpho-syntax, that is, they tap areas where children with G-SLI show particular weaknesses, namely verb agreement, tense, reversible passives and pronominal reference (van der Lely, 1996; van

der Lely, 1997; van der Lely, 2000). On each test the child has to produce at least 20% of errors while typically developing children rarely make any.

However, there is still disagreement among SLI researchers over the existence of this purely grammatical impairment in SLI and some suggest only very few in this population meet all the criteria for G-SLI (Bishop, Bright, James, & van der Lely, 2000; Norbury et al., 2002). The advantage of sub-grouping, however, is that it enables researchers to study more uniform population and to see if the phenotypic heterogeneity reflects underlying biological heterogeneity.

In sum, the behavioural manifestation of SLI is very heterogeneous. However, some studies have tried to approach this heterogeneity by sub-grouping children with SLI into more homogenous groups. The argument for sub-grouping is that if we assume that the phenotypic variation is caused by a single core deficit, we need a plausible explanation why the same deficit causes such a wide variety of linguistic and non-linguistic problems —and so far, there has been none. In the next Section, I introduce some suggested causes of SLI.

2.4 Causes of SLI

Philosophers and cognitive scientists in general have long been arguing about the general architecture of the human mind and the role of experience in forming the mind (Chomsky, 1976; Fodor, 1983; Elman et al. 1996). Investigating the causes of a developmental disorder that appears to be specific to language without any major impact on other cognitive domains fits particularly well into this discussion.

The underlying causes of the specific language impairment can be approached from two different levels: the biological and cognitive level causes. At the biological level, we are interested in identifying language-related genes and possible risk factors leading to these impairments. At the cognitive level, on the other hand, we are interested in identifying the underlying and accompanying cognitive deficits in SLI.

2.4.1 The biological causes of SLI

Several studies have proposed that the development of specific language impairment is under genetic influence (for a review, see Bishop, 2009). Evidence for genetic factor in SLI comes from studies evidencing increased familial incidence of SLI, higher male-to-female ratios in SLI and most importantly from twin studies (Stromswold, 2001).

By observing twins, it is possible to hypothesize that, if genes are important in causing SLI, monozygotic twins (MZ) should resemble each other more closely than dizygotic twins (DZ) because MZ twins are genetically identical, sharing all their genes whereas DZ share on average half of their genes, that is, only 50% alleles in common.

In fact, Lewis & Thompson (1992), Bishop, North, & Donlan (1995) and Tomblin & Buckwalter (1998) all reported a significantly higher concordance rate for SLI for identical than in non-identical twins providing strong evidence that genes are involved in causing language disorders. The exact genetic mechanisms, however, underlying the language impairments are still poorly understood.

In one British family (the KE family) (Gopnik & Crago, 1991), a dominant mutation in a single gene on chromosome 7 encoding the transcription factor FOXP2 is associated with severe familial speech and language disorders (Lai, Fisher, Hurst, Vargha-Khadem, & Monaco, 2001). These findings elicited a great deal of research on the causes of language impairment and on general mechanisms guiding language development. Despite the intense research, however, this deficit in a single gene has not been systematically found across SLI population.

A disorder with a genetic basis can constitute a trait attributable to a single gene or to multiple factors that are influenced by combinations of environmental factors and multiple genes. Recent studies have established a linkage between language functions and several separate loci of chromosomes 2, 13, 16 and 19 where the locus on chromosome 16 is associated with performance on a non-word repetition task and

chromosome 19 is linked with expressive language (O'Brien, Zhang, Nishimura, Tomblin, & Murray, 2003). A general consensus is that several factors influence language learning and even if a genetic component can lead to a language disorder, an environmental component may also be necessary (O'Brien et al., 2003). However, no one environmental component has been identified as yet.

2.4.2 The cognitive causes of SLI

At the cognitive level, there are two major perspectives as to the causes of SLI. The domain-specific perspective, or the linguistic perspective, claims that the deficit in SLI is specific to some core components of languages, such as grammar. The domain-general approach, however, proposes that the underlying (or core) deficit in SLI is in lower-level input-processing which interferes with the acquisition of other cognitive skills.

2.4.2.1 The domain-specific account

The domain-specific accounts usually aim at explaining the linguistic deficit observed in SLI in the framework of the linguistic theory. According to this view, it is generally assumed that the impairment in SLI is at the level of underlying linguistic mechanisms, for example, representations and that this is reflected in different sub-systems of language (syntax, morphology, phonology, semantics). Furthermore, the linguistic perspectives propose that there is a specific linguistic core deficit that can occur without any general lower-level deficit.

The majority of domain-specific accounts propose that there is a deficit in the syntactic features that are either missing, underspecified or develop later, as proposed by the Missing Agreement Hypothesis (Clahsen, 1989; Clahsen, Rothweiler, Woest, & Marcus, 1992), Missing Feature Hypothesis (Gopnik, 1990) and Extended Optional Infinitive Hypothesis (EOI, Rice & Wexler, 1996).

According to Missing Agreement hypothesis (Clahsen, 1989), SLI children have particular problems in establishing structural relationship of agreement affecting, among other things person and number agreement between verb inflections, auxiliaries and copula forms and the subject. The Missing Agreement hypothesis proposes that the locus of linguistic problem in SLI lies primarily in morpho-syntax, that is, mainly the features that enter into agreement relations (i.e., semantically redundant features) are impaired in SLI and, therefore, those features that do not involve agreement should not be problematic. To account for the findings that SLI children do occasionally produce correct forms, the Missing Agreement hypothesis proposed that these forms are memorized as separate lexical items in SLI. The Missing Agreement hypothesis was originally based on German data but it also explains some of the difficulties found in the English data (e.g., verb inflections *He walks*). However, it fails to account for the relatively frequent deficits unrelated to agreement, such as the regular past tense formation.

The Missing Features hypothesis (Gopnik, 1990) proposes that children with SLI are impaired in acquiring the implicit rules to mark tense, number and person and, as a consequence, these semantic-syntactic features of tense/number/person are missing in the child's underlying grammar. According to the Missing Feature hypothesis, to compensate for this inability to access the implicit rules, SLI children can either memorize inflected forms or to employ explicit (metalinguistic) rules taught to them accounting for some of the grammatical errors found in the SLI data. However, both the Missing Agreement Hypothesis and the Missing Feature Hypothesis focus mainly on explaining the deficits in certain aspects of morpho-syntax and do not provide a particularly plausible explanation that covers other weaknesses found in SLI.

The Extended Optional Infinitive hypothesis (EOI) (Rice & Wexler, 1996) is based on typical language acquisition and accounts specifically for tense marking difficulties found in SLI. According to EOI, at the so called Optional Infinitive (OI) stage, children fail to consistently mark finiteness in main clauses which require it. In other words, they

have marked problems in paste tense -ed and in 3^{rd} person singular -s, in copula and auxiliary verbs. However, in SLI this optional stage of marking finiteness is markedly extended. Unfortunately, the EOI account is also limited in scope since it is centred on one of the core problems of SLI, that is, on tense and agreement marking deficits and fails to account for the heterogeneous nature of SLI.

The Computational Grammatical Complexity (CGC) account of van der Lely and her colleagues (van der Lely, Rosen, & McClelland, 1998; van der Lely & Battell, 2003; van der Lely, 2005; Marshall & van der Lely, 2006) is an extension of the Representational Deficit of Dependent Relations (RDDR) that was primarily developed to account for a subgroup of SLI children, namely Grammatical SLI (G-SLI) (van der Lely, 1996). Van der Lely and colleagues claim that the behavioural linguistic deficit observed in these children lies in the abstract representational level and in the underlying computations of complex forms in syntax as well as in the morphological and phonological domains (van der Lely & Ullman, 2001; van der Lely et al., 2004; van der Lely, 2005; Marshall & van der Lely, 2007).

The core deficit according to CGC is in hierarchical complexities. This deficit manifests in different ways in the computational grammatical language components. With respect to syntax, the CGC hypothesis predicts that all syntactic structures requiring dependencies involving "movement" (e.g., passive sentences and wh –questions) are impaired. Morphological deficits in SLI, in turn, stem from morphological complexity e.g., an impaired computation of the suffixation rule to form past tense in English. This abstract rule (add –ed) creates a complex hierarchical branching structure that is preferentially stored in SLI, as opposed to forming past tense by applying a rule as in normal development. Phonological deficits in SLI are imposed by structural complexity (in terms of markedness) at the syllable and metrical levels. In other words, the errors observed in SLI are not only simple cluster reductions or simplifications and, therefore, due to processing limitation. The CGC account proposes that in G-SLI, only the unmarked parameter values (simple onsets, open rhyme, vocalic word ending) are

available to them. For example, when the prosodic complexity of a word increases (e.g., it contains consonant clusters) it becomes more difficult for children with SLI. Moreover, Yung Song, Sundara and Demuth (2009) investigated impact of phonological complexity on the production of 3rd person singular morpheme –s in typically developing children. They reported that, first of all, more accurate production of –s is observed in phonologically simple contexts (such as in *sees*) compared to phonologically complex contexts (such as in *needs*). Secondly, they reported that more accurate production of 3rd person singular morpheme –s occurred utterance-finally as to utterance-medially suggesting that phonological complexity and positional effects should be taken into account for when investigating the development of phonology and morphology.

CGC hypothesis specifically predicts deficits in processing hierarchical complex structures of components of grammar and that all these components can independently affect the sentence processing. CGC account also predicts that different sub-systems of grammar can be selectively impaired and can affect the functioning of the remaining cognitive systems, thus accounting for the differential phenotypes observed in SLI. In other words, a core deficit in some components of grammar does not rule out other co-occurring or secondary language impairments but it still entails that there is no consistent causal relationship between auditory processing and grammatical abilities (van der Lely, 2005). However, to what degree the CGC account generalizes to the SLI population at large is an open question.

2.4.2.2 The domain-general account

The domain-general perspective proposes that the underlying cause in SLI is not in linguistic knowledge but a general input-processing deficit or memory limitation. The domain-general view, therefore, claims that, by definition, selective impairments cannot exist and language impairments are accompanied by a lower-level input-processing deficit (Elman et al., 1996; Karmiloff-Smith, 1998). According to the domain-general perspective, domain-specificity is an emergent property of the system and an originally

domain-general system only becomes domain-specific after it is repeatedly used to process certain type of input. This account is in opposition to domain-specific perspective in that is proposes that the underlying deficit in SLI can be traced back to (a set of) lower level impairments and is not a disorder that uniquely and specifically affects the higher cognitive functions.

The Generalized Slow Processing Deficit hypothesis (Kail, 1994) proposes that the deficit in SLI lies in reduced processing speed predicting slower response rates in SLI across a wide range of tasks. This account suggests that the grammatical impairments in SLI are caused by general processing limitations and, therefore, are secondary to the underlying more general cognitive deficit. According to Kail (1994) these processing limitations are not restricted to processing the auditory signal: slower reaction times are also found in non-linguistic tasks such as mental rotation. Adopting this view, Miller and colleagues (Miller, Kail, Leonard, & Tomblin, 2001) measured reaction times in both linguistic and non-linguistic stimuli in children with SLI. They reported that SLI children had significantly slower reaction times in linguistic tasks (e.g., rhyme judgement) and in non-linguistic tasks (e.g., simple RT, visual search) and concluded that SLI children (or at least *some* children with SLI) are generally slower in all input-processing than typically developing children providing support for the slow processing model.

Another influential account of SLI, the Auditory Processing Deficit (APD, also known as the rapid auditory temporal processing deficit, RATP) hypothesis (Tallal & Piercy, 1973), claims that there is an impairment of processing rapid temporal changes (such as formant transitions) that are typical to speech. According to RATP, acoustically less salient contrasts, that is, segments in unstressed positions or of short duration are particularly affected.

In the majority of studies, Tallal and her colleagues (Tallal & Piercy, 1973; Tallal et al., 1980) compared the performance of language impaired and typically developing

children in behavioural discrimination tasks with verbal and non-verbal stimuli (of different durations) occurring at different presentation rates. They concluded that the poor discrimination ability is the primary deficit in SLI, and that all other linguistic and non-linguistic impairments were secondary to this inability to discriminate auditory stimuli. Tallal's conclusion was that children with SLI have special difficulties with speech stimuli of brief duration or containing rapid spectrotemporal changes. This problem is particularly evident in tasks such as $[b_{\Lambda}] - [d_{\Lambda}]$ discrimination where the main difference between these two syllables is less than 50 ms of initial formant transition.

According to the RATP hypothesis, people identified as slow processors, or those who have problems in temporal aspects of input-processing have difficulty in accessing the rapid auditory information despite normal hearing, and that is assumed to be the major factor contributing to the language problems in many different disorders such as SLI or dyslexia. These difficulties are found both in speech and in non-speech stimuli (Tallal & Piercy, 1973; Tallal et al., 1980) and when the language impaired children are trained with acoustically modified material (e.g., longer formant transition durations, longer intervals between successive sounds) their processing skills are significantly improved (Cohen et al., 2005; Tallal et al. 1996, however, see McArthur, Ellis, Atkinson, & Coltheart, 2008; Gillam et al., 2008 for opposite results, and Rosen, 2003 for general discussion). In short, Tallal's view attributes the language problems to difficulties in discriminating brief sounds that impede the learning of certain details of language, such as forming reliable phonological representations in childhood.

The work of Tallal and her colleagues has triggered a great deal of research on the role of auditory processing in developmental language disorders. However, why similar input-processing problems would result in such a different linguistic manifestations, as is the case in SLI and dyslexia, is not yet clear. Furthermore, it is well acknowledged that only a subgroup of SLI children are likely to reliably show auditory processing problems (van der Lely, Rosen, & Adlard, 2004; McArthur & Bishop, 2005) and these

input processing deficits are not necessarily restricted to brief and rapid stimuli (Corriveau, Pasquini, & Goswami, 2007). Furthermore, this input-processing problem is not able to fully explain the wide range of syntactic problems found in SLI (Norbury, Bishop, & Briscoe, 2002).

Gathercole & Baddeley (1990), in turn, propose that SLI is a deficit in phonological short-term memory (PSTM). This model proposes that in children with SLI their core difficulties are in storing and holding information in the short-term phonological storage within the phonological loop of working memory. This view is supported by an extensive body of data where children with SLI show problems in recalling serial lists of real words and non-words with increasing number of syllables (e.g., in the Children's Test of Nonword Repetition, CNRep, Gathercole & Baddeley, 1996). These data systematically show that the performance of language impaired children is markedly poorer in longer non-words. Gathercole & Baddeley (1990) hypothesise that the shortterm memory system intervenes with their ability to learn novel words and morphemes due to insufficient temporary representations that form the basis for more permanent representations (Gathercole & Baddeley, 1990; 1996). They claim that the deficit in forming temporary representations may lay on one of the three following levels. Firstly, the system may suffer from imprecise initial segmental analysis that leads to less salient phonological representations. Alternatively, the phonological traces may decay too rapidly or, thirdly, the phonological store can be limited in that it is capable of storing fewer items. A deficit in either of these levels would then have an impact on grammar and lexical learning.

Taking a different perspective, Ullman & Pierpont (2006) propose that SLI can be explained by abnormal development of brain structures underlying the procedural memory system. The procedural system is memory storage of skills and procedures (such as how to ride a bike) and of non-associative, or rule-based, learning. The Procedural Deficit Hypothesis (PDH) proposes that abnormalities within this system result in grammatical and lexical deficits and impaired non-linguistic functions to

different degree depending on which structures are affected. The PDH predicts that in the majority of children with SLI we should find, firstly, abnormalities in brain structures (specifically in caudate nucleus and in Broca's area), secondly, deficits in those linguistic functions that depend on the procedural memory system (rule-based processing of grammar, lexical retrieval) and thirdly, impaired non-linguistic functions. In other words, the PDH claims that the impairments in syntax, morphology and phonology in SLI are a direct consequence of structural abnormalities affecting the procedural memory system; therefore, it makes a direct prediction that these abnormalities should always co-occur with the behavioural deficits. At present, we need more structural and functional brain imaging data on SLI to evaluate the validity of this account.

However, it is still very much an open question to what degrees these different domaingeneral accounts are able to explain the variety of linguistic deficits found in SLI. The hypothesis is that the lower-level processing deficit causes, for example, unreliable phonological representations thereby affecting other aspects of language learning. However, this issue is highly controversial because the linguistic realization of SLI is very heterogeneous and systematic correlations between language measures and auditory measures are rarely found. A more thorough specification of how a slow processing speed or problem with rapid temporal changes could affect, for example, comprehension and production of past tense or agreement is needed. Moreover, only a subgroup of SLI children seem to exhibit an auditory processing deficit and, more importantly, even some typically developing children show problems in these auditory tasks despite their normal linguistic abilities (Rosen, 2003).

However, it may well be that an initial auditory processing problem may not be detectable later in life due to different compensation mechanisms. Thus further research to disentangle the role of auditory processing skills in language acquisition and normal and atypical language processing is required.

2.6 Summary

There is a growing body of behavioural evidence that some SLI children - but not necessarily all - exhibit an auditory processing deficit. Moreover, recent electrophysiological studies have shown that auditory processing deficits could exist in some children with SLI. However, the basic questions that any 'input-processing model' needs to account for are: Is there a general input-processing deficit underlying SLI that can be reliably detected in *all* children of SLI? If not, then which SLI children then show this problem and what is their phenotype? What is the relationship between an auditory perceptual deficit and different linguistic measures and memory? Is the deficit present in adulthood or does brain plasticity and compensation camouflage this deficit? And lastly, is this deficit specific to speech, and more interestingly, to specific features of speech? In order to provide an answer to these questions, we need to look at auditory processing in typically developing children and children with language impairments and see how these processes correlate with different language measures and how they relate to the functioning of brain.

3. Developmental dyslexia

3.1 Introduction

Learning to read and write is a complex process that, in alphabetic writing systems, involves abstracting the alphabetic mapping between letters and sounds (i.e., 'grapheme to phoneme mapping'). When entering school, children usually acquire this skill at a relative ease. Some children, however, fail to learn to read and write at a normal rate without any apparent reason (i.e., despite appropriate training opportunities and normal intelligence) and these developmental literacy impairments usually have a lifelong persistence. These children who fail in acquiring the literacy skills at normal rate are generally referred to as 'reading disabled', 'reading impaired', children with 'specific reading disability' (SRD) or children with 'developmental dyslexia' (the latter two are used interchangeably in the current thesis). It has been estimated that developmental reading disability affects approximately 3-10% of the population. The underlying causes of this reading disability, however, remain largely unknown (see Snowling, 2000).

Developmental dyslexia is traditionally defined as a discrepancy between child's reading ability and intelligence despite normal opportunities to learn to read (Vellutino, 1979; Snowling, 2000). To diagnose dyslexia child's performance is usually assessed on various standardised psychometric tests of literacy and evaluated against what is expected on the basis of a test of IQ or on the basis of their chronological age (i.e., the estimation of their actual 'reading age'). This is not, however, a straightforward criterion. First of all, the choice of appropriate standardised reading test and method for assessing IQ (verbal, non-verbal or full scale IQ) are crucial issues in determining which children are classified as dyslexics (Stanovich, 1986). Moreover, this psychometric definition is purely behavioural and based on *exclusionary* criterion on child's reading attainment and what is expected on the basis of their IQ or age (Snowling, 2000; Bishop & Snowling, 2004). Therefore, in recent years, there has been considerable debate

concerning this age-/intelligence-based exclusionary criterion (especially for clinical use) and many researchers argue that the discrepancy criteria should be complemented with some *positive* diagnostic markers (Snowling, 2000; Bishop & Snowling, 2004). However, defining reliable positive diagnostic markers for dyslexia is not straightforward because there does not appear to be simple behavioural manifestations for dyslexia. Overall, dyslexic children have been reported to encounter heterogeneous problems in printed word recognition and spelling. They also show deficits in different areas of phonological processing (repeating non-words, poor phonological awareness) and sensory problems (in visual, auditory and tactile domains) (Ramus et al., 2003). The underlying cause(s) of these deficits, however, remain largely unknown. The suggested causes of dyslexia are reviewed in the following section.

3.2 Understanding dyslexia

Due to the heterogeneous nature of behavioural and cognitive manifestations, dyslexia has been characterized via three separate but interacting levels of description: **biological**, **cognitive** and **behavioural** levels that represent causal links between brain and behaviour. In other words, the central aims in dyslexia research are to understand the *biological* bases of this disorder (i.e., its genetic and neurological basis), the *cognitive* bases of the disorder (i.e., to identify cognitive deficits associated with dyslexia) and to describe the *behavioural* signs and symptoms of dyslexia and how these change as a function of age.

3.2.1 Biological level

It is well acknowledged that reading problems tend to run in families suggesting that dyslexia is an inherited condition under genetic control. However, in addition to proportion of their genes, families also share similar environments. Therefore, the most convincing evidence for genetic origins for dyslexia comes from studies on concordance rates in genetically identical vs. genetically non-identical [i.e., monozygotic (MZ) vs. dizygotic (DZ)] twins (see Chapter 2). As is the case in SLI, reported concordance rates of dyslexia in MZ twins are higher than in DZ twins implying that genetic factors are

involved, the estimated concordance rates for MZ and DZ twins being approximately 68% and 38%, respectively (DeFries & Alarcon, 1996; Fisher et al., 1999). Moreover, based on a study of Finnish familial dyslexics, Taipale and colleagues (2003) proposed a first candidate gene for dyslexia (*DYX1C1* on chromosome 15q21) and identified two sequence changes in *DYX1C1* that could be associated with dyslexia. The *DYX1C1* gene has been proposed to be involved in neuronal migration, and the deletion found in one dyslexic family may disrupt its function (Ramus, 2004).

Early anatomical evidence for a neurological basis for dyslexia was derived from postmortem and brain imaging studies by comparing dyslexic and control brains. These studies revealed abnormalities at medial/lateral geniculate nuclei (M/LGN), primary visual and auditory cortices, the cerebellum and corpus callosum (see Habib, 2000; Eckert, 2004 for reviews). These focal abnormalities include cytoarchitectonic anomalies (e.g., ectopias and microgyri) at the perisylvian cortex and in thalamus (LGN and MGN) of the dyslexic brains. These anatomical findings have been linked to the phonological and oral language deficits associated with dyslexia as well as to the visual and auditory deficits sometimes found in dyslexics (see the magnocellular theory of dyslexia in section 3.2.2) (Ramus, 2004; Eckert, 2004). Most attention, however, has been directed to planum temporale that is situated posterior to Heschl's gyrus and contains auditory association areas. The results, however, have been heterogeneous, several structural imaging studies reporting symmetrical plana temporala in dyslexic brains and several studies failing to establish this lack of asymmetry (see Habib, 2000, Eckert, 2004 and Leonard & Eckert, 2008 for reviews). According to Eckert (2004) the most consistent differences between dyslexic and control brains have been found in inferior frontal gyrus, temporal-parietal region, the medial occipital lobe and the cerebellar anterior and posterior lobes, each area contributing differently to the type of deficit observed at the cognitive and behavioural levels.

On the basis of the genetic evidence and a large body of brain imaging data, Ramus (2004) postulated 'a neurobiological model' for dyslexia accommodating genetic,

structural/functional brain imaging studies, animal models of brain function and behavioural findings into a new neurobiological framework. Overall, according to this framework, there are multiple ways to become dyslexic. Ramus (2004), however, argues that the primary cause(s) of dyslexia are genetically driven cortical abnormalities that are sometimes accompanied by non-genetic factors (e.g., foetal hormonal conditions). These two factors contribute to the various cognitive and behavioural manifestations of dyslexia.

Evaluating and incorporating results from various anatomical studies on dyslexia is not straightforward. Firstly, acquiring detailed clinical and behavioural histories of affected individuals in earlier post-mortem studies can be challenging and sometimes comorbidity issues cannot be reliably ruled out. Secondly, the anatomical patterns of results tend to vary from one study to another. This variation could, in principle, reflect the differential inclusion criteria for dyslexics employed in different studies or the cognitive/behavioural heterogeneity within dyslexia. From this perspective, Eckert (2004) argues that comprehensive neurobiological understanding of dyslexia will largely depend on studies conducted on homogeneous perceptual, cognitive and genetic backgrounds.

To summarize, it is well established that reading is a complex task that involves multiple neural networks and, therefore, dyslexia is more likely to stem from heterogeneous structural abnormalities rather than constituting a single anatomical marker. Overall, dyslexics' brains are found to be more variable than controls' brains which may reflect the underlying genetic variability as well as the well-documented behavioural heterogeneity observed in dyslexia.

3.2.2 Cognitive level

At the cognitive level, there are two influential approaches to dyslexia: the magnocellular theory of dyslexia and the phonological theory of dyslexia. The magnocellular theory of dyslexia emphasizes the role of sensory impairments (such as

auditory, visual and motor) in causing dyslexia and claims that most reading problems have a fundamental sensorimotor cause (Stein & Walsh, 1997: Stein, 2001). The phonological theory, on the other hand, postulates that literacy deficits found in dyslexia are caused by underlying impairment in cognitive function, for example, in phonological processing.

3.2.2.1 Magnocellular theory

According to the magnocellular theory, the development of the thalamocortical magnocellular system (or more precisely the magnocellular layers of the LGN) is impaired in dyslexia resulting in impairments in tasks that engage this system. In the visual pathway the magnocellular system is important for direction of visual attention and, therefore, can have an impact on orthographic skills. The magnocellular system is specialized in, for example, detecting moving and rapidly changing stimuli and is sensitive to low spatial (=organizing of visual information in space) and high temporal (=capture of visual information over a brief period of time) frequency information picked up from peripheral vision (approximately 5-6 letters to the right of fixation). Thus its impairments are thought to affect reading. However, magnocells are found in all sensory and motor systems (e.g., in the LGN, in the MGN on the auditory pathway and in cerebellum) as well as in areas guiding attention (e.g., parietal cortex). Therefore, in recent years, this visual impairment theory for dyslexia has been modified a great deal to account also for the auditory and motor deficits found in dyslexia (Stein, 2001; Ramus, 2003; 2004).

In its current form, the magnocellular theory of dyslexia postulates a direct causal pathway between, for example, MGN dysfunction and auditory deficits. At the behavioural and cognitive levels this dysfunction reflect as deficits in auditory temporal processing as well as in phonological processing both of which are often reported in conjunction with dyslexia. The rationale for this model is as follows: speech sounds consist of rapid changes of frequency and amplitude over time. Accurate speech perception and successful phonological development, therefore, requires an intact

auditory system that is able to identify these transient segments in the highly variable speech signal (see, e.g., Tallal, 1980).

An auditory processing deficit model for dyslexia has, in fact, been put forward independently of the magnocellular theory for dyslexia (Tallal, 1980; Tallal et al. 1996). This auditory model (the 'rapid auditory temporal processing theory', RATP) argues that, like children with SLI, dyslexics show poor performance on a number of auditory tasks that tax rapid temporal processing in the auditory domain (e.g., in gap-detection, frequency discrimination, temporal order judgement and backward masking; for a review see McArthur & Bishop, 2001). According to this model, a failure to correctly represent short sounds and fast transitions would cause further difficulties in processing segments where these short and rapid acoustic events are cues to phonemic contrasts (such as [bʌ] and [dʌ]). In other words, this model assumes a direct causal link between poor auditory processing and phonological problems. Furthermore, the RATP model claims to account for the cognitive deficits reported in dyslexia *and* SLI (Tallal, 1980; see Chapter 2 for the description of experimental tasks).

The RATP model has evoked intensive research during the past decades. The accuracy of auditory perception (e.g., detection thresholds) in dyslexic individuals has been assessed, for example, with frequency and amplitude modulated (FM and AM) tones that contain rapid changes in frequency/amplitude thought to be crucial to successful speech perception and normal development of literacy skills (see Talcott et al. 2000).

Witton and colleagues (Witton et al., 1998; Witton, Stein, Stoodley, Rosner, & Talcott, 2002) reported that dyslexic individuals are less sensitive than controls to particular rates of frequency and amplitude modulation (2-Hz and 40-Hz FM and 20-Hz AM) and this (in-)sensitivity was significantly correlated with their phonological abilities (e.g., non-word reading). Ramus and colleagues (2003), in turn, investigated auditory processing in dyslexia by adopting the FM stimuli used in the Talcott et al. (2000) study. Like in other studies, Ramus et al. (2003) also found a significant group effect at 2-Hz FM but not at a

240-Hz FM. They concluded, however, that this group effect was due to only a subgroup of dyslexics performing poorly in this task. Moreover, Tallal et al. (1996) reported that training language and/or reading impaired children with acoustically modified (e.g., amplified and temporally extended) material leads to significant improvements in temporal processing, phonological processing and language comprehension.

The implications of these auditory deficits, however, are not well established (see also below). Boets, Ghesquière, van Wieringen, & Wouters (2007) tested pre-school children at (genetic) high- and low-risk (HR and LR) for dyslexia on a set of auditory tasks. They found that these auditory tasks could not reliably differentiate HR and LR groups at a later stage -neither at group nor at individual levels- despite the FM detection and the tone-in-noise detection being significantly related to phonological awareness measures.

In short, the auditory theory postulates that the auditory deficit is the direct cause of the phonological deficit and hence the difficulty of learning to read. In other words, the RAPT model predicts that dyslexic individuals should be impaired both in non-speech and speech processing. However, as already mentioned in Chapter 2, behavioural measures may not always be sensitive enough to detect subtle input-processing differences. Therefore, in addition to the wide range of behavioural experiments, some recent electrophysiological experiments have tested the auditory processing deficit model in dyslexia (see Kujala & Näätänen, 2001; Bishop, 2007, for reviews).

Schulte-Körne and colleagues investigated the non-speech and speech processing in dyslexic children and adults in three subsequent studies (Schulte-Körne, Deimel, Bartling, & Remschmidt, 1998; Schulte-Körne, Deimel, Bartling, & Remschmidt, 1999a; Schulte-Körne, Deimel, Bartling, & Remschmidt, 2001). They investigated non-speech auditory discrimination (the MMN component of auditory ERPs) by using tones differing in frequency (1000Hz/1050Hz and 2200Hz/2640Hz) and speech processing by using CV syllables ([dʌ]-[bʌ] and [dʌ]-[gʌ]). Moreover, they used auditory patterns composed of tones with different pitch. Overall, Schulte-Körne et al. (1998, 1999 and

2001) reported attenuated MMN amplitudes for speech and auditory tone patterns but normal MMN amplitudes for simple tones. On the basis of these results, they concluded that the deficits in the tone pattern and speech stimuli processing in dyslexics may arise from impairments in processing 'rapid' temporal patterns as suggested by the RATP model of dyslexia. Other subsequent studies have also reported significant differences between dyslexics (or at-risk populations) and controls by using complex tone patterns, speech segments, simple tones of different frequencies, tones differing in the SOA and backward masking (Hugdahl et al., 1998; Baldeweg, Richardson, Watkins, Foale, & Gruzelier, 1999; Kujala et al., 2000; Guttorm et al., 2005; Kujala, Lovio, Lepistö, Laasonen, & Näätänen, 2006; van Leeuwen et al., 2008).

The RATP theory has also received a huge amount of criticism. First of all, not all studies have succeeded in replicating the behavioural and electrophysiological findings reported above (see McArthur & Bishop, 2001; Rosen, 2003; Bishop, 2007 for reviews).

For example, Mody, Studdert-Kennedy, & Brady (1997) investigated the discrimination of CV syllables that contain rapid formant transitions ([bʌ]-[dʌ]) and their non-speech analogues (F₂ and F₃ trajectories) in dyslexic and control groups. Interestingly, in contrast with the RATP model, dyslexics were unimpaired in the non-speech discrimination but differed from their controls in the speech discrimination. On the basis of these results Mody et al. (1997) concluded that the core deficit in dyslexia is specific to speech and not necessarily causally related to general auditory processing. Similar findings were also reported by Rosen & Manganari (2001). Moreover, failures in replicating the impaired performance have been reported in several behavioural studies (e.g., gap-detection and backward masking: Bishop et al. 1999; Schulte-Körne, Deimel, Bartling, & Remschmidt, 1999b; Ramus et al. 2003;) as well as in electrophysiological studies (Lachmann, Berti, Kujala, & Schröger, 2005; Paul, Bott, Heim, Wienbruch, & Elbert, 2006; Alonso-Búa, Díaz, & Ferraces, 2006). Secondly, the validity of auditory processing measures used to assess auditory temporal processing abilities among dyslexics and controls have been frequently questioned. The main arguments are the

weak correlations between different paradigms aimed at tapping the same underlying deficit, the selection of controls tasks, the effect of task-related (extraneous) factors such as attention and memory (see Rosen & Manganari, 2001; McArtur & Bishop, 2001; Rosen, 2003). Moreover, different auditory and speech processing measures do not necessarily correlate with each other (Rosen & Manganari, 2001; Boets et al. 2006; Boets et al., 2007) or with the higher-level language abilities (Rosen et al. 2009) suggesting, therefore, that auditory/speech processing as such is an unlikely candidate to cause or maintain the literacy/language deficits.

To summarize, the magnocellular theory of dyslexia seems to accommodate both the auditory-phonological deficits and the visual-spatial deficits found in conjunction with dyslexia. The original RATP model Tallal (1980), however, made no direct biological claims but it is, nevertheless, nowadays specified within the magnocellular framework (Tallal, Miller, & Fitch, 1993).

A substantial amount of experimental evidence has been presented to back up both the RATP model and the magnocellular theory of dyslexia. In his recent reviews, however, Ramus (2003; 2004) points out that both the magnocellular theory and the RATP model are theoretically and empirically only partially successful. Firstly, the magnocellular theory does not explain why the prevalence of sensory-motor dysfunction is much lower than, for example, the prevalence of phonological impairments in dyslexia (i.e., it fails to account for the absence of sensory impairments). Secondly, there are number of studies that have failed to replicate the previous results of auditory deficits in dyslexia (or found auditory deficits only in subgroups of dyslexics, see e.g., Mody et al. 1997; McArthur & Bishop, 2001; Rosen & Manganari, 2001). Thirdly, there is no consistent evidence that the processing problems would lie in 'rapid' auditory processing as proposed both by the Tallal's model and by the magnocellular framework (Ramus et al. 2003). Lastly, there is no clear evidence that the auditory deficit would predict phonological deficits (Mody et al. 1997; Bishop et al. 1999; Rosen & Manganari, 2001; Ramus et al. 2003). Moreover, studies that investigated auditory training (such as Fast ForWord®, FFW) and reported

that training SLI/dyslexic/typically developing participants, for example, with acoustically modified material improves their auditory and speech perception abilities and literacy/language scores (see e.g., Tallal et al. 1996; Merzenich. et al., 1996; Kujala et al., 2001; Moore, Rosenberg, & Coleman, 2005) were not blind assessments of outcome (e.g., randomized controlled trials, RCT). In fact, Cohen et al. (2004) and Gillam et al. (2008) reported that when conducting a RCT, FFW did not induce greater pretest-to-posttest improvement than any other non-specific training method. In other words, all groups made progress and FFW did not show any additional benefit of intervention.

3.2.2.2 Phonological theory

The phonological theory of dyslexia holds that the most prominent cognitive symptom (phonological deficit) represents the most direct causal pathway while the other symptoms are simply comorbid markers with no causal relationship with the reading disorder. The phonological theory argues that dyslexics have specific impairment in the representation, storage and/or retrieval of speech sounds that hinders learning the grapheme-phoneme associations and impacts their literacy development. However, whereas the proponents of this account all agree on the central and causal role of phonological skills for dyslexia, they do not agree on the exact nature of the phonological deficit (Snowling, 2000; Ramus, 2003; Ramus & Szenkovits, 2008).

Support for the phonological theory comes from evidence that many dyslexics show poor phonological awareness (apparent, e.g., in spoonerism and phoneme deletion tasks), poor verbal short-term memory (digit span, non-word repetition) and slow lexical retrieval (rapid automatised naming). The most commonly accepted hypothesis regarding the nature of phonological deficit in dyslexia is that phonological representations are poorly specified or degraded (i.e., 'noisier' or 'fuzzier'). In other words, the deficit could be a consequence of a basic speech processing problem. Several studies have aimed at investigating this aspect of input-processing in dyslexia, and, again, the results have been heterogeneous. Most studies have employed the categorical

perception (CP) paradigm (see Chapter 4) and investigated the speech sound representations related to various acoustic features (e.g., VOT: [bA]-[phA], place of articulation: [bʌ]-[gʌ]) in dyslexia. Adlard & Hazan (1998) investigated 13 children with specific reading difficulties and their age and reading level matched controls in various auditory and speech perception tasks. They found that the dyslexics did not differ from controls in any of the psychoacoustic measures. However, a subgroup of the dyslexics showed impaired phonemic discrimination (with stop, fricative and nasal consonants) suggesting that some individuals with dyslexia may have poorly specified phonological categories that does not necessarily derive from lower-level auditory problems. Moreover, Blomert & Mitterer (2004) investigated categorisation of natural (nonsynthetic) $[t\Lambda]$ - $[k\Lambda]$ and synthetic $[b\Lambda]$ - $[d\Lambda]$ continua in dyslexic and controls. They found that, first of all, on the synthetic continuum, the dyslexic group gave less consistent responses at the category endpoints than their typically developing peers. On the natural continuum, there were no overall group differences and natural stimuli were harder to categorize by both groups. On the basis of these results, Blomert & Mitterer (2004) argued that instead of being impaired in speech perception, dyslexics are less able to apply their phonological representations based on natural speech to novel synthetic stimuli.

Serniclaes and colleagues (Serniclaes, Sprenger-Charolles, Carre, & Demonet, 2001; Serniclaes, Heghe, Mousty, Carre, & Sprenger-Charolles, 2004) postulated a hypothesis that the core problem in dyslexia is, indeed, poorly specified phonological categories and this is due to those categories containing irrelevant allophonic detail (see Chapter 4). Serniclaes and his colleagues found that individuals with dyslexia are worse than controls in discriminating speech sounds that crossed the category boundary (e.g., in VOT in French: [b]-[p^h]). However, they also noticed that dyslexics are actually *better* than controls in distinguishing sounds within these categories by showing increased discrimination in the VOT continuum at a point irrelevant for their native language phonological inventory (i.e., [b]-[p]-[p^h]). On the basis of these data, Serniclaes and

colleagues argue that dyslexics are more sensitive to acoustic-phonetic variation caused by the surrounding speech sounds (e.g., phonetic detail that is irrelevant for lexical processing) and, therefore, less able to abstract the relevant category information than their controls. This perceptual 'hyper-sensitivity' can lead to problems in creating stable grapheme-phoneme mapping principles and thus lead to literacy deficits.

In order to test several theories of dyslexia in one study, Ramus et al. (2003) presented a battery of psychometric, phonological, auditory, visual and cerebellar tests to high achieving dyslexic university students and their controls. Within each domain Ramus et al. (2003) tested the participants with various subtests that have elicited consistent differences between dyslexics and controls in previous studies (e.g., backward and simultaneous masking, phonemic categorization, FM detection, TOJ-task, automated picture naming, spoonerism, non-word repetition). Out of these subtests, Ramus et al. (2003) created summary variables for 'phonology' and 'auditory' and out of auditory measures summary variables for 'rapid'/'slow' and 'speech'/non-speech'. The results showed that a significant proportion of dyslexics were impaired in the auditory domain. However, all auditory measures showed a rather heterogeneous pattern. Moreover, there was a clear correlation between the 'auditory' and 'phonology' variables but this correlation was only apparent in controls. On the basis of these data, Ramus et al. (2003) concluded that poor auditory processing can entail poor phonological processing but not necessarily vice versa, that is, phonological deficit can exist without auditory problems. Most importantly, Ramus et al. (2003) did not find any significant differences between auditory summary variables of 'rapid' and 'slow' or 'speech' and 'non-speech'.

Auditory and speech perception abilities of dyslexic individuals have attracted a huge amount of interest and research. As mentioned before, the results, however, have been heterogeneous and equivocal. Overall, many of these studies reported above agree on that auditory problems often do (co-)occur with dyslexia. The phonological theory, however, argues that these auditory deficits are not necessarily causally related to

dyslexia, that is, the phonological deficit could exist without apparent auditory problems (see Rosen, 2003; Ramus, 2003; 2004).

Nonetheless, the question still remains: What is the nature of the phonological deficit? Ramus & Szenkovits (2008) reviewed the dyslexia literature in trying to tackle this question and argued that, in fact, the data show that the phonological representations are normal and the 'phonological grammar' (i.e., development of language-specific phonological processes such as voicing assimilation in French) has developed normally in dyslexia. However, where dyslexics fail is tasks that load short-term memory (e.g., spoonerisms) or require speed (e.g., rapid naming) tentatively suggesting that the core problem may lie in phonological access or in verbal short term memory rather than at the representational level.

To summarize, the phonological theory postulates that dyslexics have a specific impairment in the representation, storage and/retrieval of speech sounds. Furthermore, the majority of researchers agree that phonological processing is at the core of impairments in dyslexia. The evidence, however, is not conclusive as to the exact nature of the deficit(s).

3.2.3 Behavioural level

Figure 3.1 summarizes the main cognitive and behavioural deficits in visual, auditory, motor and 'other' domains introduced in previous sections. Dyslexia is defined as deficit in acquiring literacy skills (e.g., reading and writing) at a level expected on the basis of the IQ (Snowling, 2000). Generally, dyslexics make more spelling and reading errors and/or are slower than their typically developing peers in reading and writing. However, the manifestation of reading difficulties can vary according to the writing system and the transparency of the writing system. For example, in languages such as English, the phoneme-grapheme correspondences are fairly complex and arbitrary resulting in persistent problems in reading and spelling accuracy in dyslexic individuals. In languages with more transparent writing systems (such as Finnish and Spanish), on

the other hand, the literacy problems usually manifest in spelling and reading speed. However, despite these cross-linguistic surface-level differences, the factors governing phonological development and the primary deficit(s) underlying dyslexia are assumed to be similar across languages, for example, a deficit in phonological representation (Goswami, 2000; Goswami, 2002).

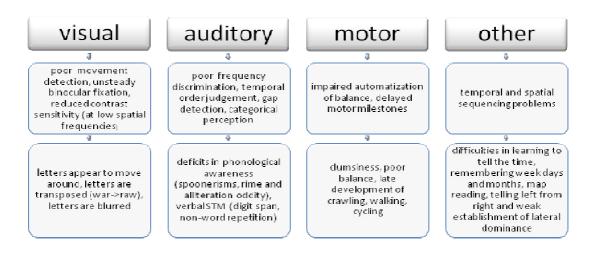


Figure 3.1. A summary of cognitive and behavioural deficits and symptoms in dyslexia. Figure is based on Stein & Walsh, 1997; Ramus et al. 2003; McArthur & Bishop, 2001.

As is the case with SLI (see Chapter 2), dyslexia is a heterogeneous disorder with a wide range of behavioural and cognitive deficits and the underlying biological and cognitive cause(s) of dyslexia are unknown. Dyslexia manifests itself as poor reading, poor verbal short-term memory (as exemplified in digit span and non-word repetition tasks), poor phonological awareness (as exemplified in phoneme deletion and spoonerism tasks) and slow lexical retrieval (as exemplified in rapid automatic naming tasks). Moreover, visual deficits with respect to reading and writing are found in dyslexia. And lastly, slight motor and temporal/spatial deficits are sometimes found in conjunction with dyslexia (see Figure 3.1).

There is a high comorbidity between dyslexia and other developmental disorders (such as ADD/ADHD, SLI and dyspraxia). Therefore, the variety of symptoms manifested in dyslexia and SLI and the high comorbidity between dyslexia and SLI has lead to a theoretical debate about the causal pathways to these two developmental language disorders.

3.3 SLI and dyslexia: the same disorder?

As introduced in previous chapters, SLI is impairment in acquiring spoken language that particularly impacts on the acquisition of syntax and morphology. Dyslexia, in turn, is impairment in acquiring the written form of a language. Therefore, at a first glance, SLI and dyslexia seem like distinct disorders. Recently it has been acknowledged, however, that there may be a close connection between SLI and dyslexia. Several studies show that SLI and dyslexia tend to frequently co-occur in the same individual (McArthur, Hogben, Edwards, Heath, & Mengler, 2000) and both disorders tend to run in families (Bishop, 2009). Moreover, depending on the definition criteria, several dyslexics meet the criteria for SLI and vice versa (see McArthur et al. 2000; Catts, Adlof, Hogan, & Weismer, 2005).

Furthermore, there seems to be a substantial overlap between the disorders at the cognitive and behavioural levels because many of the phonological deficits found in dyslexia (e.g., phonological awareness, verbal STM) are evident in individuals with SLI. Studies on at-risk populations also show that delays in oral language development (e.g., semantic and syntactic deficits) are evident in young children who later on develop dyslexia (McArthur et al., 2000). Therefore, it has been suggested that instead of considering SLI and dyslexia as two distinct disorders, they could be best characterized as stemming from similar underlying cognitive deficit and manifesting different ends of a single continuum.

This 'single-source model' or 'severity model' proposes that the underlying cognitive cause is the same (i.e., auditory-phonological) in SLI and dyslexia but these two

developmental disorders differ in the degree of severity of the deficit (Kamhi & Catts, 1986; Tallal et al. 1997). In other words, in the severe case of phonological impairment the affected individuals end up with oral language problems (syntax, semantics, discourse) and reading problems (SLI) whereas in the less severe impairment the affected individuals develop word reading problems (dyslexia). This model predicts that SLI and dyslexia both have their basis in phonological impairments (and consequently in the underlying auditory temporal deficits) but the deficits are more severe in SLI. Moreover, Kamhi & Catts (1986) state that while categorical labels such as 'reading impaired' and 'language impaired' may be useful for some purposes, these developmental disorders could also be viewed as subgroups of a more general group of 'language-disabled children'. Moreover, Tallal and her colleagues (Tallal et al. 1993; 1996) merge these two categories, SLI and dyslexia, into a single category of 'language learning impairment' (LLI). Sharma, Purdy and Kelly (2009), however, argued that the comorbidity of auditory, language and reading disorders could be largely dependent on the diagnostic criteria used. In their study, 68 children with suspected auditory processing disorder (APD) were assessed using a range of auditory, language, reading, attention and memory measures. Sharma, Purdy and Kelly (2009) reported that, overall, 47% of children demonstrated auditory, language and reading disorders of which 10% had either auditory + language disorder or auditory + reading disorder. Moreover, 12% of children had language and reading deficits without concurrent auditory deficit and 4% of children had APD alone. They concluded that even though APD can frequently cooccur with language and reading deficits, the causal relationship is not clear.

An alternative model to the severity model was put forward by Bishop & Snowling (2004, see Figure 3.2). Bishop & Snowling (2004) propose that despite these well documented phenotypic similarities, it is helpful to retain the terminological distinction between SLI and dyslexia. Bishop & Snowling (2004) argue that despite these similarities, there are differences between SLI and dyslexia that cannot be properly captured by one dimension of severity. They argue that oral language and phonological development can independently affect language and literacy outcomes. In dyslexia,

imprecise phonological representations lead to problems in phonemic orthographic associations and difficulties in literacy development. However, dyslexics can nevertheless benefit from top-down semantic and syntactic contextual information to compensate for their poor decoding skills. The literacy problems found in SLI, in turn, may have somewhat different origins because also non-phonological skills (e.g., syntax and semantics) seem to play role in literacy development and, therefore, children with SLI and dyslexia seem to demonstrate different developmental trajectories (Snowling, Bishop, & Stothard, 2000). Moreover, individuals classified as 'poor comprehenders' exhibit good reading accuracy but have difficulties in understanding of what is written without apparent phonological problems (Bishop & Snowling, 2004).

In other words, some cases of SLI can bee seen as a 'double-deficit' of co-occurring oral language difficulties and phonological deficits. In such cases the phonological deficits are similar to those found in dyslexia and do not differ, for example, in the degree of severity. The phonological deficits would, therefore, explain the overlap between SLI and dyslexia. Poor phonological (awareness) skills would be the primary cause of literacy deficits in dyslexia whereas poor comprehenders would demonstrate similar oral language deficits as is found in SLI but without co-occurring phonological deficits (Bishop & Snowling, 2004).

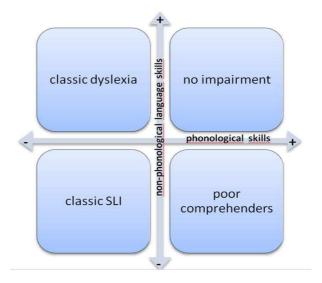


Figure 3.2. A model of the relationship between SLI and dyslexia. Figure is adapted from Bishop & Snowling, 2004.

The quadrant model by Bishop & Snowling (see Figure 3.2) demonstrate in two dimensional space how phonological and non-phonological skills contribute independently to the profiles of dyslexia and SLI

In short, according to this model, the overlap between SLI and dyslexia can be explained by phonological deficits but these deficits are not responsible for full extent of the language deficits in SLI. Furthermore, in SLI, the difficulties in syntax and semantics can also affect their literacy skills whereas poor comprehenders demonstrate difficulty in understanding what is read in the absence of phonological impairments, that is, they show weak semantic processing and normal phonology.

Recently, however, Catts et al. (2005) have put forward a more extreme form of this quadrant model. Catts et al. (2005) argue, similar to Bishop & Snowling (2004), that SLI and dyslexia are distinct disorders that have different developmental trajectories and different behavioural and cognitive manifestations. However, Catts et al. (2005) claim that the phenotypic and cognitive overlap observed in SLI and dyslexia is not due to underlying similar phonological deficits but to comorbidity rates, that is, the disorders are related but distinct. The comorbidity model of Catts and colleagues, therefore, predicts that due to high comorbidity of these disorders there should be several cases of SLI without any phonological problems and several cases of dyslexia without oral language problems as well as a subgroup of individuals with double deficit (SLI+dyslexia). To support this distinction, Catts et al. (2005) reported data from a longitudinal study and argued that, in their sample, only 15-20% of dyslexics meet the criteria for SLI and approximately 17-29% of those children that met the criteria for SLI in the kindergarten later fulfilled the criteria for dyslexia (NB McArthur et al. 2000 proposed figures of approximately 55%). Based on these data, Catts et al. (2005) proposed a model similar to the quadrant model by Bishop & Snowing (2004) differing, however, on the fact that where the quadrant model refers to individuals with oral language deficits and spared phonology as 'poor comprehenders', the comorbidity model considers these as 'SLI-only' (or children with a history of SLI).

3.5 Summary

There is a significant amount of evidence that, by far, not all dyslexics exhibit sensory deficits. There is also a growing body of evidence that dyslexia and SLI are related but distinct developmental disorders. Moreover, both SLI and dyslexia are heterogeneous disorders traditionally diagnosed by using psychometric exclusionary criteria that has proven to be unsatisfactory for some purposes (see Snowling, 2000; Bishop & Snowling, 2004). Therefore, it has been put forward that, in the search of underlying biological and cognitive causes of SLI and dyslexia future work should include investigations of reliable *cognitive* markers for SLI and dyslexia. This line of research would result in more homogeneous subgroups (in cognitive terms) that would allow researches to better investigate causal links between the brain and behaviour. Moreover, in order to disentangle the role of auditory processing in unsuccessful language development, we should, first of all, better establish its role in successful language development and, secondly, investigate the long-lasting effects of an APD by adopting, for example, a trajectory approach (Thomas, Annaz, Ansari et al. 2009).

4. Speech Perception: From Phonetics to Phonology

4.1 Introduction

In this chapter, I introduce the auditory theories of speech perception with reference to the models of how the surface level acoustic variance is treated by the auditory system (i.e., cue weighting and categorical perception). After establishing the auditory theoretical framework for the thesis, I explore how the underlying phonological categories develop in infancy and how distinct sounds and differences between sounds are processed in the auditory cortex as measured by electrophysiological methods.

4.2 The problem of speech perception

In normal situations speech sounds are perceived fairly rapidly without any major effort. The ability to perceive speech with such precision and at such a rate is a remarkable achievement that is unique to humans. During the past five decades, much research has been done to disentangle how the acoustic signal is processed and transformed in the peripheral auditory system and subjected to lower-level phonetic and higher-level (abstract) phonological processing. However, despite vast amount of research on speech processing, there is still much controversy surrounding the process of decoding speech. One problem in modelling speech perception arises from the nature of the speech signal itself. As the acoustic speech signal contains a huge amount of variation (within a speaker and between speakers), the identification of invariant phonetic features that would systematically map onto a phonological unit is virtually impossible. So far no theory has been able to reliably indicate what features in the signal are the crucial ones for perceiving the intended message.

Some researchers have suggested that the invariant properties are not found in the physical signal itself but the perceiver perceives the intended abstract gestures (e.g., lip rounding, tongue rising) of the speaker, that is, the invariance is associated with the production of speech (Liberman & Mattingly, 1985). The Motor Theory of speech

perception proposes that human beings employ specialized decoding patterns developed via evolution just for speech (Liberman & Mattingly, 1989). This "phonetic module" is specialized to process only speech and while it is active, other modules cannot access the auditory properties of the signal. In other words, speech is treated as a special code and processed independently from all other (non-speech) sounds.

The opposing theories suggest that the invariance is either embedded in the acoustic signal or extracted from the signal via decoding processes. Stevens (1980) proposed that, in fact, phonological distinctive features (Jakobson, Fant, & Halle, 1952) could be the answer to the invariance problem in speech perception. In phonological theory, distinctive features are a bundle of labels assigned to specific acoustic or articulatory features of a sound. Using distinctive features, phonemes can be broken down into smaller components, e.g., a nasal phoneme /n/ might be represented as a feature matrix [+sonorant, -continuant, +voice, +nasal, +alveolar]. These features are usually binary (e.g., ± voice, ±alveolar) establishing natural classes that undergo similar phonological processes and form larger units (e.g., source features, place features). The majority of the features are intended to be universal, so finding the acoustic correlates of these features could potentially unravel the invariance problem. The traditional abstractionist view of speech perception, however, has been challenged by the exemplar-based approaches (Goldinger, 1996). The exemplar models argue that particular instances of speech sounds are stored in the memory of the listener and compared against the sensory input in the categorization process. The exemplar models, therefore, consider variation across talkers as 'noise' and do not assume that normalized abstract representations exist. Experimental evidence showing that familiar voices are easier to process supports the exemplar-based approaches. Opponents of these models, however, argue that exemplar accounts are unrealistic due to the memory capacity requirements they assume (Johnson, 2005; Cutler, 2008).

In addition to the invariance problem, the fact that speech is a rapidly fading continuous signal proposes a major challenge to speech perception theories. In connected speech

there are no pauses between different sounds or even between words and the acoustic realization of each sound varies according to the context it is produced in. The exact mechanism how these physically different and overlapping (or co-articulated) segments are mapped onto (abstract) discrete units, such as phonemes or words, is more or less unknown. Co-articulation of speech segments increases variability in the signal and induces problems in segmenting speech into smaller units. For example, in a CV syllable the place of articulation in stop consonants (e.g., /b/, /d/ or /g/) affects the formant onsets of the following vowels (Delattre, Liberman, & Cooper, 1955), making the acoustic realization of a given vowel very different. When trying to identify invariant features within the speech signal, Delattre and colleagues (1955) found that segmenting these CV syllables into discrete units (e.g., [d] or [b]) that would yield a reliable percept of that one segment, is impossible. On the basis of their findings, Delattre et al. (1955) concluded that because of co-articulation, the speech signal contains two different types of acoustic "cues" signalling a discrete percept: steady-state cues (e.g., formant frequencies for vowels) and overlapping transitional cues (e.g., formant transitions cueing the place of articulation) that both contribute to speech perception.

Thereby, each sound segment usually consists of several acoustic cues (e.g., formant frequency, duration, voice onset time) which are dependent on the properties of the surrounding sounds and shared by several other sounds. Somehow the human perceptual system is capable of overlooking this heterogeneity produced by lack of invariance, lack of discrete units and by multiple simultaneous acoustic cues underlying the segments. And any plausible theory of speech perception should be able to account at least for the three fundamental issues: firstly, how the system deals with acoustic variance. Secondly, how the continuous signal is segmented into linguistic units and how these units develop in infancy, and, thirdly, to what extent the decoding processes are species-specific and even speech-specific.

4.2.1 Auditory theories of speech perception

In this section, I introduce three auditory theories of speech perception that assist in understanding some of the phenomena introduced later on and studied in the present thesis, namely the Auditory Enhancement Theory (Diehl, Kluender, & Walsh, 1990), the Fuzzy-Logic Model of Perception (Oden & Massaro, 1978; Massaro, 1987) and the TRACE model of speech perception (McClelland & Elman, 1986).

The auditory enhancement theory (Diehl et al., 1990) argues that perceptual properties determine the articulatory patterns, and not vice versa as suggested by gestural theories (Liberman & Mattingly, 1989). It also proposes that there is a direct and simple relation between acoustic/auditory and the symbolic/phonological levels. In other words, the acoustic signal (as opposed to articulatory gestures) provides the necessary information for perceiving speech. Whereas Stevens (1980) was looking for distinct invariant cues in the speech signal, Diehl and colleagues (1990) claim that even though a single acoustic property may correspond to a single unit in perception, listeners usually employ multiple simultaneous features that are mapped onto abstract distinctive features. They propose that the main unit in speech perception is the discrete sound or the distinctive features and, therefore, speech perception purely relies on categorization of auditory qualities. Moreover, whereas Stevens (1980) claimed that phonetic segments can be broken down to distinctive features which, in turn, are directly mapped onto the physical signal, Diehl and colleagues (1990) argue that there must be an intermediate layer between distinctive features and acoustic signal. This intermediate level, the Intermediate Perceptual Properties (IPP), can contribute to more than one independent auditory property. Moreover, the underlying phonological features have their individual phonetic correlates (the IPPs) that can also be integrated to contribute to a single auditory property. In other words, certain phonological distinctions relevant for a given language are perceptually enhanced and then combined to contribute to the identification of a distinctive feature. For example, in the distinctive feature [voice], the main perceptual property corresponding to [+voice] value is the presence of low frequency energy that can be subcategorized into several underlying acoustic-phonetic properties all contributing towards a single perceptual unit.

Similarly, The Fuzzy-Logic Model of Perception (FLMP) (Massaro, 1987) proposes that the primary information listeners employ when listening to speech is the auditory information –assuming it is clear enough. But unlike auditory enhancement theory, the FLMP argues that other modalities, such as the visual domain, can affect the perception by supplementing the sensory information, not necessarily by changing it. According to FLMP, each syllable is represented in memory as a prototype that consists of features with ideal values. Perceiving speech, therefore, involves estimating how well these ideal features match the signal. Generally, these features correspond to phonetic properties, for example, the syllable /bʌ/ consists of a visual feature (e.g., rapidly opening lip closure) and the corresponding auditory feature (e.g., rapid rise in all formant frequencies). According to FLMP, speech perception relies on general perceptual categorization principles and comprises of three stages: an evaluation, integration and a decision stage that are successive and overlapping.

The FLMP proposes that each property of the signal is first evaluated with respect to the expected value by assigning so called fuzzy-logic or fuzzy-truth values (continuous values between 0-1). These independent values are then combined or integrated to provide the final value and finally checked against the best fitting prototypical values. According to FLMP, different sources of information (e.g., auditory and visual) are evaluated independently. In FLMP framework, the principles governing the perception of speech are considered to be universal and common to all pattern recognition -not necessarily specific to speech.

The interactive spoken word recognition model TRACE (McClelland & Elman, 1986) is probably one of the best known and most influential connectionist models of human speech (word) recognition. Whereas FLMP argued that each source of information is

independently evaluated and integrated only at later stages of processing, the TRACE model of speech perception is highly interactive.

TRACE proposes that the auditory input excites a three-layer network of units that are all connected by excitatory (between layers) or inhibitory (within layers) connections. These units correspond to standard linguistic units: phonetic features, phonemes (that are position sensitive) and words. All connections between units are bi-directional, thus, information flow from bottom-up is similar to the top-down flow. During the initial processing the speech signal is transformed into an "auditory spectrogram" and the time-slices are fed to the input units which are sensitive to surface-level acoustic-phonetic features. For example, if in the initial analysis the features correspond to properties "voice", "alveolar", "stop", this excites /d/ at the phoneme layer which in turn activates words *dog*, *dark*, *deep* etc. at the word layer. So, different lexical items can share same segments and listeners have to unconsciously consider several parallel alternatives and choose the one that is most likely candidate based on probabilities.

In summary, auditory theories suggest that the invariance in speech perception lies in the acoustic signal or in the auditory system. Furthermore, these theories propose that the initial unit of perception is a sub-syllabic or even a sub-phonemic unit and that the initial stage of speech perception consists of some type of preliminary acoustic feature analysis. However, since the speech signal consists of several simultaneous acoustic features or cues that vary according to the specific context, we need more detailed accounts how these cues are treated in the perceptual system.

4.3 Integrating multiple cues: trading relations and acoustic cue weight

In natural situations, phonetic contrasts contain several cues that can signal a single percept and different combinations of these cue-values can result in an equivalent percept despite the acoustic variation. In other words, a change in one cue that normally would change the percept can be offset by changing another cue to maintain the original percept. This multiple cue integration where cues are able to "trade" in the amount they

are needed perceptually is called *phonetic trading relation* (Repp, 1982). Phonetic trading relation reflects perceptual specialization that takes into account the common origin of acoustic-phonetic cues.

How these different acoustic cues interact and influence one another is an empirical question in speech perception research. For instance, a consonantal contrast [t] – [d] is distinguished by a feature [voice] that can be acoustically realized via a contribution of several cues such as aspiration, F1 onset frequency and the duration of the preceding vowel. In laboratory conditions, a single cue may be sufficient for the identification of a particular contrast but in natural situations people are very likely to utilize several different cues that signal towards the same entity. In specific contexts some of these cues are considered more informative than other cues. In other words, trading relation is a manifestation of sensitivity to multiplicity of acoustic information.

Fitch, Hawles, Erickson, & Liberman (1980) investigated the use and perceptual equivalence of multiple acoustic cues in perceiving the English voiceless stop consonant [p]. They used synthetic words "slit" – "split" and varied, firstly, the duration of silence between the offset of fricative /s/ and onset of liquid /l/ and, secondly, the formant transitions at the onset of /l/. In other words, they manipulated two distinct cues that both can signal the presence of voiceless consonant [p]: the duration of silent interval and the formant transition appropriate for bilabial stop closure. They used a paradigm where one of the cues varies continuously (duration) and one of the cues is fixed (formant transition: indicating either [p] or absence of [p]). They found that when the formant transitions cued presence of [p], only 55 ms of silence was required for accurate identification, whereas when the formant transition cued absence of [p], about 80 ms of silence was required for perceiving [p]. In other words, both cues influenced the phonemic boundaries, that is, listeners integrated both cues in forming a unitary percept, giving rise to phonetic trading relations.

So, variable combinations of acoustic cues can result in a unitary percept. However, these multiple cues can either cooperate with each other (i.e., point towards same percept) or they can create a conflicting situation (i.e., point towards a different percept). In the same study by Fitch and colleagues (1980), the two cues signalling presence/absence of stop consonant were manipulated by creating a condition where these cues are in co-operation/conflicting. The cues are co-operating when the duration of silent interval is long and accompanied by formant transitions (i.e., both cues are signalling [p]) or when the duration of silence is short and there is no formant transition present (i.e., both signalling the absence of [p]). However, these cues conflict when long duration of silence is accompanied with no formant transition and when short silence is accompanied with formant transition typical for [p]. By using the two sets of cooperating and conflicting cues, Fitch et al. (1980) found that the discrimination of tokens where the two cues both signalled the presence of [p] from those tokens where the both cues signalled the absence of [p] was relatively easy. However, people made significantly more errors when discriminating two configurations of the same cue (i.e., a within-category difference of transitions appropriate for [p] from silence appropriate for [p]). According to Fitch and colleagues (1980), the perceptual system can treat two different cues to the same percept as equivalent indicating that the presence of either one of these is sufficient to arrive at the same percept. However, Hazan and Rosen (1991) demonstrated that there is a substantial amount of variability across listeners in labelling performance in full-cue vs. reduced-cue situations. Hazan and Rosen (1991) argue that participant's performance on this task could be dependent on other (intrinsic) factors such as cue salience that is influenced by overall speech pattern complexity and vocalic environment.

In short, speech sound perception is based on the use of an integrated phonetic percept where acoustically different stimuli can be perceptually equivalent. Listeners seem to make use of multiple acoustic cues in the speech signal but these acoustic cues do not necessarily have the same relative role in all situations. In fact, listeners are able to assign more "weight" to different cues, for example in the presence of background noise.

In addition to noise, factors such as change in the task demand or attentional manipulation may change the perceptual strategy as to what cues to employ in deciding the phonetic label of a token (Gordon, Eberhardt, & Rueckl, 1993; for a review see Mayo, 2000). Furthermore, the use of these cues does not seem to be developmentally a fixed property of the perceptual system (Krause, 1982; Nittrouer & Studdert-Kennedy, 1987; Nittrouer, Manning, & Meyer, 1993; Ohde, Haley, Vorperian, & McMahon, 1995; Nittrouer, 2005).

Investigating development, Nittrouer and Studdert-Kennedy (1987) reported that children and adults generally use different cues to distinguish between phonetic contrasts. Nittrouer and Studdert-Kennedy (1987) used two cues, the frequency of the friction noise and vowel onset transition, signalling two different fricatives, /s/ and /ʃ/, in different vowel contexts: <sue>, <shoe>, <sea>, <she>. They found that younger children (3 years of age) use primarily the vowel transition cue whereas older children (5 years) weight this cue slightly less. Interestingly, adults and children from about 7 years of age rely more on the friction noise than on the transition cue.

However, a later study by Nittrouer (1992) failed to replicate this result. In this study, adults and children identified $/d\Lambda/ - /g\Lambda/$ contrasts embedded in two-syllable VCCV tokens $/\Lambda r d\Lambda/ /\Lambda l d\Lambda/ /\Lambda r g\Lambda/ /\Lambda l g\Lambda/$. Unlike the previous study by Nittrouer and Studdert-Kennedy (1987) where children were shown to be sensitive to transitional cues, in this particular study adults were more influenced by the preceding VC syllable, that is, the transition by giving more $/g\Lambda/$ responses when preceded by syllable $/\Lambda l/$ demonstrating carryover co-articulation (Mann, 1980).

On the basis of these findings, Nittrouer and colleagues (1987, 1992, and 1993) concluded that this difference is due to children using a different perceptual unit to adults. Whereas adults generally use sub-syllabic units as central units of perception, children can only make use of larger chunks, such as syllables or words. The change in

perceptual strategy takes place during the first 5-7 years of life as a result of increasing linguistic experience. This Developmental Weighting Shift (DWS) introduced by Nittrouer and colleagues (1993) proposes that in perceiving speech segments, children generally assign more weight to the dynamic cues as they signal syllable structure whereas adults weight more the segment-intrinsic cues.

However, Sussman (2001) proposed a sensory hypothesis that, as opposed to the DWS, holds that children merely make use of those cues that are spectrally more informative (e.g., louder and longer) that also explains the differences found in adults' and children's cue weighting scores. Sussman (2001) argues that these differences are due to general sensory processing differences between children and adults.

Mayo & Turk (2005) compared these two (DWS vs. sensory hypothesis) accounts in an identical cue-weighting task used by Nittrouer and colleagues. Mayo and Turk (2005) compared vowel onset formant transition cues that are spectrally distinct to those that are spectrally similar (e.g., /no-mo; do-bo; ta-da/ vs. /ni-mi; de-be; ti-di/) in groups of young children (3/4-, 5- and 7-year olds) and adults. They reported, unlike what is predicted by Sussman's model, that spectral informativeness plays a role in cue weighting in *all* participants (i.e., children and adults alike) and that children differed from adults only for some consonant contrasts. Moreover, Mayo & Turk (2005) argued that the pattern of results did not support the DWS hypothesis either, in that children did not give more weight to transitional information than adults (see also Hazan & Barrett, 2000; Mayo & Turk, 2004 for similar results). They concluded that it is likely that cue-weighting is influenced by both the segmental context and the physical distinctiveness of available cues.

To summarize, cue weighting is a quantitative measure of perceptual categorization. We know that listeners may change the relative weighting given to different sources of acoustic information and they seem to do so in noisy situations or when the task demands change to maximize the accuracy. On the whole, it seems that even though the

learning mechanisms of perceptual categorization are not fully understood yet, the experience with certain patterns and regularities in the native language form the basis of listeners' abilities of weighting of acoustic dimensions. In the next Section, I describe the categorical perception of speech and the development of these native language perceptual categories.

4.4 Development of speech perception

4.4.1 Categorical perception of sounds

Categorical perception (CP) refers to the listeners' ability to organise heterogeneous input into finite number of categories. As opposed to trading relations and cue weighting where the system deals with multiple cues simultaneously, in a traditional CP experiment the effect of a single cue in forming a percept is observed. CP emerges whenever perceived within-category differences are compressed and between-category differences are enhanced. The perceptual system seems to arrange input into meaningful entities or contrasts ignoring the "irrelevant" variation. Intense research during the past 50 years indicates that this is how the perceptual system operates. In other words, the equal physical changes in a signal are not always treated as equal across the perceptual space but perception seems to be discontinuous, or non-linear, at some points. Thus, some changes seem to be more meaningful and create an abrupt change in perception whereas equal changes (on the same physical scale) have a negligible effect on the percept (see Figure 4.1).

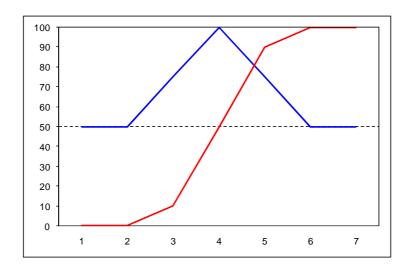


Figure 4.1. Categorical perception. Theoretically ideal identification (red) and discrimination (blue) functions. The x-axis represents the stimulus number along a particular continuum and the y-axis % of category [x] responses and discrimination accuracy. The 50% point is indicated by the dashed line.

In a typical CP study a set of stimuli (e.g., 7, Figure 4.1) are synthesized and their acoustic properties are varied along a continuum (e.g., changes in VOT, vowel duration, formant frequency change of a transition). These stimuli are then played to the listeners in a labelling (identification) task where the participant is asked to provide a linguistic label to the stimuli (e.g., either [x] or [y]). Moreover, these stimuli are then presented in pairs in a discrimination task (e.g., "same-different" task) to the participant.

This CP phenomenon was first reported by Liberman, Harris, Hoffman, & Griffith (1957) who showed that in certain acoustic cues, such as VOT, at the category boundary (i.e., the 50 % point where the subject is at a chance level in their labelling, see Figure 4.1) the stimulus identity (or the "label") changes relatively abruptly resulting in steep labelling functions whereas within the categories this function is flat (the perception is more uniform). Interestingly, however, the stimuli are easier to distinguish from one another at the category boundary when compared with within-category differences, that is, the discrimination sensitivity is enhanced where the labelling performance is decreased. However, this perceptual phenomenon is not thoroughly uniform across all

speech sounds. For example, vowels are generally perceived less categorically than consonants (Fry, Abramson, Eimas, & Liberman, 1962). Moreover, Massaro (1987) even claimed that categorical responses do not necessarily require categorical perception and that, in fact, CP is merely a pseudo-phenomenon or an artefact of the experimental design affected by the fact that linguistic labels tend to be categorical by nature.

Categorical perception was originally thought to be a species-specific property unique to humans and unique to perceiving speech. However, later research showed that CP is neither speech specific (Pisoni, 1977) nor unique to humans (Kuhl & Miller, 1975; Kuhl & Miller, 1978). Instead of categorical perception and perceptual separation at the category boundary, Kuhl (1991) argued that it is the internal hierarchical organization of the category that is unique to humans. In cognitive science, it was already well established that some instances within a given category can be perceived as being better representatives of that category and preferred over the other members (Rosch, 1973). The notion of category representatives, or "prototypes", was extended from semantic categories to phonological categories.

Generally, the perceptual prototypes are easier to remember and are stored in the memory. In addition to prototypicality, (Kuhl, 1991) reported that within a speech sound category, those instances that are acoustically close to the prototype are perceptually harder to discriminate than those instances that are closer to the category boundary - despite equal acoustic distances. This phenomenon was called as the "perceptual magnet effect" (PME) where the prototype serves at a magnet pulling nearby elements towards it, thus shrinking the perceptual distance near the prototype. Furthermore, Kuhl (1991) proposed that this perceptual phenomenon is unique to humans, that is, the category goodness influences the perception of human adults and infants but does not appear to play a role in other animals. The magnet model, however, has since been challenged by other researchers (Lively & Pisoni, 1997; Lotto, 1998).

Categorical perception and the perceptual magnet effect both hypothesize that the goal in adult speech perception is derivation of a sequence of *phonemes* relevant to a listener's native language. However, how and when these phonological categories develop when a child begins to speak and to what extent are they separated from non-phonetic acoustic information is not yet clear.

4.4.2 Development of phonological categories

Adults' phonological categories are characterized by trading relations and cue weighting that develop gradually during approximately the first 5-7 years of life. However, acquisition of language starts well before children enter the school and native language phonological categories must be established prior to this age. In this Section, I review what is known about phonological development during the first year in infancy.

Perception of fluent speech is a complex process —and even more so with children's less mature information-processing mechanisms. When infants are exposed to their native language(s) they have to learn to identify relevant segments (words, syllables, phonemes) from the continuous stream of speech. Different languages have different phonological units (phonemes) and realizations of these units (allophones) and the infant's task is to learn to connect the variable surface forms to the underlying phonological forms relevant to the given language.

As stated above, successful speech perception cannot depend upon responding to any absolute set of acoustic properties, but infants must also be capable of dealing with the variation, that is, solving the normalization problem. Moreover, natural language learning requires social interaction possibly ensuring that infant's attention focuses on speech produced by other members of the community. In other words, infants' language learning is communicative learning resembling that demonstrated, for example, in songbirds (Kuhl, 2004).

Much research has been devoted to investigating the perceptual capabilities of infants and young children. Eimas and colleagues (Eimas, Siqueland, Jusczyk, & Vigorito, 1971; Eimas, 1975; Eimas & Miller, 1980) investigated infants under 4 moths of age in CP along several acoustic dimensions such as VOT (/p^h_A/ - /b_A/), place of articulation (/b_A/ - /d_A/) and manner of articulation (/b_A/ - /w_A/). They showed that infants are excellent in distinguishing between category differences between sounds and show similar tendency for CP as adults and chinchillas from very early age. Jusczyk, Copan, & Thompson (1978) tested infants at 2 months in their ability to discriminate glides /j/ and /w/ syllable initially and medially and concluded that infants were able to distinguish these contrasts early on regardless of its position in a syllable. Moreover, infants also showed adult-like ability to categorize non-speech sounds (Jusczyk, Pisoni, Walley, & Murray, 1980).

In addition to CP and discrimination of NL contrasts, Werker and Tees (1984) showed that, infants, unlike adults, are able to discriminate between a wide range of *universal* acoustic-phonetic differences, that is, contrasts that are irrelevant for the ambient language. Werker & Tees (1984) and Aslin and colleagues (1981) presented several different foreign language (FL) consonant contrasts to English-learning infants and reported that infants showed accurate discrimination even in these novel FL sounds. In other words, infants show the general ability to categorize speech sounds but they are more sensitive to acoustic-phonetic variation than adults. These findings indicate that during the first few months of life, infants' phonological categories seem to be fundamentally different from adults' categories. Moreover, Hazan & Barrett (2000) showed that CP development continues well into adolescence. They argued that children and adolescents are less flexible than adults in their perceptual strategies and therefore also less consistent in their categorizing performance.

It is well established that experience of a particular language alters perception and that this general ability to discriminate non-native phonetic contrasts disappears gradually during the first 12 months of life (for a review see Kuhl, 2000). After the initial

"universal perceiver" phase, around 6 months, phonetic categories start to be structured in language-specific ways around the prototypes, showing similar magnet effect to adults (Kuhl et al., 2006). But what mechanism drives this developmental change in speech perception? One suggestion is that infants calculate statistical distributions of the auditory-phonetic input that provide clues about the phonological structure of a language, e.g., about the vowel inventory (Maye, Werker, & Gerken, 2002). In addition to sensitivity to distributional patterns within a sound system of a language, infants initially also calculate transitional probabilities between syllables which guide lexical learning (Saffran, Aslin, & Newport, 1996). Saffran and colleagues (1996) played 2 minute strings of synthesized continuous speech consisting of three-syllable pseudowords such as "tibudo" "pabiku" and "golatu". The transitional probability among the syllables in these pseudowords was 1.0 whereas it was only 0.33 between other adjacent syllables. To detect the "words" embedded in the continuous speech stream, infant's had to be able to track the statistical relations among syllables. These statistical learning skills observed in human infants are not, however, restricted to language learning or only to humans. Similar effects are found using other auditory stimuli (tones), visual stimuli and by presenting speech to monkeys (Hauser, Newport, & Aslin, 2001; Kirkham, Slemmer, & Johnson, 2002).

Johnson and Jusczyk (2001) reported that about 8 months of age, infants change their strategy to recover words from the speech stream. Johnson and Jusczyk (2001) argue that by this time infants begin adopting a more adult-like strategy to use prosodic cues (e.g., detecting stress patterns of words) instead of transitional probabilities.

In short, during the initial 12 months of life, infants change from universal perceivers to language-specific perceivers, losing sensitivity to detect "irrelevant" phonetic information. At around this stage phonological categories start shaping (magnet effect) and possibly the abstract representations of sounds start to emerge (e.g., prototypes). In addition to this, infants change their strategy to detect words in the continuous speech stream from statistical cues to prosodic cues. Kuhl (2000) calls this phase as developing

"neural commitment" to native language (Native Language Neural Commitment, NLNC) that is essential for successful language learning. The NLNC hypothesis argues that losing the sensitivity to FL contrasts is a necessary condition for excellent NL speech perception and later language development. Moreover, Kuhl (2000) proposes that the degree of commitment (i.e., the ability to "tune into" NL) is a predictor of later language skills, that is, phonetic abilities would 'bootstrap' language learning (or at least lexical learning). However, investigating the neural commitment to learned structure requires understanding the cerebral bases for phoneme perception. In the next two Sections, I shortly review what is known about phoneme processing at the auditory cortex and about decoding native and non-native speech sound contrasts.

4.5 Speech sound processing in the auditory cortex

Speech perception seems to be highly automatic and obligatory in nature. However, despite the great interest in structural and functional organization of the brain during the past decades, the neural substrates underlying the processing of speech are not well understood.

Auditory cortex is responsible for the processing of all auditory events, including speech. It is located at the temporal lobe in the posterior half of the superior temporal gyrus, STG. During the early stages of processing the acoustic-auditory signal travels to the primary auditory cortex, A1, from the cochlea where the initial processing of the signal takes place. This pre-cortical processing seems to be general to all sounds and, therefore, speech-specificity may only arise at the cortex (Scott & Johnsrude, 2003).

The majority of the information about the functioning of the auditory cortex is gained from studies in animals, for example, in nonhuman primates. Generally, the primate auditory cortex can be divided into three regions, the core, belt and parabelt, on the basis of their connections and organization (Kaas, Hackett, & Tramo, 1999) (see Figure 4.2). This distinction appears to be paralleled in the human brain.

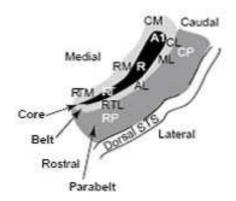


Figure 4.2: Primate Supratemporal plane. Figure adapted from Scott & Johnsrude, 2003. Abbreviations: A1, primary auditory area; AL, anterior lateral belt; CL, caudal lateral belt; CM, caudal medial belt; CP, caudal auditory parabelt; ML, medial-lateral belt; RM, rostro-medial area; RP, rostral parabelt; RTL, lateral rostrotemporal auditory belt; RTM, medial rostrotemporal auditory belt. Reprinted with permission from Elsevier.

Cortical auditory processing originates from the core region projecting to the surrounding regions, that is, to the belt and parabelt with further transmission from the parabelt region to higher level processing areas as the dorsolateral frontal cortex and the superior temporal sulcus (STS). The primate core, belt and parabelt are highly hierarchical in their connections and response properties (Kaas & Hackett, 2000). This hierarchical processing entails that these primary sensory areas deal with basic processing of the stimuli whereas the higher-order (secondary) areas are engaged in extracting more complex aspects of the incoming signal (Scott & Johnsrude, 2003). However, in addition to this hierarchical processing, Inui, Okamoto, Miki, Gunji, & Kakigi (2006) argue that there are also several parallel processing streams between core, belt and parabelt where multiple attributes of the auditory stimuli are processed by segregated pathways.

The core and belt areas also display some functional specificity (e.g., tonotopy) that arises from the mechanical properties of the cochlea (Hackett, Preuss, & Kaas, 2001; Rauschecker, Tian, & Hauser, 1995). Thus, tonotopy can be referred to as a basic principle of the information processing in the auditory system. In addition to this

topological organization of frequency information at the primary auditory areas, also other mapping principles for speech sounds have been put forward. Recent research has proposed that the auditory cortex is organized phonotopically or phonemotopically thus enabling each vowel category to be represented by separate neural populations (Diesch & Luce, 2000; Obleser, Elbert, Lahiri, & Eulitz, 2003; Shestakova, Brattico, Soloviev, Klucharev & Huotilainen, 2004).

Obleser and colleagues (2003) proposed that phonetic distance in vowels (such as F1-F2 distance) is preserved at the cortical representations. They investigated the distance of cortical representation (as indexed by N1m) of German vowels [i], [e] and [a] where the vowels [a] and [i] are phonologically and spectrally further apart in vowel space than vowels [i] and [e]. They hypothesized that if auditory cortex is capable detecting this F1-F2 distance it would provide strong evidence for phonemotopic organization of the human auditory cortex. The study of Obleser et al. (2003) as well as a later study by Shestakova and colleagues (2004) confirmed this prediction, suggesting that at least vowels are represented at the auditory cortex according to their phonological properties.

Inevitably, we can consider phonetic perception and extracting phonological properties as linguistic by nature. However, an interesting question is, at what point does phonetic processing depart from general auditory processing? And, moreover, are there distinct neural pathways for speech and non-speech? Recent electrophysiological techniques have provided a useful tool in investigating this relationship between speech and non-speech processing.

4.5.1 Mismatch negativity (MMN) as an index of auditory sensory memory

Mismatch negativity (MMN) is a component of auditory event-related brain potentials (ERPs) usually measured with EEG (*electroencephalography*) or MEG (*magnetoencephalography*) techniques. Both EEG and MEG have a high temporal resolution (at a millisecond range) that makes them an ideal tool for studying speech processing where events take place at a fast rate. Moreover, M/EEG do not generate

noise (such is the case with *functional magnetic resonance imaging*, fMRI) which makes them particularly appealing in the auditory speech perception research.

EEG records the ongoing electrical activity of the brain using an array of electrodes on the scalp. Cortical neurons produce two main types of activity: action potentials that are discrete voltage spikes and postsynaptic potentials (PSP) that arise when neurotransmitters bind to receptors. EEG signals are mainly produced by excitatory postsynaptic potentials generated at apical dendrites of pyramidal cells in the cortex. A single PSP produces a current dipole (i.e., a pair of negative and positive electrical charges) that is too weak to be measured outside the head. Therefore, cumulative summation of (approximately) simultaneous activity of at least thousands of neurons is required. The cumulative summation of the potentials is enhanced by the spatial alignment of the cortical neurons (i.e., aligned perpendicular to the cortical surface).

These electric currents measured at the scalp also induce a magnetic field that is detected outside the head with SQUID (Superconducting Quantum Interference Device) sensors. In other words, these two methods, EEG and MEG, are closely related since both techniques measure the same synchronized neuronal activity. The advantage of using MEG over EEG is that the tissues outside the brain are more or less transparent to the signal so that, unlike in EEG, the signal is not distorted, improving the spatial resolution (see Hari, Levänen, & Raij, 2000 for a review). However, MEG is selectively sensitive to tangential currents, that is, it fails to detect electric currents that are vertically oriented to the brain surface. Thus, magnetic signals are largest for superficial dipoles that run parallel to the surface of the skull, and fall off rapidly as the dipoles become deeper or perpendicularly oriented.

As is the case in the majority of cognitive studies, the ongoing M/EEG is time-locked to some experimental event trigger, thus providing a neural response with respect to this outside event, called event-related potential (ERP, or event-related magnetic field, ERMF). ERPs are extracted from the ongoing background EEG activity with simple

averaging technique. ERP components are characterized by polarity of voltage deflections (positive/negative), latency (in millisecond) and topography. ERPs can be time-locked to for example sensory, cognitive or motor events, therefore proving a useful tool in cognitive sciences to investigate online processing of external and internal events.

MMN is a frontocentrally negative component of auditory ERPs that usually peaks at 100-250 ms after stimulus onset. MMN has its main generators at the primary auditory cortex (Näätänen, Gaillard, & Mäntysalo, 1978, for a review see Näätänen, 2001 and Näätänen, Jacobsen, & Winkler, 2005) and it is elicited when subject's attention is directed away from the stimuli, i.e., it is relatively automatic. However, some studies show that strongly focused attention can modulate MMN amplitude suggesting that MMN is not completely attention-free (Woldorff, Hillyard, Gallen, Hampson, & Bloom, 1998).

MMN reflects automatic change-detection where the incoming deviant signal is compared to the sensory-memory representation of the regular aspects of the frequently occurring stimuli. This comparison process can also trigger a frontal component (and even the subsequent P3a component) reflecting the involuntary attention switch to stimulus change (Giard, Perrin, Pernier, & Bouchet, 1990; Rinne, Alho, Ilmoniemi, Virtanen, & Näätänen, 2000).

MMN is elicited by any discriminable auditory change such as simple sinusoidal tones of different frequencies, amplitudes and durations as well as more complex tones, such as vowels and consonants, or even syllables, words and sentences (for a review of MMN, see Cheour, Leppänen, & Kraus, 2000; Näätänen, 2001; Shtyrov, Pulvermuller, Näätänen, & Ilmoniemi, 2003). In other words, MMN is thought to reflect a memory-related neuronal activity taking place at the auditory cortices.

During the past 30 years MMN has been widely used in humans: healthy adult subjects of different language backgrounds, different patient groups (e.g., people with schizophrenia, alcoholism, dyslexia, SLI, autism), coma patients, young children and even sleeping newborn infants, as well as different non-human animals such as cats, guinea pigs and rats.

Näätänen and colleagues (Näätänen & Winkler, 1999; Näätänen, 2001) propose that MMN elicitation is based on short-term memory (STM) traces at the auditory cortex. Their model suggests that so called Central Sound Representations (CSR) link the perception and memory. The CSR is encoded as a memory trace (or interlinked traces) and a sound can only enter into long-term memory (LTM) in a form of this CSR. Näätänen's model of initial sound perception (and consequently of speech perception) holds that as a sound enters the auditory cortex, the "feature analyzers" perform an initial acoustic analysis (frequency, intensity and duration) of the incoming signal.

The initial processing is part of the pre-representational system (indexed by N1) that is not directly accessible to higher controlled processing. The output of the initial feature analysis is mapped onto sensory memory where the feature integration and temporal integration processes aid in forming a unitary percept. This integration stage corresponds to conscious percept and thus reflects full sensory analysis. In other words, at this integration stage the stimuli become representational and available for other cognitive operations.

The CSR also determines the auditory accuracy traditionally measured with recognition and discrimination tasks. However, the neurophysiological discrimination accuracy, as indexed by MMN, precedes (or defines) actual behavioural discrimination, and not vice versa (Tremblay & Kraus, 2002; Näätänen, 2001). In addition to the neurophysiological measure of general discrimination accuracy, MMN has been proposed to reflect *speech-specific* and even *language-specific* perception.

Näätänen and colleagues (1997) studied Finnish and Estonian subjects demonstrating that MMN is sensitive to linguistic experience. Näätänen et al. (1997) used five different types of stimuli: a standard stimulus /e/ that is a phoneme in both Finnish and Estonian, and fours deviants that are either phonemes in both languages (e.g., /ø/) or phonemic only in Estonian (e.g., /õ/). They reported that despite equal acoustic differences, the MMN to the non-native Estonian contrast was significantly smaller in the Finnish subjects. Similarly, Winkler and his colleagues (1999) showed that MMN can also reflect perceptual plasticity such as language learning. Winkler and colleagues (Winkler, Kujala et al., 1999) studied Hungarian and Finnish native speakers and Hungarian speakers learning Finnish. They found that whereas Hungarian and Finnish only showed reliable MMNs to their native language contrasts, the Hungarian who had been learning Finnish were also sensitive to the non-native language contrast.

Following Näätänen's theory, Cheour et al. (1998) proposed that speech perception is based on a set of phoneme traces that are thus specific to speech and also to a particular language. Moreover, Cheour et al. (1998) claimed that these traces are gradually formed during the first year in infancy providing the recognition models for native language speech perception. Cheour and colleagues (1998) studied Finnish and Estonian infants (using the same setup as in Näätänen et al. 1997) and reported that at around 6 months of age, the MMN in infants reflected only acoustic distance between stimuli. However, by 12 months of age the native language phoneme traces had developed, supporting the earlier behavioural findings (Aslin et al., 1981; Werker & Tees, 1984; Kuhl, 2000). Several cross-linguistic studies as well as studies in speech and non-speech processing have confirmed that MMN reflects speech- and language-specific memory trace activation (Dehaene-Lambertz, 1997; Näätänen et al., 1997; Winkler, Kujala et al., 1999; Winkler, Lehtokoski et al., 1999; Rivera-Gaxiola, Csibra, Johnson, & Karmiloff-Smith, 2000; Peltola, Tuomainen, Koskinen, & Aaltonen, 2007).

In short, Näätänen (2001) proposes that phonetic memory traces are permanent, their development depends on attention (but they are automatically activated) and they can

provide reference information, for example, for the control of pronunciation. To account for the relationship between auditory discrimination and speech/language-specific processing Näätänen and his colleagues (1997) suggested that these language-specific traces are *additive* to acoustic perception. Additive traces would indicate that a speech sound simultaneously activates both the speech-specific and the general acoustic (feature) traces, thus predicting larger responses to speech contrasts. Furthermore, Näätänen (2001:12) argues that MMN provides evidence for the existence of neuronal populations that encode the invariant features of the acoustic signal. In other words, the primary function of these phoneme traces is to serve as templates in speech perception without which "one would perceive spoken language acoustically, similarly to any other complex sound, with the main difference from speech perception being that there is no category effect".

However, not all agree with this view. In fact, Jääskeläinen and colleagues (2004) proposed that MMN is produced by neurons generating the obligatory N1 wave (Jääskeläinen et al., 2004; see also the response Näätänen et al., 2005). Moreover, some studies fail to find similar speech-specific effects when using more complex stimuli as controls (Sussman et al., 2004; Tuomainen & Tuomainen, 2006). It is also noteworthy that Näätänen's CSR model does not specify what the invariant features are that are encoded as a memory trace. Moreover, if MMN is seen as the "neural mechanism of categorical perception" (Näätänen, 2001:8) this cognitive model for MMN elicitation does not provide plausible explanation to when and where does speech-specificity arise from since categorical perception reflects general functioning of the auditory system in humans and non-human animals. Recently, a neurophysiological predictive coding framework has been put forward to account for the elicitation of the mismatch response (see Garrido, Kilner, Stephan, & Friston, 2009). According to this framework, mismatch response is generated when the representations at the higher-level cortical areas do not fit with the data received from the lower-level areas, that is, with the current inputs predicted from past inputs (see Baldeweg, 2006; Garrido et al. 2009).

4.6 Summary

Perceiving speech seems to be a highly automated and even obligatory task, where the acoustic signal is passed though a chain of different stages of analysis, the ultimate goal being to derive the *meaning* of the speaker's utterance. The fundamental questions in speech perception research are: What are the units of perception: features, phonemes, allophones, articulatory gestures or syllables? How do the acoustic-phonetic units actually map onto linguistic units or different levels of representations? Could these different levels of linguistic representations be neuroanatomically confirmed? And finally, when and where does speech-specificity and language-specificity arise from?

In other words, the models of speech perception have to explain *what* information is derived and *where* and *when* this information is processed, stored and integrated. Recent electrophysiological techniques have provided as a useful tool to investigate speech and language processing and their neural correlates. Moreover, with these techniques we can gain new insights into typical and deviant language development.

5. Aims and structure of the thesis

The main aim of the present thesis is to clarify the nature and locus of auditory deficits in Specific Language Impairment (SLI) and dyslexia.

By investigating the *locus* of the auditory deficit in SLI and dyslexia I focus on the following points:

- Is SLI associated with persistent auditory deficits and how many individuals with SLI still show these deficits in (early) adulthood?
- Is dyslexia associated with persistent auditory deficits and how many individuals with dyslexia still show these deficits in (early) adulthood?
- If SLI and dyslexia are associated with auditory problems, are the auditory deficits different in SLI and dyslexia?
- Is the auditory deficit specific to speech?

By investigating the *nature* of the input-processing deficit in SLI and dyslexia I focus on the following points:

- Is the input-processing deficit a consequence of inability to focus on relevant acoustic cues (Chapter 6)?
- Is the input-processing deficit a consequence of general inability to discriminate sounds (Chapter 7)?
- Is the input-processing deficit a consequence of deficient memory trace formation (e.g., slower encoding of auditory trace) (Chapter 8)?

Moreover, the experimental data will explore two different language impaired groups: adolescents and adults with 1) SLI and 2) dyslexia –and their typically developing controls matched on chronological age (CA). These three groups provide us with

information on, firstly, if SLI and dyslexia arise from common input-processing deficit and, secondly, if these language impaired groups show age-appropriate, delayed or deviant input-processing capacity.

The thesis is structured as follows. Chapter 6 investigates how individuals with SLI and dyslexia use different acoustic cues in determining the voiced-voiceless consonant distinction in English. In Chapter 7, I establish the nature of the input-processing in SLI, dyslexia and age-matched typically developing controls via behavioural and electrophysiological experiments using speech and non-speech stimuli to ascertain whether they exhibit normal or impaired processing. Finally, Chapter 8 investigates the formation of memory traces in SLI and dyslexia. In Chapter 9, I summarise my findings and how they relate to the research questions presented above and fit into the theoretical framework on SLI, dyslexia and speech perception introduced in Chapters 2, 3 and 4.

The studies in this thesis primarily took place at a single testing session. Due to time constraints not all individuals managed to complete all experiments. In Appendix A_1 , I list the individuals who undertook each task and in each chapter I detail the characteristics of just the group of SLI and dyslexic individuals who took part in that particular study.

5.1 Participant selection in the current thesis

Individuals with SLI used in this thesis were selected from a larger group of adolescents and adults who have been taking part in research projects conducted at the Centre for Developmental Language Disorders and Cognitive Neuroscience (CDLDCN) over the past years.

The SLI group at the CDLDCN were originally recruited from residential language schools or from language units within schools with the help of speech and language therapists (SLTs) and Educational Psychologists. These children are diagnosed as having severe difficulties with language despite normal hearing, normal articulation and normal

non-verbal IQ (i.e., IQ above 85). Only those children with English as a first language, and without a diagnosis of autistic spectrum disorders are selected.

The selection of SLI participants at CDLDCN is done in two phases. The selection is based, firstly, on their performance on standardised language tests including Test for Reception of Grammar (TROG), British Picture Vocabulary Scales (BPVS), Test of Word-Finding (TWF) and Clinical Evaluation of Language Fundamentals (CELF). The child must have a score that is > 1.5 SD below the population mean on at least one of the standardised language tests. Secondly, after obtaining scores in the standardised tests, a series of non-standardised tests to assess specific aspects of grammar are administered. These are devised by van der Lely and they target areas of grammar such as **verb agreement and tense** (Verb Agreement and Tense Test, VATT), **reversible passives** (Test of Active and Passive Sentences, TAPS-R) and **pronominal reference** (Advanced Syntactic Test of Pronominal Reference, A-STOP-R).

The Verb Agreement and Tense Test is a test of English morpho-syntax. More specifically, the test looks at the production of tense and agreement in a sentence completion task (e.g., *Buzz is trying to fly. Everyday Buzz_____. Yesterday Buzz_____.* Where the child should produce <u>tries-tried</u> to fly). This test also taps children's ability to produce a correct form of the past tense (i.e., *gave* instead of **gived*) and whether there are any differences between regular and irregular verb forms (e.g., verbs such as *try-tried* and *give-gave*).

The Test of Active and Passive Sentences is a test of syntax. The test is a picture pointing task that investigates whether a child can use syntax to distinguish "who does what to whom" in active and passive sentences (i.e., who is the actor and who is the recipient in sentences such as *The man eats the fish* and *The man is eaten by the fish*.). The test consists of 48 sentences classified in four ways: 1) active 2) long passive 3) short passive and 4) short ambiguous sentences. (G-)SLI children have been reported to show difficulties with long and short passive sentences and preference to adjectival state

over verbal state in their picture response in the short ambiguous passive sentences (van der Lely, 1996).

The Advanced Syntactic Test of Pronominal Reference, in turn, is a test of syntactic knowledge of pronouns and reflexives (e.g., *her/herself*). The child is asked to evaluate if the sentence matches a picture in sentences e.g., *The wolf says the boy is tickling himself* with any of the three possible pictures: 1) boy tickling himself, 2) boy ticking the wolf, 3) wolf tickling himself. The comprehension of these sentences requires syntactic knowledge of the grammatical constraints for reflexives.

Overall, these tests tap specific aspects of English syntax and morpho-syntax and the grammatically impaired (G-)SLI children usually make 20% or more errors on each specific test where as typically developing children rarely make any errors after about 6 years of age (van der Lely, 1996; van der Lely & Stollwerck, 1997; see section on G-SLI in Chapter 2).

However, research has shown that the clinical profiles can vary throughout the child's development and, furthermore, the intensive therapy these children receive in language schools or language units can have an effect on their language profiles when tested again later in life (Bishop, 1997). In the current thesis, the individuals with SLI were between 14 and 25 years of age at the time of experimental testing. Therefore, the core language tests (TROG-2, BPVS-II, VATT, TAPS-R, A-STOP-R) and nonverbal IQ test (Ravens Progressive Matrices, RPM) administered (approximately) more than 18 months ago were re-administered prior the experimental testing (see their individual scores in Table 5.1 below).

As seen in Table 5.1, some of the individuals with SLI still score below the 1.5 SD in tests that tap different aspects of grammar. However, it is clear that not all individuals necessarily meet the pre-defined criteria for SLI when tested at a later age. This may be due to a few factors. Firstly, most of these tests used here are standardised only up to 15

or 16 years of age (16;11 for TROG-2 [17;0-64;0 for adults]; 15;08 for BPVS-II and 15;08 for RPM). Secondly, the majority of the SLI individuals taking part in the study had received intensive therapy and training in special schools therefore affecting their performance on some of these tests. However, all of these children have been taking part in the ongoing research at the CDLDCN and when they were tested earlier in life all these individuals met the criteria for SLI. Moreover, when looking their current scores on the more specific grammar tests such as VATT, TAPS and A-STOP, the individuals with SLI tend to still score lower than they score on the standardised language tests (for more information about the development of their language profiles, see Gallon 2007).

As already mentioned earlier in Chapter 2, the issue of a purely grammar impaired subgroup of SLI is still largely debatable. In the current thesis, however, the majority of participants were selected from the G-SLI subgroup but as they all did not fall into this subgroup and the existence of this subgroup is largely a theoretical issue which is not addressed in this work, I use the general term SLI throughout the thesis.

Table 5.1: Selection criteria for the SLI group (N=13)

		RAVEN	TROG-2	BPVS
ID	Age	Raw SS z	Raw SS z	Raw SS z
S010	15;06	42 90 -0.73	17 95 -0.33	110 77 -1.53
S013	18;04	41 88 -0.80	11 67 -2.20	102 69 -2.07
S015	14;06	45 95 -0.33	16 97 -0.20	84 65 -2.33
S017	21;05	45 96 -0.27	17 95 -0.33	115 81 -1.27
S024	17;02	48 103 0.20	13 76 -1.60	105 72 -1.87
S025	14;10	47 101 0.07	12 71 -1.93	96 70 -2.00
S031	25;00	56 123 1.53	15 85 -1.00	144 122 1.47
S032	24;06	42 90 -0.73	12 71 -1.93	101 68 -2.13
S049	14;00	39 87 -0.87	10 69 -2.01	82 65 -2.33
S067	13;00	45 96 -0.27	14 88 -0.80	109 90 -0.67
S071	15;03	49 104 0.27	14 81 -1.27	103 72 -1.87
S112	15;06	50 106 0.40	15 85 -1.00	105 72 -1.87
S116	19;02	60 135 2.33	17 95 -0.33	147 128 1.87
Mean	17;06	46.85 101.08 0.06	14.08 82.69 -1.15	107.92 80.84 -1.28
SD	3;96	5.96 14.06 0.95	2.36 11.00 0.73	19.14 20.77 1.39
Range	13;00-25;00	39-60 87-135 -0.9-2.33	10-17 67-97 -2.20.2	82-147 65-128 -2.33- 1.8

Continued on the next page

		VATT	TAPS-R	A-STOP-R ¹	A-STOP-R ²
ID	Age	(% correct)	(% correct)	(% correct)	(% correct)
S010	15;06	80	69	69	58
S013	18;04	5	48	73	63
S015	14;06	30	46	84	88
S017	21;05	73	65	86	75
S024	17;02	38	67	n/a	n/a
S025	14;10	65	40	67	46
S031	25;00	88	73	90	83
S032	24;06	48	75	71	67
S049	14;00	3	46	85	90
S067	13;00	n/a	58	72	63
S071	15;03	75	52	44	25
S112	15;06	35	73	92	92
S116	19;02	98	69	96	96
Mean	17;06	52	57	78	71
SD	3;96	32	16	15	22
Range	13;00-25;00	3-98	23-75	44-96	25-96

 $^{^{1}}$ Average of two experimental condition: Semantic Mismatch and Syntactic Mismatch 2 Syntactic Mismatch score alone

Dyslexic participants were recruited via University College London (UCL) mailing lists, Dyslexia Action and via local advertisements (e.g., at the London Academy of Music and Dramatic Art). They all had received a formal diagnosis of developmental dyslexia by an educational psychologist and all had history of reading difficulties. All participants were interviewed (see Appendix A₂) and those showing history of other disorders (e.g., autism, ADHD, SLI) were excluded from the group. Moreover, due to high comorbidity rates between SLI and dyslexia, two standardised language measures (TROG-2 and BPVS-II) were administered.

Age and IQ matched controls were recruited via UCL mailing lists and from DLDCN participant database. Non-matched adult controls who took part in the behavioural testing were recruited from UCL mailing lists (Cue-weight study) and from a commercial website (www.gumtree.co.uk; the discrimination study). All subjects (or their legal representatives if under 18 years of age) gave informed consent to participate in the study. See also Appendix A_2 for participant background screening questions.

5.2 Stimulus selection in the current thesis

The thesis is divided into three experimental chapters consisting of two behavioural studies [Chapter 6: Cue weighting (Exp 1) and Chapter 7: Discrimination (Exp 2)] and one ERP study that is analysed using two different methods [Chapters 7 (Exp 2) and 8 (Exp 3)]. All experiments exploit the same CVC syllables [bɒt]-[bɔ:d] both of which follow the phonotactic rules of British English and are non-words. These non-word stimuli were selected because 1) The perception of the acoustic features contributing to the syllable-final stop consonant voicing has been previously studied with similar stimuli in American English by Nittrouer (2004) and Crowther & Mann (1994) in TD children and adults (but not in SLI or dyslexia) 2) instead of more commonly used syllable-initial contrasts (e.g., $[b\Lambda]-[d\Lambda]-[g\Lambda]$ or $[b\Lambda]-[p^h\Lambda]$), the aim was to create a more challenging or perceptually less salient contrast syllable-finally where the consonantal part is masked by the preceding vowel. In Experiment 1, two stimuli continua were created varying the F1 offset frequency ('high' or 'low') and the vocalic duration (100-220 ms, in 20 ms

steps). In subsequent Experiments, four stimuli were selected (out of 14 created for Exp 1), where two represented typical exemplars in British English ([bɒt]-[bɔ:d]) and two atypical versions of these non-words (see Chapter 6). Moreover, in addition to speech stimuli, complex non-speech control stimuli (sinewave speech analogues, SWS analogues) were used in Experiments 2 and 3. All stimuli were English non-words because there is some indication that lexical information can bias categorisation and the lexical status of the deviants can affect the MMN amplitude (see Pulvermuller, 2001; van Linden, Stekelenburg, Tuomainen, & Vroomen, 2007 for lexical effects and Jacobsen et al., 2004 for opposite results).

Prior to the experiments reported in Chapters 6-8, pilot testing was conducted by using five native British English speakers (either members of staff or students at UCL) as participants. Pilot testing consisted of an identification experiment (2-AFC task, see Chapter 6) and a "same-different" discrimination task (AX-task) and revealed that the endpoint exemplars can be reliably identified and are easily discriminated. The task instructions are presented in Appendix B and C. In addition to the experimental testing, a set of language (grammar and vocabulary) and IQ tests were conducted either at the time of or prior to the experimental testing.

6. Cue weighting in Specific Language Impairment (SLI) and dyslexia

6.1 Introduction

As established in previous chapters, the speech signal contains different speech patterns some of which are acoustic cues to phonetic contrast (such as in place, manner, voicing). These acoustic cues occur simultaneously and change rapidly as a function of time. Listeners, however, do not necessarily make equal use of all available cues in all situations, that is, listeners can perceptually *weight* these cues to different degree. Moreover, listeners are able to change the cue they give more relative weight to when the circumstances change (e.g., noise is added, Wardrip-Fruin, 1985) or when the task demands change (Gordon, Ebenhardt & Rueckl, 1993). Furthermore, it has been suggested that children and adults weight some acoustic cues differently in identifying certain speech contrasts (Nittrouer, Manning & Meyer, 1993; Sussman, 2001; Nittrouer & Lowenstein, 2009).

Despite the fact that a great deal of studies on auditory and speech processing in developmental language disorders have focused on discrimination of speech contrast or on discrimination of simple tones, some studies have also investigated the labelling accuracy (see Chapters 2 and 3). To date, however, the results have been somewhat contradictory. In an attempt to account for these sometimes contradictory findings in auditory and speech processing in SLI, Leonard (1998:276) suggested that instead of a general deficit in 'rapid auditory processing', the underlying impairment in SLI (or in some children with SLI) could, in fact, be an inability to focus on those acoustic cues that are relevant for their native language (NL) speech sound categorisation. Furthermore, to account for the underlying impairment(s) in dyslexia where the existence of phonological deficits is well documented, it has been suggested that the core deficit lies at the representational level. In other words, it has been argued that, in dyslexia, phonological representations are 'noisy' or 'inaccurate' leading to failures to

accurately categorise their native language speech sounds that would, in turn, cause phonological deficits and reading difficulties (however, see discussion in Ramus & Szenkovitch, 2008). Moreover, as established in Chapter 4, in addition to the search for underlying deficits in SLI or dyslexia, the relationship between these two developmental disorders has been under debate recently. One influential account, namely the auditory temporal processing deficit hypothesis by Tallal and colleagues (Tallal & Piercy, 1973; Tallal, 1980; Tallal et al. 1996), argues that both SLI and dyslexia stem from a similar underlying auditory processing deficit differing only in the degree of severity. This hypothesis has evoked substantial amount of research producing evidence for and against this single-source model (see Rosen 2003 for a critical review).

The present study investigates, firstly, whether adolescents with SLI make equal use of available acoustic cues as their matched controls in perceiving the syllable final stop consonant voicing contrast ([t]-[d], see Chapter 5 for rationale). Secondly, the study investigates if individuals with dyslexia show a categorical perception (CP) deficit. The third aim is to examine the locus and nature of the possible input-processing problem in SLI and dyslexia by investigating if these two groups that show distinct patterns of linguistic problems nevertheless share a similar underlying speech processing problem (see Chapters 2 and 3 for theoretical discussion).

On the basis of previous findings, I predict that:

- 1) If the underlying phonological impairment in SLI is the inability to attend to those acoustic cues that are important for NL phoneme contrasts, SLI group should weight the NL acoustic cues differently, i.e., show differences in phoneme boundary measures, in the steepness of the categorization functions and in boundary/slope separation measures.
- 2) If dyslexia is caused by a CP deficit (e.g., their NL phoneme categories are 'noisier' or less categorical), they should, overall, show shallower categorization functions than

the control group and more within-category sensitivity than their typically developing peers.

3) If subgroups of SLI and/or dyslexic individuals show speech perception deficits, a larger proportion of individuals with SLI and/or dyslexia should show 'impaired' performance.

6.2 Method

6.2.1 Participants

The participants in this study consisted of a group of young adults with SLI, dyslexia and a group of matched control subjects (see details of participants in Table 6.1). Moreover, to test for the adult performance, a group of non-matched adults was pretested in this task.

In the language impaired groups, 13 young adults with SLI aged between 14;00 and 25;00 (9 males, the selection criteria for these individuals are detailed in Chapter 5) and 12 young adults with dyslexia (8 males) aged between 14;09 and 24;06 participated. In total, 25 individuals were used as controls and they were split into two groups. One group was matched with SLI and dyslexia on chronological age (CA), non-verbal IQ, gender and handedness (10 males, between 15;05-25;01 years). Because some of the younger participants in the SLI, dyslexia and CA groups were anticipated to find the identification task difficult, to establish the adult performance, the second control group consisted of 13 non-matched adults (3 males, between 18;00-36;00 years, mean 25;08 years). All participants were native British English speakers who were neurologically healthy, right-handed and all reported normal hearing.

Table 6.1: Summary of group matching details.

	Groups		
	SLI n=13	DYS n=12	CA Control n=12
AGE			
Mean (years)	18;03*	19;05	19;08
Range	14;00-25;00	14;09-24;06	15;05-25;01
(SD)	3;4	3;7	3;5
RAVENS			
Raw	47	50	50
Range	39-60	44-57	42-56
(SD)	(6.0)	(4.0)	(5.4)
SS	101	108	109
Z-Score	0.06	0.45	0.61
TROG-2			
Raw	14.1 ^a	18.6 ^a	n/a
Range	10-17	17-20	n/a
(SD)	(2.4)	(1.1)	n/a
SS	82.7	102	n/a
Z-Score	-1.15	0.11	n/a
BPVS			
Raw	108 ^b	138.6 b	n/a
Range	82-147	121-155	n/a
(SD)	(19.1)	(12.8)	n/a
SS	80.8	116.1	n/a
Z-Score	-1.28	1.10	n/a

^{*} at the time of ID experiment a,b p < .01

The three groups' (SLI, dyslexia and CA) chronological age did not differ significantly [F(2,36)=0.392, p=.679] neither did their non-verbal IQ scores [F(2,36)=1.297, p=.286] nor their gender distribution (Fisher's exact, p=.722). However, to establish that, in our

study, individuals with dyslexia do not have co-occurring language impairments, their performance on TROG-2 and BPVS-II was assessed. All individuals with dyslexia had normal language skills and their scores on TROG-2 and BPVS-II differed significantly from those of SLI participants [TROG-2: t(20)=-4.706, p<.001, BPVS-II: t(19)=-4.467, p=.002, see Table 6.1 for group scores].

6.2.2 Stimuli

The phonetic contrasts used in this study were adapted from previous literature (Flege, Munro, & Skelton, 1992; Crowther & Mann, 1994; Nittrouer, 2004). Two continua of synthetic speech sounds were created for English syllable final stop consonant voicing, more precisely for the plosives [t] and [d]. The synthesis parameters closely resembled those used by Crowther & Mann (1992) and Nittrouer (2004). However, because both these studies used the syllable final /t-d/ contrast in American English subjects, some parameters, such as duration of the vocalic portion and vowel formant frequencies, were adjusted for British English in the present experiment.

The voiced-voiceless consonant contrast in English differs phonologically only by one feature [voice]. In phonology, this laryngeal feature [voice] has only binary values, that is, it is either "on" or "off" ([+voice] or [-voice]). In English, this phonological feature is phonetically implemented, for example, in the duration of the preceding vocalic element and in the offset frequency of the first formant (F1) in syllable final position. In other words, in syllable final voiced stop consonants, the preceding vowel is longer and the F1 offset frequency is lower than in voiceless consonants (Wolf, 1978).

To account for these acoustic-phonetic voicing features, two synthetic continua for English non-words [bɒt] and [bɔ:d] were created by using the Klatt-type cascade-parallel formant synthesizer within the High-Level Speech Synthesizer (HLsyn, Sensimetrics Inc., 1.0). This synthesis method allows the user to control two sets of parameters: constant and dynamic. During the synthesis, only the dynamic parameters (e.g., voicing,

F1 and F2) were manipulated as a function of time whereas the constant parameters (such as higher formant frequencies and bandwidths) were kept at their default values.

I adopted a traditional cue-weighting setup where one acoustic parameter is fixed and the other parameter varies continuously. In the present experiment, the fixed property is the F1 (either "high" or "low", i.e., 250 Hz or 570 Hz) and the continuously varying parameter is the duration of the vocalic portion (i.e., the duration of the steady-state vowel *and* the formant transition phase). The vocalic duration changed from 100 ms to 220 ms in 20 ms steps. In other words, the two continua ("high" and "low") each contained seven different durations, i.e., there were 14 stimuli in total (see Figure 6.1 for continua end point stimuli).

In the synthesis, all vocalic portions were preceded by 50 ms of silence (signalling the initial stop consonant [b]) during which the amplitude of voicing (AV) parameter was interpolated from 40 to 60 dB. The fundamental frequency (F0) was set to increase from 100-130 Hz during the initial 50 ms after which it linearly decreased to 95 Hz to imitate a natural pitch contour in speech. In the voiceless token (/bot/) the F1 frequency was set to a constant value of 570 Hz throughout the syllable. The voiced consonant /d/ was created by lowering the offset frequency of F1 to 250 Hz during the final 50 ms. The F2 frequency was set to 1000 Hz, rising to 1500 Hz during the final 50 ms. The F3 frequency was kept at a constant value of 2650 Hz. All higher formant frequencies (F4, F5, F6) were kept in the HLsyn default values (3500, 4500, 4990 Hz respectively). Finally, a 15 ms linear onset and offset ramp was used to remove clicks and the amplitudes were normalized (rms -10 dB of the maximum amplitude) in all stimuli by using CoolEdit96. All formant frequencies were checked using Praat (4.4.16, Boersma & Weenink, 2006).

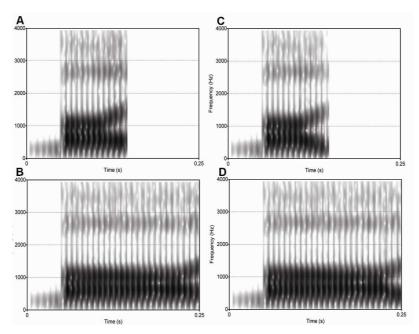


Figure 6.1: Continua endpoint stimuli. A: High100, B: High220, C: Low100, D: Low220.

6.2.3 Procedure

In this study, two continua of seven synthetic stimuli are used, that is, F1 is either "high" as in voiceless consonants (continuum 1) or "low", as in voiced consonants (continuum 2). The 14 stimuli are played 10 times in a pseudo-random order in a two-alternative forced choice task (2-AFC). The stimuli are played at a laptop computer one sound at a time via earphones (Sennheiser) at a comfortable level, and the subjects are asked to identify the stimulus as English non-words "bot" or "bod" by pressing a relevant key in the keyboard (SOA 1000 ms, total 140 stimuli, see Appendix B). In the keyboard, the "z" and "m" keys were labelled with stickers as "bot" and "bod". A short practice session (15 stimuli, presented in a fixed order) preceded the actual experiment to establish that participants hear the stimuli as "bot" and "bod" and were able to associate the sounds with the relevant keys. All participants received a written instruction followed by an oral explanation and the practice session (see instructions in the Appendix B). The identification task took approximately 5-7 minutes to complete.

6.3 Results

In the identification data, the percentage of [bo:d] responses to each stimulus was calculated (see Figure 6.2). The identification data from synthetic [bpt] - [bp:d] continua were fitted using a probit transformation (Cohen & Cohen, 1983) that gives an estimation of the Point of Subjective Equivalence (PSE, i.e., the 'category boundary') and the slope of the categorization function (i.e., the 'categoricality' of the perception) (see Chapter 4 for categorical perception). Moreover, as in the study of Nittrouer (2004), weight assigned to F1 offset (i.e., the boundary separation between the two continua) and to vocalic duration (i.e., the mean slope values) were estimated. The boundary separation value was calculated from the absolute boundary mean values (i.e., boundary_{highF1}-boundary_{lowF1}). This value indicates how much category boundary placement is affected by the formant transition ("high" or "low") in that the greater the separation value, the greater the weight assigned to formant transition cue (see Table 6.2). Mean slope values were calculated across the two functions and it, in turn, indicates how much participants weighted vocalic duration, i.e., the steeper the functions are (=bigger the slope value), more weight is assigned to duration cue (see Table 6.3). These variables were then subjected to ANOVAs. All statistical group-level comparisons were done between three groups: SLI, dyslexia and CA controls. Power analyses were only conducted after the study (Post Hoc) by using G*Power 3 software (F-test, α =0.05, power=0.8).

The data from three subjects in the dyslexia group and one subject in the CA group were excluded from the analyses because they could not complete the task or label the sounds. All four participants that were excluded failed to perceive the sounds categorically (i.e., the probit transformation failed to estimate the slope value). Moreover, all of them categorised the sounds randomly most likely due to motivational issues (i.e. they either pressed only one button throughout the experiment or pressed the two buttons alternately). The data from the remaining 46 subjects are illustrated in Figure 6.2.

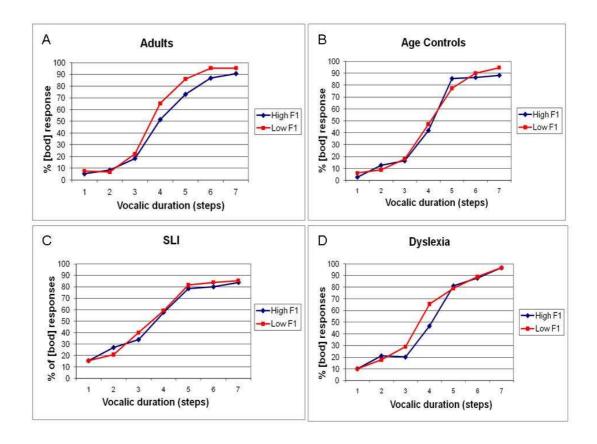


Figure 6.2: Labelling functions of synthetic [bot] - [bo:d] continua in A) non-matched adults (n=13), B) matched controls (n=11), C) SLI (n=13), D) dyslexia (n= 9). X-axis represents the vocalic duration by step number (steps 1-7, i.e., 100-220 ms) and y-axis the % of [bo:d] responses.

In category boundary measures, repeated measures ANOVA showed no significant main effects or interactions [Condition: High/Low F1: F(1,30)= .209, p=.651, η_p^2 =.007, NS; Group*Condition F(2,30)=1.229, p=.228, η_p^2 =.080, NS; Group F(2,30)= 1.586, p=.221, η_p^2 =.096, NS, see Table 6.2]. The Post Hoc power calculations revealed that 96 subjects would be needed for significant main effect of group.

In similar fashion, no significant effects were found in one-way ANOVA for the boundary separation value (i.e., the weight assigned to F1 transition) [F(2,30)=1.301, p=.287, NS].

Table 6.2: Category boundary values for two continua (High F1 and Low F1) and the relative weight assigned to F1 transition. Values represent the step number (seven different durations between 100-220 ms, i.e., steps 1-7). Standard deviations (SDs) are given in parenthesis.

	High F1	Low F1	Weight assigned to F1
CA	4.17 (0.70)	4.05 (0.50)	0.12 (0.59)
SLI	3.47 (1.23)	3.69 (0.76)	-0.22 (0.89)
DYS	3.79 (0.90)	3.50 (0.86)	0.29 (0.80)
Total	3.81 (0.94)	3.75 (0.71)	0.06 (0.76)
Adults	4.39 (0.76)	3.64 (0.51)	0.75 (0.88) ³

Furthermore, no significant effects were found for the slope values [Condition: High/Low F1: F(1,30)=.012, p=.915, $\eta_p^2=<.001$, NS; Group*Condition F(2,30)=1.906, p=.116, $\eta_p^2=.113$, NS. The main effect of Group approached statistical significance $[F(2,30)=2.519, p=.097, \eta_p^2=.144, see Table 6.3]$. The Post Hoc power calculations revealed that 36 subjects would be needed for significant main effect of group.

Table 6.3: Slope values for labelling functions and the relative weight assigned to vocalic duration (mean of the slopes). Standard deviations (SDs) are given in parenthesis.

	High F1	Low F1	Weight assigned to duration
CA	0.71 (0.28)	0.75 (0.30)	0.73 (0.29)
SLI	0.44 (0.20)	0.57 (0.48)	0.51 (0.32)
DYS	0.84 (0.52)	0.69 (0.21)	0.77 (0.34)
Total	0.66 (0.33)	0.67 (0.33)	0.67 (0.32)
Adults	0.88 (0.56)	1.10 (0.67)	0.99 (0.62) 4

³ Paired sample t-test showed that the category boundaries between High and Low continua differed significantly in non-matched adult group [t(12)=3.054, p=.010]

⁴ Slope values between High and Low continua did not differ significantly in non-matched adult group [t(12)=-1.420, p=.181, NS].

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Post Hoc analyses (pairwise comparisons, LSD) showed that the marginal main effect of group was due to SLI group's performance marginally differing from CA (p=.079) and from dyslexia (p=.058) groups.

6.3.1 Correlations

As established in the previous literature, phoneme boundary sharpening still takes place during the second decade of life (Hazan & Barrett, 2000), continuing even late into adolescence (Flege & Eefting, 1986). In current experiment, the youngest participant was only 14 years of age, therefore, in order to see whether the age of the participant had an effect on the performance in the experimental task, correlations between dependent variables and age were calculated. However, the results showed that, in this sample, age did not correlate with any of the experimental conditions (all p-values > .10).

Moreover, in SLI and dyslexia, the relationship between different phonological measures (i.e., the 'categoricality' of perception and phoneme boundary) and standardized grammatical (TROG-2) and vocabulary measures (BPVS-II) was investigated (see Figure 6.3). In SLI, the results showed that while the TROG score did not correlate with either boundary or slope measures (p>.05), the BPVS-II score was significantly correlated with the slope measure (r=.806, p=.001, n=13) accounting for up to 65% of the variation (see Figure 6.3). However, this significant correlation was due to one participant scoring high on the slope and BPVS-II (see Figure 6.3). After removing that data, the effect was no longer statistically significant (r=.427, p=.166, n=12), i.e., accounting for up to 18% of the variation. Furthermore, no significant correlations were found in dyslexics (p>.10).

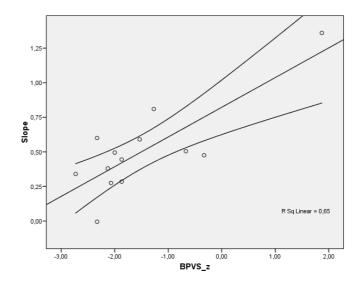


Figure 6.3. Correlation between BPVS-II (z-score) and slope in SLI group.

6.3.2 Individual data and within-category variation

Due to the commonly acknowledged cognitive heterogeneity within SLI and dyslexia and to the comorbidity of these two disorders, previous research has demonstrated that when investigating the underlying causes of SLI and dyslexia one should, in addition to the group performance, also address the individual performance (see e.g., Ramus et al. 2003; Ramus, 2003; White et al., 2006).

The distribution of individual scores in both boundary and slope values is demonstrated in Figures 6.4-6.6. Individual differences were studied by indentifying abnormally low scores following the procedure used by Ramus et al. (2003) and White et al. (2006). In other words, the normal performance is estimated on the basis of control group scores. In the study by White and colleagues (2006), any control score exceeding -1.65 SD of the control mean was removed and a new control mean was calculated. Outliers were defined as those performing below 1.65 of this new control mean (i.e., those in the bottom 5th percentile). In the current experiment, however, all control participants where within the selected cut-off value (with the exception of one CA participant scoring below the cut-off in boundary separation measure).

Figures 6.4-6.6 show individual scores for SLI, CA and dyslexia groups in phoneme boundary and steepness of the slope conditions. The cut-off value of 1.65 below CA mean is shown by a dashed line, that is, the participants who score below this are considered outliers. Furthermore, the individual identification functions are plotted in Figure 6.7.

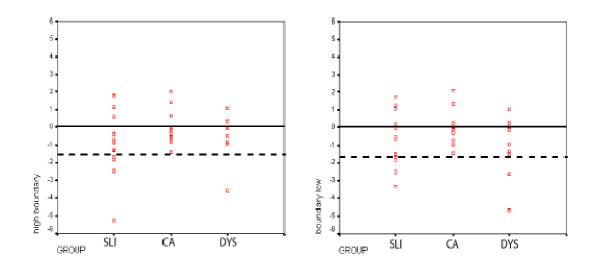


Figure 6.4 Individual performance for boundary measure (High and Low continua) in SLI, CA matched and dyslexic participants. The y-axis values are z-scores, control mean is shown by continuous line and -1.65 SD cut-off from the control mean by dashed line.

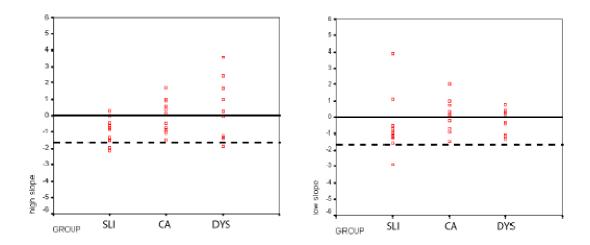


Figure 6.5 Individual performance for slope measure (High and Low continua) in SLI, CA matched and dyslexic participants. The y-axis values are z-scores, control mean is shown by continuous line and -1.65 SD cut-off from the control mean by dashed line.

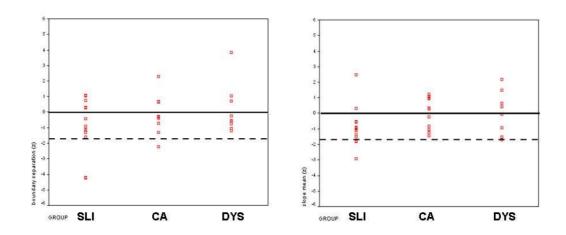


Figure 6.6 Individual performance for boundary and slope separation measures in SLI, CA matched and dyslexic participants. The y-axis values are z-scores, control mean is shown by continuous line and -1.65 SD cut-off from the control mean by dashed line

Only one participant in the SLI group performed below norm in all conditions (participant S013, see Chapter 5 for details). Two participants had both (high and low) boundary values below norm (S031 and S032), one participant had both (high and low) slope values and High boundary value below norm (S015) and one participant scored below norm in Low boundary condition (S010). None of the dyslexic participants had

values below the norm in all four conditions and only one participant had values below the norm in both boundary conditions. Overall, 31% of SLI were outliers in boundary condition (31% in High and Low boundary) and 12% of SLI were outliers in slope condition (15% in High slope and 8% in Low slope). Among dyslexics 17% (11% in High boundary and 22% in Low boundary) were outliers in boundary condition and only 6% (11% in High slope and no outliers in Low slope) in slope condition. In terms of boundary separation value 15% of SLI and 9% of CA performed below norm, the corresponding figures for slope mean being: SLI 23% and dyslexia 11%. However, due to a small sample size and the fact that the distributions of scores were largely overlapping between all three groups, there is no evidence of any significant subgroups that would show impairments on any of the CP measures used in the present experiment.

Several recent studies (Serniclaes et al. 2001; Serniclaes et al. 2004; Bogliotti, Serniclaes, Messaoud-Galusi, & Sprenger-Charolles, 2008) suggest that the core deficit in dyslexia is the preserved sensitivity to within category differences ('allophonic detail'). However, Serniclaes and colleagues base their argument for enhanced perception of allophonic detail on discrimination data whereas the current study quantifies the within category variation in identification data. In order to investigate the variation in the identification data, the stability of categorization (i.e., the identification performance in the continuum end-points that fall outside the category boundary) was compared between groups (see Figure 6.7 and Table 6.4).

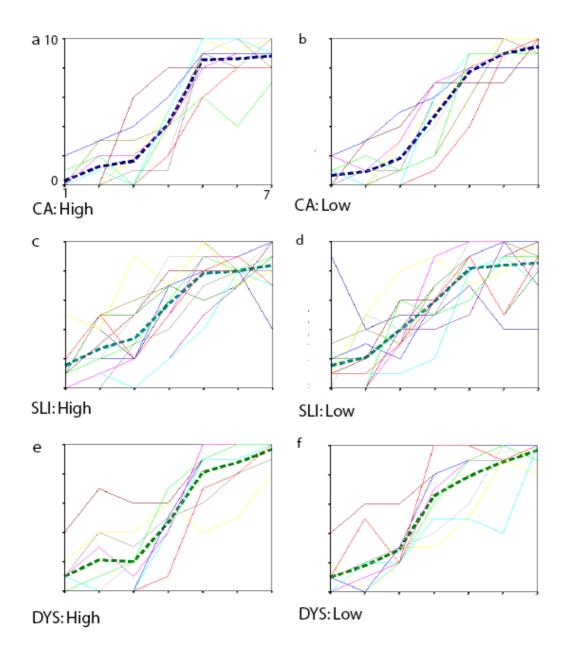


Figure 6.7. Individual identification functions in CA (a and b), SLI (c and d) and dyslexia (e and f). The stimulus number is represented on x-axis (1-7) and the number of [bɔ:d] answers (out of 10) at the y-axis. Responses to 'High' are on the left-hand side and 'Low' on the right-hand side. Group mean is shown by the dashed line.

Statistical analyses were performed for the mean value of category endpoints (i.e., the mean of stimuli numbered 1 & 2 and 6 & 7, labelled as 'position' A and B respectively).

As expected, 2(Position: A,B) x 2(Frequency: High,Low) Repeated measures ANOVA showed a significant main effect of **Position** [F(1,30)=414.014, p<.001, η_p^2 =.932, see Table 6.4]. No other significant main effects or interactions were found.

Table 6.4. Percentage of [bo:d] responses (SD) for continua (High and Low) endpoints (Position A and B) for Controls, SLI and dyslexics.

Group	High-Pos _A	High-Pos _B	Low-Pos _A	Low-Pos _B
CA	8 (8)	87 (12)	8 (8)	92 (6)
SLI	21 (16)	82 (9)	18 (18)	85 (17)
DYS	16 (19)	92 (10)	18 (18)	93 (10)
Total	15 (14)	87 (10)	15 (15)	90 (11)

6.4 Discussion and conclusion

The first objective of this study was to investigate whether young adults with SLI assign weight to the same acoustic cues as their typically developing controls. The present study contrasted two acoustic cues (vocalic duration and F1 offset frequency) that both play a role in English syllable final plosive voicing but which differ in their spectral properties. Overall, the current experiment found no evidence that individuals with SLI would make use of different acoustic cues than their typically developing controls in the given context. The results showed that, similar to matched controls, adolescents with SLI base their judgement of syllable final consonantal voicing more on vocalic duration than on F1 offset frequency. However, there was a marginal statistical trend indicating that the SLI group used the duration cue less than both the controls and dyslexics. Moreover, there was no evidence that a general inability to make use of available acoustic cues would be causally related to dyslexia. In short, all three groups weighted the vocalic duration the most in determining the voicing status of the syllable final stop consonant. Interestingly, however, contradicting with the results by Nittrouer (1994), non-matched adults used both the durational and frequency cue in syllable final [t]-[d] voicing judgement. One possible explanation for the differential behaviour between nonmatched adults and the control group (that consists of adolescents and adults) is that it reflects subtle age effects on development of categorical perception as reported by Hazan and Barrett (2000). However, the current results showed no correlations between age and any of the experimental conditions.

The second objective of this study was to investigate the claim that dyslexics show deficits in categorical perception. The results showed that dyslexics did not differ from controls in any CP measure (e.g., category boundary placement or steepness of the slope). Moreover, when looking at the within-category variation in phoneme identification performance, the dyslexic group did not show any evidence of unstable categorization. Furthermore, individual data showed no evidence of any *significant* subgroups that would show speech perception deficits in the current sample.

However, when looking at the proportion of individuals who score below the cut-off value and when looking at the categoricality of the perception (indicated by the slope value) it is clear that, firstly, in the SLI group a larger proportion of participants are classified as outliers and, secondly, their labelling performance shows a trend of being less categorical. This could, in principle, indicate that a very small proportion of the SLI group show subtle speech perception deficits. This matter, however, is properly addressed only by increasing the sample size.

On evaluating current results on cue-weighting and categorical perception in SLI and dyslexia, several factors need to be considered: first of all, the present task involved only purely synthetic and schematized nonwords and the naturalness of the stimuli can have an effect on the labelling performance (see Blomert & Mitterer, 2004). Secondly, the number of available acoustic cues was limited (i.e., only duration and frequency cues) and they were not necessarily the ideal cues for this percept for all participants possibly causing more variability in labelling data (Hazan & Rosen, 1991). Lastly, the task involved labelling 140 stimuli with no feedback or game embedded to the task. Therefore, further research that would include synthetic, semi-synthetic and natural

tokens embedded in real words and non-words in a more engaging task (thus reducing the degree of uncertainty) is required.

7. Electrophysiological investigation of auditory processing in SLI and dyslexia

7.1 Introduction

7.1.1 Chapter outline

In this chapter I investigate if individuals with SLI and dyslexia show auditory input-processing deficits. I begin in Section 7.1.2 by summarizing behavioural and electrophysiological studies on auditory and speech processing in SLI and dyslexia. In Section 7.1.3 I discuss the advantages of the methodologies chosen for the present thesis and to what extent they can complement the previous studies on auditory processing. In Sections 7.2 and 7.3 I present the methods, and in 7.4 the results. In 7.5 I summarize the findings and discuss how the data fit into the current theories on SLI and dyslexia.

7.1.2 Auditory processing in SLI and dyslexia

The input-processing deficit model of developmental language impairments (SLI and dyslexia) as introduced by Tallal and her colleagues (see Sections 2.2.4, 2.4.2.2 and 3.2.2.1) has evoked an extensive amount of research during recent decades. This research has been conducted using various techniques ranging from different behavioural detection sensitivity measures to electrophysiological and brain imaging techniques. The methods, however, have been rather variable and the results fairly contradictory (see e.g., Rosen, 2003; Bishop, 2007 for reviews). With respect to behavioural data, to account for these inconsistent results, a common trend recently has been to investigate individual data.

In this fashion, Rosen, van der Lely, & Dry (1997) investigated two teenage boys with different disorders and language profiles: one ('AZ') with G-SLI and one ('W') with a Landau-Kleffner syndrome. Whereas AZ has severe language impairment but normal non-verbal IQ, W suffered from a neurological disorder acquired in childhood. This

neurological disorder is characterized by gradual development of aphasia and an abnormal EEG resulting in language problems and even seizures in some individuals. In their study, Rosen et al. (1997) investigated the input-processing abilities of these two boys with causally different language impairments by using both auditory and speech processing tasks. Rosen and colleagues (1997) adopted a Tallalian temporal order judgement task (Tallal & Piercy, 1973) and a backward masking task (Wright et al. 1997) that have previously been claimed to distinguish language impaired children from typically developing children. In addition to these auditory measures, Rosen and colleagues investigated the performance of AZ and W in same-different word discrimination task using minimally different stimuli such as <bow> and <blow>, <scar> and <star>. They reported that W showed normal performance in the temporal order judgement task. However, in the backward masking task he showed significantly higher thresholds than AZ or the control group. Moreover, in the same-different discrimination task W made significantly more errors than AZ or controls. In contrast to W, AZ performed normally in all auditory and speech measures. On the basis of these results, the authors concluded that there is strong evidence that G-SLI can occur without an underlying auditory processing problem.

In later studies on G-SLI, van der Lely and colleagues (2004) and Rosen and colleagues (2009) investigated the input-processing skills of groups of grammatically impaired children and their controls. Van der Lely et al. (2004) presented the children with different speech and non-speech sounds at various presentation rates: a [bʌ] – [dʌ] discrimination task, a tone discrimination (the isolated F2 from the speech contrast) task and a tone discrimination task where tones are presented at different rates. The results showed that in speech sound discrimination task, only 31% of G-SLI performed normally. Furthermore, for the rapidly presented tones, 46% of G-SLI children performed normally whereas in the F2 alone condition 69% did so. Despite the group level differences, van der Lely et al. (2004) pointed out that it is evident that not all children with G-SLI show auditory impairments. Rosen et al. (2009), in turn, investigated a group of G-SLI teenagers and their age, grammar and vocabulary matched

(younger) controls in tone detection tasks (tone presented in quiet, simultaneously with noise or followed by noise). Their results showed that in noise conditions, the detection thresholds were higher in the G-SLI group than in the age matched controls but not higher than those obtained from younger controls. Moreover, Rosen et al. (2009) pointed out that despite the group-level differences, approximately half of the G-SLI participants had age-appropriate thresholds for all conditions. Furthermore, in both studies, the authors found no relationship between auditory processing abilities and grammatical, phonological or vocabulary abilities and argued that G-SLI is not caused by an underlying deficit in auditory input-processing. However, as mentioned earlier (Section 2.2.4), an underlying input-processing deficit could be camouflaged by task demands or attentional factors and, therefore, some of the methods that were previously used may not be sensitive enough to spot the deficit. Moreover, these studies mentioned above are conducted on a specific subgroup of SLI (G-SLI) and, therefore, their generalizability to SLI is an open theoretical question.

In addition to contradictory behavioural findings, the electrophysiological findings have not been straightforward. In a recent review, Bishop (2007) summarized ERP results from a wide range of studies conducted with adults and children with SLI and dyslexia. On the basis of these studies, she noted that the overall trend was to find attenuated mismatch negativity (MMN) amplitudes, longer latencies and atypical lateralization in the clinical group when compared to controls. However, by far not all studies succeeded in finding differences between children with SLI/dyslexia and controls (see Bishop, 2007 for a review). Moreover, the experimental setup (e.g., presentation rates, MMN quantification and language backgrounds) and stimulus selection (e.g., simple tones, complex tones, isolated vowels and CV syllables) varied considerably between studies and in the vast majority of studies the sample size was fairly small. Moreover, several studies have attempted to combine the electrophysiological and behavioural methods (e.g., MMN response and discrimination accuracy) to investigate the nature and locus of auditory processing deficits in developmental language disorders. The results have been contradictory in that in some studies a correlation between the MMN response and

behavioural accuracy has been found (Sams, Paavilainen, Alho, & Näätänen, 1985; Lang et al., 1990; Lang et al., 1995; Baldeweg et al. 1999; Kujala et al., 2000; Amenedo & Escera, 2000) whereas some studies failed in establishing that relationship (Uwer et al. 2002; Shafer et al. 2005; Paul et al. 2006). Bishop (2007) made several suggestions for future research in order to decrease the variability between designs (and possibly in results). Accordingly, she suggested that: 1) (whenever possible) the sample sizes should be larger, 2) in addition to grand-averaged waveforms, studies should report some measures of within-group variation (e.g., numerical data and SDs) and the effect sizes, 3) MMN quantification methods should be well justified, 4) theoretically meaningful stimulus selection criteria should be defined, 5) selected stimuli should be perceptually difficult enough in order to avoid ceiling effects, and 6) speech sound processing should be compared with non-linguistic stimuli processing (e.g., complex tones) with similar acoustical complexity.

7.1.3 Mismatch negativity response as tool to investigate auditory and speech processing In the present experiment, I will look at the behavioural discrimination accuracy in SLI and its relationship with the brain's automatic change-detection as indexed by the Mismatch negativity (MMN) component of auditory event-related potentials. MMN has been described as the sensory index of behavioural discrimination accuracy (see a review in Section 4.5.1) thus providing an ideal tool to complement more traditional behavioural methods. Moreover, in addition to auditory processing, MMN has been claimed to reflect phonological processing and sensitivity to native language speech sound categories or even native language phoneme prototypes (see Chapter 4).

MMN is a relatively stable component across ages, which is an advantage when comparing different age groups. Moreover, MMN elicitation does not require overt responses or even attention directed to the stimuli, making it very suitable for investigating young children that may sometimes suffer from poor motivation in the behavioural task. Due to the above mentioned factors, MMN has been used widely in

investigating auditory processing in different clinical populations and in small children (for a review, see Cheour et al., 2000).

In this Section, by focusing on the MMN component, I investigate the processing of two acoustic cues, namely frequency and duration, responsible for syllable-final stop consonant voicing in English by a group of young adults with SLI, group of young adults with dyslexia and their matched controls. In the present experiment, I combine various different methods to tease apart the nature and locus of the potential auditory processing deficit. More specifically, I investigate how different speech and complex non-speech sounds are attentively discriminated. In addition to these behavioural methods, I use electroencephalography (EEG) to examine whether or not there is an underlying auditory deficit camouflaged by task demands. Moreover, in order to investigate the individual variation in the behavioural data, I have adopted a method used by White et al. (2006) (see Chapter 6 for details).

7.2 Method

7.2.1 Participants

The participants in this study are the same who took part in the Cue weighting study (except one participant, S049, see Appendix A_1 for study participation details).

12 young adults with SLI aged between 15;03 and 25;00 (8 males, the selection criteria for these individuals are detailed in Chapter 5) and 12 young adults with dyslexia (8 males) aged between 14;09 and 24;06 participated in this study. A control group (N=12, 10 males) was matched with SLI and dyslexia on chronological age (CA), non-verbal IQ, gender and laterality. Moreover, in addition to the matched groups, an additional non-matched adult group was tested on behavioural task to establish the adult performance (N=14, 7 males, aged between 18;00-34;09, mean 24;07 years).

There were no significant differences between groups in chronological age [one-way ANOVA, F(2,35)=.271, p=.764], non-verbal IQ [one-way ANOVA, F(2,35)=.953, p=.396], or gender distribution (Fisher's exact p=.717). However, to establish that in our study individuals with dyslexia did not have co-occurring language impairments, their performance on TROG-2 and BPVS-II was also assessed. All individuals with dyslexia had normal language skills and their scores on TROG-2 and BPVS-II differed significantly from those of SLI participants [independent samples t-tests: TROG-2: t(19)= -4.504, p<.001, BPVS-II: t(19)=-3,383, p=.003]. All subjects were right handed (the Edinburgh handedness questionnaire), reported normal hearing and they had no known neurological conditions (see Table 7.1. for details).

Table 7.1: Summary of group matching details

		Groups	
	SLI n=12	DYS n=12	CA Control
AGE			
Mean (years)	18;07	19;05	19;01
Range	15;03-25;00	14;09-24;06	15;04-25;01
(SD)	3;3	3;7	3;3
RAVENS			
Raw	48	50	50
Range	41-60	44-57	42-56
(SD)	(5.7)	(4.0)	(5.2)
SS	102	108	108
Z-Score	0.14	0.45	0.56
TROG-2			
Raw	14.3 ^a	18.6^{a}	n/a
Range	11-17	16-20	n/a
(SD)	(2.2)	(1.1)	n/a
SS	83.7	101.9	n/a
Z-Score	-1.08	0.11	n/a
BPVS			
Raw	110 ^b	138.6 ^b	n/a
Range	84-147	121-155	n/a
(SD)	(18.3)	(12.8)	n/a
SS	82.2	116.1	n/a
Z-Score	-1.19	1.10	n/a

^{*} at the time of ERP testing a,b p< .01

7.2.2 Stimuli

The stimuli used in this experiment are chosen from the 14 synthesized speech stimuli reported in Chapter 6. In addition to synthetic speech, a similar set of acoustic ("non-speech") control stimuli were synthesized. These stimuli model the vocal tract resonances of three lowest formants of corresponding speech stimuli. Due their physical

properties, these sinewave analogues (SWS) can be perceived as speech or non-speech depending on the instruction (Remez et al., 1981) (see the Figure 7.1).

For preattentive and attentive discrimination, eight stimuli were selected based on the 2 (Duration: long, short) x 2 (Frequency: high, low) x 2 (Mode: speech, non-speech) design. Thus, two of the stimuli were reliably identified as [bot] (vocalic duration "short" i.e., 120 ms, F1 "high" i.e., 570 Hz) and [bo:d] (vocalic duration "long" i.e., 220 ms, F1 "low" i.e., 250 Hz) and represented the typical exemplars of syllable-final British English [t] and [d] (typical stimuli are named as "High120" and "Low220" or "H120" and "L220" respectively). The other two stimuli were reliably identified as either [bot] or [bo:d] but they contained conflicting cues for the consonant in question. In other words, they consisted of formant transition typical for voiceless consonant but vocalic duration typical for voiced one (named "High220" or "H220) identified as [bo:d]) and vice versa (named "Low120" or "L120" identified as [bot]) thus forming atypical within-category variants of the typical non-words. In addition to speech sound discrimination, the subjects performed a discrimination tasks with four corresponding sine wave speech analogues (SWS, see Figure 7.1).

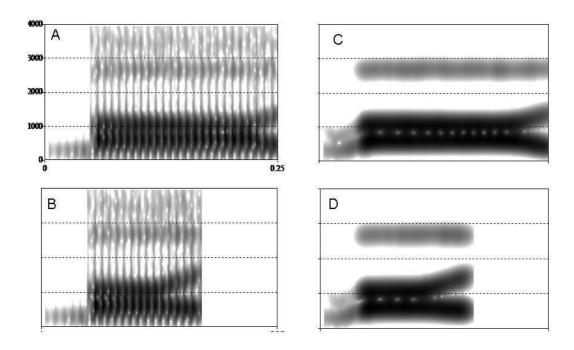


Figure 7.1 A) Spectrogram of synthesized [bo:d] (vocalic duration 220 ms, F1 end frequency 250 Hz) B) synthesized [bot] (vocalic duration 120 ms, F1 end frequency 570 Hz) and their sine wave speech analogues (SWS) (C-D). X-axis represents time (0-0.25 sec) and y-axis frequency (0-4KHz).

In the synthesis, all vocalic portions were preceded by 50 ms of silence (signalling the initial stop consonant [b]) during which the amplitude of voicing (AV) parameter was interpolated from 40 to 60 dB. The fundamental frequency (F0) was set to increase from 100-130 Hz during the initial 50 ms after which it linearly decreased to 95 Hz to imitate a natural pitch contour in speech. In the voiceless token (/bot/) the F1 frequency was set to a constant value of 570 Hz throughout the syllable. The voiced consonant /d/ was created by lowering the offset frequency of F1 to 250 Hz during the final 50 ms. The F2 frequency was set to 1000 Hz, rising to 1500 Hz during the final 50 ms. The F3 frequency was kept at a constant value of 2650 Hz. All higher formant frequencies (F4, F5, F6) were kept in the HLsyn default values (3500, 4500, 4990 Hz respectively). Finally, a 15 ms linear onset and offset ramp was used to remove clicks and the amplitudes were normalized (rms -10 dB of the maximum amplitude) in all stimuli by using CoolEdit96. All formant frequencies were checked using Praat (4.4.16, Boersma

& Weenink, 2006). The non-speech control stimuli were created by replacing the three lowest formants with sinusoids by using Praat (4.4.16, see Figure 7.1).

7.2.3 Procedure

In the present study CA, SLI and dyslexic participants took part in two experiments: Behavioural discrimination task and an ERP task. The study was conducted during one day or during two consecutive days. On the whole, it took approximately 2-3 hours to complete the study. Participants received £20-30 for participating. Non-matched adults took part only in the behavioural discrimination and received £5 for participating. The order of speech and non-speech blocks were counterbalanced to overcome order effects (e.g., to control for effects of practice and motivation/fatigue). Counterbalancing the order of the SWS stimuli is problematic because the participant can learn to hear the SWS sounds as speech if hearing the speech block first. However, when asked after the study, none of the participants had heard the SWS sounds as speech.

7.2.3.1 Roving-standard paradigm

The discrimination tasks (behavioural and MMN tasks) are presented in a roving-standard (or varying standard) paradigm (Huotilainen, Kujala, & Alku, 2001; Shestakova et al., 2002). In this paradigm all four stimuli ([bɒt], [bot/d], [bod/t], [bɔ:d]) are standards and deviants. In other words, each of the four deviants becomes a standard stimulus thus avoiding the control conditions (i.e., presenting deviants in isolation or reversing the presentation order) required by the standard oddball paradigms. The same procedure is applied to the SWS stimuli presented in a separate block.

The rationale of the paradigm is as follows: once the target (deviant) sound is presented it becomes the new standard. This standard is then repeated 2-5 times (see Figure 7.2) before the next deviant appears.



Figure 7.2: an illustration of the roving-standard paradigm.

In the data analysis, those standards immediately following a deviant are removed from the average following the studies by Huotilainen et al. (2001) and Shestakova et al. (2002). Therefore, only the third stimulus of each new sequence is considered as standard.

7.2.3.2 Behavioural discrimination

In this experiment, the speech and non-speech stimuli were presented in a roving-standard paradigm where each deviant becomes the standard stimulus (SOA 1000 ms, 187 stimuli in total, 40 deviants) in separate blocks. All four target non-words (two typical, two atypical tokens) were presented ten times in a pseudo-random order. The number of standards preceding a deviant varied and the change was not predictable. During the experiment, subjects were asked to press a button as quickly as possible as soon as they heard a change in the stimulus train (go/no-go task). A short practice session (a total of 34 stimuli, 5 deviants) preceded the experiment to make sure the participants understood the instructions. The experiment took approximately ten minutes to complete (see instructions Appendix C).

To analyze the speech and non-speech data, the d-prime (d') measure of discrimination sensitivity (signal detection theory, SDT; Macmillan & Creelman, 1990) was calculated to account also for the possible differences in the response strategies between subjects. D' measure of detection creates a model of participant's response by taking into account two parameters: the difficulty of the task and participant's response strategy. The difficulty of the task means that if the task is easy, participant is more likely to accomplish more "hits" (i.e., pressing button correctly when the change occurs) and have less "false alarms" (i.e., pressing button incorrectly when there is no change). The response strategy refers to participant's "tactics" in the experiment (i.e., if someone always says "NO" there are no false alarms whereas when someone always says "YES" the hit rate is maximal). A participant who is more likely to respond "NO" is called a "conservative" responder and a participant who is more likely to respond "YES" is

called a "liberal" responder. A C-criterion reveals participant's strategy giving, therefore, additional information about their performance in the task. An ideal perceiver has a C-value of "0".

7.2.3.3 EEG data acquisition and analysis

The same stimuli (speech and non-speech) and the same paradigm (roving-standard; SOA 800 ms, total 2160 stimuli) as in the behavioural task were used in the EEG recording. The SOA was shortened to 800 ms from the behavioural study in order to reduce the overall EEG recording time. The sequence was created using the Sequence Maker toolbox (available at http://www.cbru.helsinki.fi/seqma/). There were 120 deviants in each category (total 480 deviants). Those standards immediately following a deviant were removed from analysis. EEG was recorded with 128 channel electrode net (Electrical Geodesics Inc.) using Net Station (4.1.2) software for data acquisition and analysis. Amplifier sampling rate was 250 Hz with a 0.1-100 Hz band pass filter. The auditory stimuli were presented with Biological E-Prime Program (Psychology Software Tools, Inc. version 1.0.20.1) via loudspeakers at a comfortable level while subjects were seated in a Faraday cage in a comfortable chair. Subjects watched children's cartoons (sound off), conducted a simple counting task (e.g., "count how many cats/dogs/cows you see during the nine minute block") and were asked to ignore the auditory stimuli. Subjects' performance in the counting task was monitored and they were told that the experimenter will ask questions relating to the cartoons after each block.

The current experiment consisted of two separate sessions (speech and non-speech) each of which was divided into four nine minute blocks with a short break between the blocks. The recording session took approximately 90 minutes. EEG data were off-line filtered with a 1-30 Hz band pass filter, baseline corrected with respect to 100 ms prestimulus baseline. Instead of artifact correction (e.g., for eye blinks), a conservative artifact detection criterion (70 μ V) was used to remove epochs contaminated with eye blinks and movements. Each participant had to have at least 85 accepted trials to be included in the study. The data were segmented from 100 ms prestimulus to 600 ms

poststimulus and averaged offline. Finally the data were re-referenced to the common average voltage of all electrodes.

ERP data analyses were done on mean and peak amplitudes and peak latencies. Both analyses methods were included because peak amplitude measures tend to be less reliable especially in noisier data and mean amplitude measures tend to be sensitive to latency jitter of a component. Peak N1 amplitudes and latencies were quantified as the maximum negativity in the standard wave between 150-250 ms after the stimulus onset (i.e., 100-200 ms after vowel onset). Seven electrodes (Fz, Fcz, Cz, F3, F4, C3, C4, see Figure 7.3) were chosen for statistical analyses (Analysis of Variance, ANOVA). However, if no interactions between the experimental variables and electrode position (i.e., topographical differences) were identified, analyses were conducted on a single central electrode that showed the most negative peak amplitudes (Fcz).

The mean and peak MMN amplitudes and peak latencies were quantified in the difference wave between 280-380 ms after stimulus onset (i.e., about 150-250 ms after the critical point). The selection of time-window for MMN analysis was both theory-driven (i.e., when MMN is expected to peak after change) and based on visual inspection of the group data. MMN analyses were firstly performed on averaged amplitudes for standards and deviants across nine Regions of Interest (ROI, see Appendix D): **Anterior:** left-central-right, **Medial:** left-central-right and **Posterior:** left-central-right. After this, seven electrodes (Fz, Fcz, Cz, F3, F4, C3, C4) were chosen for further analysis (ANOVA). Lastly, if no interactions between variables and electrode position were present, analyses were conducted on a single central electrode that showed the most negative peak/mean amplitude (Fcz). All statistics were Greenhouse-Geisser corrected when necessary. The Epsilon (ε) value is reported when Mauchly's test of sphericity was significant and subsequently the Greenhouse-Geisser corrected p-values and uncorrected degrees of freedom are reported. The critical alpha level is .05 unless otherwise stated and all effects below .10 are considered as statistical trend.

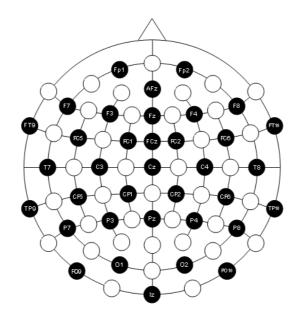


Figure 7.3: Electrode names and locations in a 32-channel system. Three mid-line electrodes (Fz, Fcz and Cz), two left-side electrodes (F3 and C3) and two right-side electrodes (F4 and C4) were chosen for statistical analysis.

7.3 Results

7.3.1 Behavioural results

Tables 7.2 and 7.3 show the means and standard deviations (SDs) of the d'discrimination index and Figure 7.5 shows the mean absolute hit rates for 34 subjects. Data from one dyslexic subject was excluded due to technical problems and from one SLI participant who did not finish the task.

Table 7.2: D' -scores for SLI, dyslexia and Controls (CA) and non-matched adults in speech discrimination (SD) task.

	High120	Low220	High220	Low120	Total _{SPEECH}
CA	1.91 (0.25)	1.56 (0.32)	1.85 (0.24)	1.84 (0.38)	1.79 (0.30)
SLI	1.81 (0.36)	1.38 (0.29)	1.54 (0.46)	1.69 (0.49)	1.61 (0.40)
DYS	1.85 (0.38)	1.81 (0.45)	1.92 (0.26)	1.68 (0.27)	1.82 (0.34)
Total	1.86 (0.33)	1.58 (0.35)	1.77 (0.32)	1.74 (0.38)	1.74 (0.35)
Adults	1.66 (0.33)	1.42 (0.20)	1.75 (0.31)	1.46 (0.40)	1.57 (0.31)

Table 7.3: D'-scores for SLI, dyslexia and Controls (CA) and non-matched adults in non-speech discrimination (SD) task.

	High120	Low220	High220	Low120	Total _{SWS}
CA	1.60 (0.61)	1.68 (0.29)	1.88 (0.35)	1.56 (0.33)	1.68 (0.40)
SLI	1.63 (0.44)	1.27 (0.59)	1.73 (0.39)	1.62 (0.41)	1.56 (0.46)
DYS	1.65 (0.68)	1.61 (0.68)	1.72 (0.38)	1.43 (0.63)	1.60 (0.59)
Total	1.63 (0.58)	1.52 (0.52)	1.78 (0.37)	1.54 (0.46)	1.61 (0.48)
Adults	1.32 (0.59)	1.53 (0.55)	1.17 (0.44)	1.23 (0.44)	$1.32 (0.51)^5$

Repeated measures ANOVAs revealed a significant main effects of **Mode** [F(1,31)=4.622, p=.039, η_p^2 =.130, see Tables 7.2 and 7.3] and **Frequency** [F(1,31)=14.665, p=.001, η_p^2 =.321; **High**: 1.76, 95% CI: 1.65-1.86; **Low**: 1.59, 95% CI: 1.48-1.71]. The main effect of **Group**, however, was not significant [F(2,31)=.945, p=.400, η_p^2 =.057, NS]. Lastly, a **Duration*Group** interaction was significant [F(2,31)=4.075, p=.027, η_p^2 =.208, see Figure 7.4].

For this interaction, one-way ANOVA showed that the three groups differed significantly in the "long" condition but not in the "short" condition [**Short**: F(2,33)=.139, p=.871, NS; **Long:** F(2,33)=3.433, p=.045]. Moreover, Post Hoc analysis (LSD) for the "long" condition revealed that SLI group differed significantly both from Controls (p=.035) and from dyslexics (p=.026).

⁵ Non-matched adults showed a significant main effects of **Mode** [F(1,13)=7.840, p=.015, η_p^2 =.376; **Speech:** 1.57, 95% CI: 1.45-1.69; **SWS:** 1.32, 95% CI: 1.10-1.54] and **Frequency** [F(1,13)=45.305, p<.001, η_p^2 =.777;**High:** 1.57, 95% CI: 1.41-1.72; **Low:** 1.32, 95% CI: 1.17-1.48]

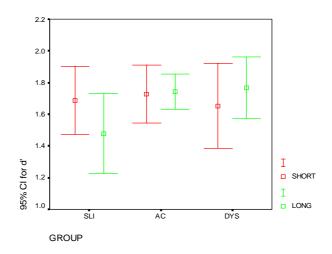


Figure 7.4: 95% Confidence Intervals in d' measures for Group*Duration interaction.

In the current experiment, the d' scores were relatively high suggesting a possible ceiling effect in the data. However, the absolute hit rates (see Figure 7.5) indicate that the task was not easy and the participants detected less than half of the targets. Therefore, high d' scores are most likely due to a very low false alarm rate typical to a go/no-go task in which the relative amount of targets is low (mean FA rates for **CA**: 0.7 and 0.5; **SLI**: 1.3 and 1.0; **DYS**: 1.0 and 0.8 for speech and non-speech respectively). In other words, the participants had adopted a conservative response strategy [C-criteria (SD) for SLI: 1.1 (0.7); for Dyslexics: 1.1 (0.7); for CA 1.2 (0.7), where positive value indicates a conservative observer].

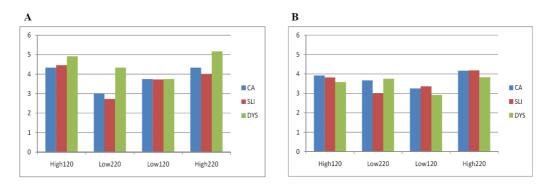


Figure 7.5: Absolute hit rates for speech (A) and non-speech (B) in CA, SLI and dyslexia groups (number of correct responses out of 10).

7.3.1.2 Individual data

Scatterplots for discrimination sensitivity (d') in speech and non-speech (SWS) conditions are presented in Figure 7.6. To identify poor performers, the same method as was used for the identification data (see Chapter 6) was employed. In other words, -1.65 SD from the control mean was set as the cut-off value and any participant scoring below this value was defined as an outlier.

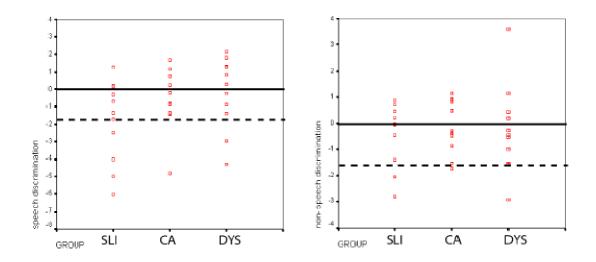


Figure 7.6. Scatterplots for discrimination sensitivity (d') in SLI, controls and dyslexics for speech (left) and non-speech (right). The y-axis value are z-scores, solid line represents the control mean and dotted line the -1.65 cut-off value for impaired performance.

Overall, 36% of participants in the SLI group, 18% of dyslexics and 8% of controls scored below this cut-off in the speech condition. In the non-speech condition, however, 18% of SLI, 9% of dyslexics and 8% of controls were outliers. These poor performers generally tended to score lower on both speech and non-speech conditions in both SLI and dyslexia groups.

7.3.2 ERP results

Due to time constraints, one subject from dyslexia group did not complete the EEG recording session (see Appendix A_1 for group participation details) and one subject from

the SLI group had to be excluded due to extensive artefacts in the data. The data from the remaining 34 subjects are presented below.

7.3.2.1 N1 response

The mean voltages for standards and deviants are presented in Figures 7.7-7.8 and the deviant-standard difference waves in Figures 7.9-7.10. Repeated measures ANOVA with four within-subjects factors: Electrode (7), Mode (2), Frequency (2), Duration (2) and a between-subject factor: Group (3) revealed a main effect of **Electrode** [F(6,186)=14.654, p<.001, η_p^2 =.321, ϵ =.366]. The main effect of **Group** was not significant [F(2,31)=.231, p=.795, η_p^2 =.015, NS]. Moreover, the **Electrode*Mode** interaction was significant [F(6,186)=5.423, p=.003, η_p^2 =.149, ϵ =.422, see Appendix F] for standard stimuli.

After removing the Electrode factor the subsequent analyses on peak amplitudes at Fcz electrode showed a statistically significant main effect of **Mode** [F(1,31)=6.474, p=.016, η_p^2 =.173: **Speech**: -1.32 μ V; 95% CI: -1.84 – (-.80); **SWS**: -1.84 μ V; 95% CI: -2.33 – (-1.35)]. The main effect of **Group** was not significant [F(2,31)=.914, p=.411, η_p^2 =.056, NS].

Furthermore, the latencies of the N1 peak amplitudes at Fcz electrode showed a statistically significant main effect of **Mode** [F(1,31)=23.954, p<.001, η_p^2 =.436: **Speech**: 189ms; 95% CI: 179-200; **SWS**: 216ms, 95% CI: 206-226]. The main effect of **Group** was not significant [F(2,31)=1.422, p=.256, η_p^2 =.082, NS].

Overall, no other significant main effects of interactions were found on the N1 response.

7.3.2.2 Mismatch Negativity (MMN) response

Regions of Interest (ROI)

The mean voltages for standard and deviant across the nine ROIs are presented in Table 7.4.

Table 7.4: Mean voltages (in μ V) for nine regions of interest for standard and deviant (SE). AL=Anterior Left, AC=Anterior Central, AR=Anterior Right, ML=Medial Left, MC= Medial Central, MR= Medial Right, PL=Posterior Left, PC=Posterior Central, PR=Posterior Right.

	AL	AC	AR	ML	MC	MR	PL	PC	PR
STD	-1.4 (0.3)			-0.7 (0.2)					
DEV	-2.4 (0.3)			-1.2 (0.2)					

The repeated measures ANOVA [five within-subject factors: ROI (9), Condition (2), Mode (2), Duration (2), Frequency (2) and a between-subject factor: Group (3)] for ROI analysis revealed significant main effects of **ROI** [F(8,256)=53.588, p.<001, η_p^2 =.626], **Condition** [standard vs. deviant: F(1,31)=43.337, p.<001, η_p^2 =.575] and **Mode** [speech vs. SWS: F(1,31)=4.941, p=.033, η_p^2 =.134]. The main effect of **Group** was not significant (F(2,31)=.217, p=.806, NS).

The follow-up analyses for ROI showed that all anterior regions (Left, Central and Right) differed significantly from one another (see Tables A-E in Appendix E for statistical details). However, in Medial and Posterior regions the left/right areas differed from central area but not from each other whereas in the Anterior region the activation was larger at the right (see Table B in Appendix E). Furthermore, as expected, in the left-right axis, all regions (Anterior, Medial, Posterior) differed significantly from each other (see Table C in Appendix E). The ROI analyses did not indicate any differences in lateralization between the three groups (CA, SLI and dys) or between the two modes (speech and SWS).

The main effect of **Condition** was due to the fact that deviants were generally more negative than standards [**deviant**: $-1.35\mu V$, 95% CI: -1.65 - (-1.04); **standard**: $-0.72\mu V$, 95% CI: -1.01 - (-0.42), see Table 7.4]. The main effect of **Mode** was due to the fact that

sinewave speech (SWS) elicited more negative responses than speech [SWS: -1.15 μ V, 95% CI: -1.42 – (-0.87); speech: -0.92 μ V, 95% CI: -1.25 – (-0.59)].

In addition to the main effects reported above, the interaction **ROI*Condition** was statistically significant $[F(8,256)=28.870, p<.001, \eta_p^2=.474, see Table 7.4 for mean voltages] indicating that standards and deviants differed at different regions of interest (see Table D in Appendix E for statistical details).$

Mean amplitude

Repeated measures ANOVA revealed a main effect of **Electrode** [F(6,186)=9.837, p<.001, η_p^2 =.241, ϵ =.562]. No other significant effects with factor Electrode were found (p>.10).

At Fcz electrode, One-sample t-tests showed that not all stimuli elicited a significant MMN response (see Figure 7.7-7.10 for responses to standards and deviants and the corresponding grand-averaged difference waves and Table 7.5 for the mean amplitudes at Fcz). Consequently, a significant MMN was elicited for five of the total eight contrasts in the CA and dyslexia groups whereas the corresponding figure was three out of eight in the SLI group.

Table 7.5: MMN mean amplitudes at Fcz in μV (SD) for deviant-standard difference waves for CA, SLI and dyslexic groups and for speech and non-speech (SWS).

	High120	High220	Low120	Low220	Total
CA					
SPEECH	-0.50 (1.48)	-1.36 (1.55)	-0.48 (1.14)	0.25 (1.12)	-0.52 (1.32)
SWS	-1.09 (1.33)	-0.76 (1.13)	-0.39 (1.00)	-0.80 (1.33)	-0.76 (1.19)
SLI					
SPEECH	-0.62 (1.50)	-0.46 (1.48)	-0.75 (1.41)	0.17 (1.56)	-0.42 (1.49)
SWS	-0.35 (1.74)	-1.21 (1.49)	-0.97 (1.82)	-0.35 (1.69)	-0.72 (1.68)
DYS					
SPEECH	-1.06 (1.52)	-1.30 (1.53)	-0.25 (1.37)	-0.50 (1.45)	-0.78 (1.47)
SWS	-1.01 (1.54)	-1.41 (1.49)	-1.04 (1.59)	-0.57 (1.64)	-1.01 (1.57)
Total	-0.77 (1.52)	-1.08 (1.45)	-0.65 (1.39)	-0.30 (1.47)	-0.70 (1.45)

The subsequent analyses at Fcz showed a marginally significant 3^{rd} order interaction of $\mathbf{Mode*Frequency*Duration*Group}$ [F(2,31)=2.910, p=.069, η_p^2 =.158]. This group interaction was due to CA group showing a significant $\mathbf{Mode*Frequency*Duration}$ interaction [F(1,11)=7.447, p=.020, η_p^2 =.404] (see Table 7.6) whereas in the SLI and dyslexia groups this interaction was not significant [**SLI**: F(1,10)=.420, p=.535, η_p^2 =.040, NS; **Dys**: F(1,10)=.458, p=.514, η_p^2 =.044, NS].

Table 7.6: Frequency*Duration interaction and 95% CIs (μV) for speech and SWS in the CA group.

	df	\mathbf{F}	p	η_{p}^{-2}	mean	95% CI
					μV	Lower Upper
Speech (fre*dur)	1,11	9.247	.011	.457	•	
High120					-0.50	-1.47 0.47
High220					-1.36	-2.47 -0.24
Low120					-0.48	-1.21 1.25
Low220					0.25	-0.46 0.96
SWS (fre*dur)	1,11	1.270	.284	.104		
High120					-1.09	-1.93 -0.24
High220					-0.76	-1.48 -0.03
Low120					-0.39	-1.29 0.50
Low220					-0.80	-1.64 0.05

The **Mode*Frequency*Duration** interaction in the CA group is due to the control group eliciting larger (even though not significant) MMN amplitudes for atypical tokens [i.e., High220 and Low120; t(11)=1.635, p=.130, NS] compared to typical tokens [High120 and Low220; t(11)=-1.706, p=.116, NS] in the speech condition. No other statistically significant main effect or interaction for mean amplitudes was found at the Fcz electrode.

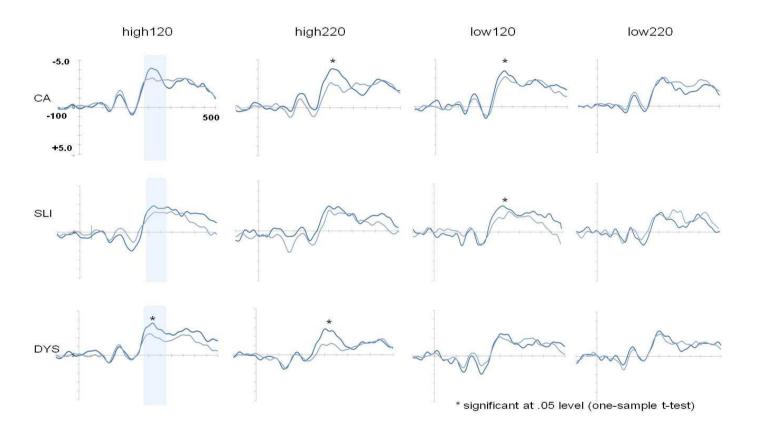


Figure 7.7. ERP waveforms at Fcz for standards (light blue line) and deviants (dark blue line) for three groups (CA, SLI and Dyslexia) in speech condition. The time-window for MMN analyses is highlighted and the onset of the vocalic portion (at 50 ms) of the stimuli is indicated with a line. Time (in ms) is represented in the x-axis and amplitude (in μ V) in y-axis.

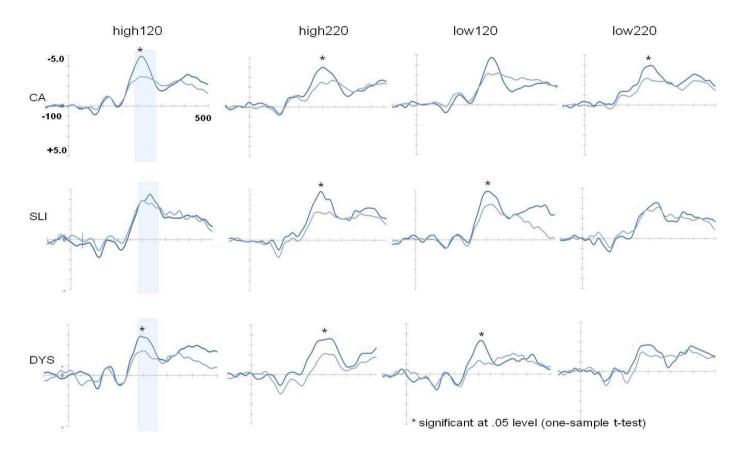


Figure 7.8. ERP waveforms at Fcz for standards (light blue line) and deviants (dark blue line) for three groups (CA, SLI and Dyslexia) in non-speech (SWS) condition. The time-window for MMN analyses is highlighted and the onset of the vocalic portion (at 50 ms) of the stimuli is indicated with a line. Time (in ms) is represented in the x-axis and amplitude (in μ V) in y-axis.

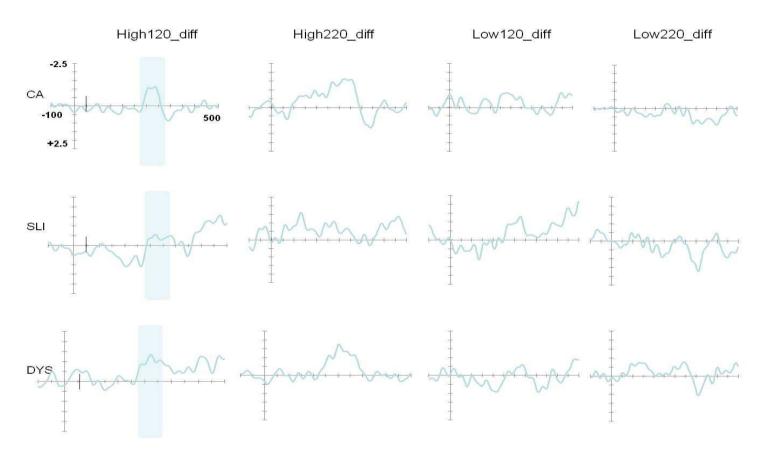


Figure 7.9. MMN difference waveforms at Fcz for three groups (CA, SLI and Dyslexia) in speech condition. The time-window for MMN analyses is highlighted and the onset of the vocalic portion (at 50 ms) of the stimuli is indicated with a line. Time (in ms) is represented in the x-axis and amplitude (in μ V) in y-axis.

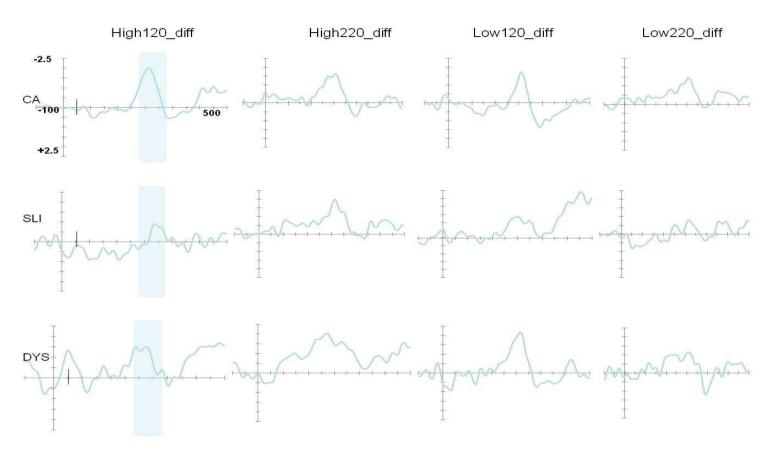


Figure 7.10. MMN difference waveforms at Fcz for three groups (CA, SLI and Dyslexia) in non-speech (SWS) condition. The time-window for MMN analyses is highlighted and the onset of the vocalic portion (at 50 ms) of the stimuli is indicated with a line. Time (in ms) is represented in the x-axis and amplitude (in μV) in y-axis.

Peak amplitude and latency

Repeated measures ANOVA showed a main effect of **Electrode** [F(6,186)=18.338, p<.001, η_p^2 =.372, ϵ =.518] for peak amplitude. No interaction with factor Electrode reached statistical significance in the peak amplitude measure (p>.10).

The peak amplitudes at the Fcz electrode for each group are presented in Table 7.7.

Table 7.7: MMN peak amplitudes at Fcz in μV (SD) for deviant-standard difference waves for CA, SLI and dyslexic groups and for speech and nonspeech (SWS).

	High120	High220	Low120	Low220	Total
CA					
SPEECH	-1.31 (2.25)	-2.09 (2.08)	-1.77 (2.01)	-0.15 (1.48)	-1.33 (1.96)
SWS	-2.37 (1.95)	-1.61 (1.13)	-1.88 (1.67)	-1.50 (1.57)	-1.84 (1.58)
SLI					
SPEECH	-1.54 (1.61)	-1.24 (1.73)	-1.77 (1.54)	-0.67 (2.16)	-1.31 (1.76)
SWS	-1.09 (1.97)	-2.12 (1.69)	-1.51 (2.29)	-1.06 (1.94)	-1.45 (1.97)
DYS					
SPEECH	-2.34 (1.76)	-2.40 (2.31)	-1.05 (1.96)	-1.38 (1.89)	-1.79 (1.98)
SWS	-1.78 (1.91)	-2.31 (2.19)	-2.45 (2.22)	-1.61 (1.58)	-2.04 (1.97)
Total	-1.74 (1.91)	-1.96 (1.86)	-1.74 (1.95)	-1.06 (1.77)	-1.63 (1.87)

At the Fcz electrode the 3rd order interaction **Mode*Frequency*Duration*Group** was significant [F(2,31)=4.260, p=.023, η_p^2 =.216]. As in the mean amplitude measure, this group interaction was due to CA group showing a significant **Mode*Frequency*Duration** interaction [F(1,11)=7.492, p=.019, η_p^2 =.405] (see Table 7.8) whereas in the SLI and dyslexia groups this interaction was not significant [**SLI**: F(1,10)=.359, p=.563, η_p^2 =.035, NS; **Dys**: F(1,10)=1.640, p=.229, η_p^2 =.141, NS].

The Mode*Frequency*Duration interaction in the CA group is due to CA group eliciting larger (even though not significant in the "High" condition) MMN amplitudes for atypical tokens (i.e., High220 and Low120) compared to typical tokens (High120 and Low220) [H120 vs. H220: t(11)=1.220, p=.248, NS; L120 vs L220: t(11)=-3.237,

p=.008] in the speech condition. No other statistically significant main effect or interaction was found at the Fcz electrode.

Table 7.8: Frequency*Duration interaction and 95% CIs (µV) for speech and SWS in the CA group.

	df	F	p	$\eta_{\mathrm{p}}^{\ 2}$	mean	95% CI
					μV	Lower Upper
Speech (fre*dur)	1,11	8.688	.013	.441		
High120					-1.31	-2.74 0.12
High220					-2.09	-3.41 -0.76
Low120					-1.77	-3.04 -0.49
Low220					-0.15	-1.09 0.79
SWS (fre*dur)	1,11	.317	.585	.028		
High120					-2.37	-3.61 -1.13
High220					-1.61	-2.33 -0.89
Low120					-1.88	-2.95 -0.82
Low220					-1.50	-2.50 -0.50

The peak latencies at the Fcz electrode for each group are presented in Table 7.9.

Table 7.9: MMN peak latencies at Fcz in μV (SD) for deviant-standard difference waves for CA, SLI and dyslexic groups and for speech and nonspeech (SWS).

	High120	High220	Low120	Low220	Total
CA					
SPEECH	324 (25)	323 (23)	331 (33)	320 (29)	325 (28)
SWS	326 (18)	312 (27)	315 (13)	308 (31)	315 (22)
SLI					
SPEECH	330 (29)	334 (29)	339 (32)	328 (33)	333 (31)
SWS	332 (25)	317 (16)	320 (24)	326 (27)	324 (23)
DYS					
SPEECH	330 (33)	332 (28)	337 (28)	321 (29)	330 (30)
SWS	307 (22)	323 (27)	315 (25)	318 (27)	316 (25)
Total	325 (25)	324 (25)	326 (26)	320 (29)	324 (27)

On peak latency measures, the analyses revealed the main effect of **Mode** $[F(1,31)=12.200, p=.001, \eta_p^2=.282;$ **Speech:** 329ms; 95% CI: 321-337ms; **SWS:** 319ms;

95% CI: 313-325ms] and an interaction of **Mode*Frequency*Duration** [F(1,31)=4.242, p=.044, η_p^2 =.125]. This interaction was due to the two "Low" stimuli differing significantly [t(33)=2.179, p=.037] whereas the two "High" stimuli did not [t(33)=-.351, p=.728, NS] in the speech condition. In short, the interaction was caused by the typical sounds eliciting overall faster peak latencies (even if not significantly) than atypical sounds in speech condition but not in sine-wave speech condition (see Table 7.10).

Table 7.10: Frequency* Duration interaction for peak latency (ms) and 95% CIs for speech and SWS across all three groups.

	$\begin{array}{cccc} df & F & p & {\eta_p}^2 \\ & & \end{array}$		mean (ms)	95% CI Lower Upper			
Speech (fre*dur)	1,33	3.391	.075	.093			
High120					328	317	338
High220					330	320	339
Low120					336	325	347
Low220					323	313	333
SWS (fre*dur)	1,33	.740	.396	.022			
High120					323	314	331
High220					317	309	326
Low120					317	309	324
Low220					318	307	328

7.3.3 Correlations

In order to investigate the relationship between different experimental variables the following correlation analyses were conducted: 1) MMN peak and mean amplitude, 2) behavioural discrimination, and MMN peak amplitude/latency in speech and SWS modes separately, 3) discrimination sensitivity, MMN amplitude/latency, IQ, age, grammar and vocabulary scores.

The results showed that, first of all, as expected, MMN mean and peak amplitudes were significantly correlated both across and within groups (only across groups correlations reported) (**Speech**: r=.905, p=<.001, N=34; **SWS**: r=.909, p=<.001, N=34), accounting

for 82% and 83% of the variation, respectively. Secondly, the MMN parameters did not correlate with the behavioural d' measure and none of these experimental variables were correlated with IQ, age, grammar or vocabulary scores (p>.10).

However, in order to further explore the behavioural discrimination sensitivity, correlations of speech and SWS discrimination were investigated for CA, SLI and dyslexia groups separately. The results showed that the correlation between speech and SWS is significant in the SLI and dyslexia groups (SLI: r=.852, p=.002, N=10; DYS: r=.800, p=.003, N=11) accounting for 73% and 64% of the total variation. In CA group, however, the speech and SWS discrimination were not significantly correlated (r=.318, p=.314, N=12, NS).

7.4 Conclusion and discussion

The behavioural data indicates that neither SLI nor dyslexia is caused by impaired ability to discriminate auditory events containing rapid transitions. Evidence against the auditory processing account comes from the fact that the stimuli used in the present study consisted of speech and non-speech (sinewave speech, SWS) stimuli that *both* contained rapid formant transitions claimed to be difficult for individuals with SLI and dyslexia (Tallal et al., 1996). Moreover, in the present experiment, these formant transitions were masked by the preceding vowel, thus making the discrimination of these contrasts harder. Furthermore, the non-matched adults had lower (absolute) d'-values than the SLI group probably suggesting that group-level differences would not have been obtained even by increasing the sample size. The response strategies between CA, SLI and dyslexia groups were also identical.

Overall, in the behavioural condition, speech was generally easier to discriminate (for all groups) suggesting that whenever attention is focused on the stimuli, speech may have a "special status". In other words, it is probably easier for the listener to focus on the critical acoustic cues in the signal when one has a linguistic label for it. SWS, on the

other hand, was perceived as random noise and probably because of the lack of these linguistic labels listeners could not focus on any specific cues as easily as in the speech condition. Among the acoustic cues (i.e., duration and frequency cues) used in the study, in the duration condition, the SLI group showed distinction of long/short duration that was not present in CA and dyslexia groups. These results do not indicate, however, that the SLI group would be impaired in processing duration as their d'-values were within normal limits.

The distribution of individual scores, however, shows that in the speech condition, both the SLI and dyslexia groups contained larger proportion of poor performers compared to the control group. This might indicate that if there is an input-processing deficit present in (some individuals with) SLI or dyslexia, it could be more apparent in speech processing than in the domain of general auditory processing.

Furthermore, in addition to the behavioural data, the electrophysiological data (N1 and MMN) indicate that neither SLI nor dyslexia is caused by a general deficit in sensory processing. The N1 response of auditory stimuli did not differ between the CA, SLI and dyslexic groups. The N1 peak amplitude was somewhat larger for non-speech stimuli whereas the N1 latency was significantly shorter for speech stimuli indicating either early differentiation of speech and other acoustic events or effects of stimulus complexity on the N1 parameters.

The MMN response was quantified by two different methods: peak amplitude/latency and mean amplitude. As expected, the peak and mean amplitudes showed similar effects. Overall, in the amplitude measures, there were no group differences nor there were any effects of mode (speech vs. non-speech) or topographical differences between groups and/or mode. The effects of stimulus 'familiarity' (i.e., typical vs. atypical stimuli in speech) were marginal and apparent only in the CA group. Moreover, there were no group differences in the MMN latency. Overall, the SWS stimuli elicited faster latencies.

Furthermore, the results showed no significant correlations between preattentive and behavioural discrimination (even when the paradigms were nearly the same) or with any of the experimental manipulations and language measures, age or IQ. Surprisingly, however, the behavioural discrimination of speech and non-speech was highly correlated in the SLI and dyslexic groups whereas there was no correlation between these two parameters in the CA group. This rather unexpected result could indicate that in the behavioural discrimination, there is a true "mode effect" in the CA group (i.e., speech discrimination is distinct from non-speech discrimination) whereas individuals with SLI and dyslexia process both sets of stimuli via either "speech mode" or "auditory mode", speech tokens being more informative for them overall.

To summarize, together these data suggest that neither SLI nor dyslexia is caused by auditory or speech processing deficit, nor is there any indication that individuals with SLI or dyslexia are slow processors. However, the SLI group failed to elicit significant MMNs to several of the presented sound contrasts indicating large within-group variation in the data. This might, again, suggest that there may be a subgroup of individuals in the SLI group showing auditory or speech processing problems.

Moreover, when looking at the relationship between preattentive and behavioural stimulus processing, the present study showed a distinct pattern for speech and non-speech. The results indicate that speech is easier to discriminate when attention is involved. However, the (simpler) non-speech sounds elicit faster brain responses when the focus of attention is directed elsewhere. Together these data could suggest that behavioural discrimination ability is distinct from automatic sensory discrimination (or change detection) possibly due to attention and task demands. Different behavioural discrimination tasks tax, for example, short-term memory to different degree. Moreover, by focusing attention to the stimuli, the listener may be able to make use of some perceptual cues that are not necessarily most prominent (or 'informative') for the preattentive sensory system.

8. Auditory memory trace formation in SLI and dyslexia

8.1 Introduction

As established before (see Chapter 4), one influential account of the Mismatch negativity (MMN) response reflects the functioning and accuracy of short-term auditory memory that stores newly received information. According to Näätänen & Winkler (1999), the mismatch response reflects perceptual analysis and comparison of the newly presented sensory input (*deviant*) with the echoic memory trace created by the previously presented stimuli (*standard*). In other words, the MMN response can be seen as a response to an auditory regularity violation where the deviant stimulus breaks the repetition 'rule'. Importantly, to elicit a MMN response, the standard stimuli do not have to be acoustically constant as long as they share a pattern or a rule (such as direction of frequency change or phonological category, see e.g., Saarinen, Paavilainen, Schröger, Tervaniemi, & Näätänen, 1992; Shestakova et al. 2002).

There is also a considerable amount of evidence showing that the traces involved in the MMN generation or change detection process could be linked to long-term memory representations (see Näätänen & Winkler, 1999 for a review). These long-term memory traces require attention to develop after which they respond automatically to familiar sounds. These long-term memory effects have been demonstrated for native and foreign language vowel phonemes (Näätänen et al. 1997; Winkler, Kujala et al. 1999). Moreover, Ceponine and colleagues (1999) demonstrated that sensory trace quality (and not trace duration) plays a role in successful phonological coding in 7-9 year old typically developing children. Furthermore, it has been shown that the strength of the memory trace (and the mismatch response) is proportional to the number of standard stimuli presented suggesting that the memory trace is enhanced by the repetition (Näätänen, 1992). Interestingly, this enhancement effect can already be observed after as few as two repetitions of standard stimuli (Huotilainen, Kujala, Alku, 2001; Bendixen,

Roeber, & Schröger, 2007). Huotilainen and colleagues (2001) argued that these long-term memory traces can facilitate the formation of short-term memory traces. They demonstrated that familiar phonemes are responded to faster and more strongly than non-familiar speech sounds or tones. Huotilainen et al. (2001) reported that familiar vowels elicit a prominent MMN response already after 'few' (2-3) repetitions of standards whereas unfamiliar vowel-like tokens and tones required 'many' (4-5) repetitions of standards for MMN elicitation.

In short, it has been demonstrated that MMN generation is dependent on both short-term and long-term memory trace activation. It has also been argued that long-term traces facilitate the formation of short-term traces thus generating more prominent and earlier MMN responses to native language speech sounds. However, the vast majority of MMN studies on native and foreign language speech sound perception are conducted on vowels (e.g., Näätänen et al. 1997; Aaltonen, Eerola, Hellström, Uusipaikka, & Lang, 1997; Cheour et al. 1998; Winkler, Kujala et al. 1999; Shestakova et al. 2002). These studies strongly suggest that the MMN response is not only dependent on the acoustic deviance but on the phonological status and category membership of the stimuli. In other words, the MMN response reflects categorical perception of vowels in native language as opposed to foreign language. Interestingly, however, the results obtained from consonants are much less clear cut (Sharma et al., 1993; Maiste, Wiens, Hunt, Scherg, & Picton, 1995; Dehane-Lambertz, 1997; Shafer et al. 2004). Moreover, as stated in Chapters 2 and 7, the results of studies on the accuracy of auditory and speech perception in SLI and dyslexia have been variable (see Bishop, 2007 for a review). Furthermore, the previous experiment of the current thesis (Chapter 7) failed to find significant differences between SLI, dyslexia and control participants in preattentive and attentive speech sound discrimination tasks. The SLI group, however, showed greater overall variability and less prominent MMNs for several sound contrasts used in the experiment.

Therefore, in the present experiment I investigate:

1) Memory trace formation for speech and complex non-speech (sinewave speech, SWS) stimuli in SLI and dyslexia.

Huotilainen et al. (2001) showed that fewer repetitions of standards were needed to form an adequate memory trace for MMN elicitation for familiar native language speech sounds as opposed to unfamiliar speech sounds and non-speech stimuli. In other words, familiar speech sounds showed faster trace development than unfamiliar sounds. Moreover, the accuracy of the neural representation of speech sounds seems to be linked with phonological coding as measured by pseudoword repetition ability (Ceponine et al. 1999). SLI and/or dyslexia, on the other hand, have been proposed to stem from phonological processing deficits caused by inaccurate or less categorical phoneme representations (Sussman, 1993; Ramus, 2003, Shafer et al. 2005). The exact nature of this phonological deficit, however, is still largely unknown (Bishop & Snowling, 2004). Therefore, the present experiment investigates the speed of the trace formation for both speech and non-speech in SLI, dyslexic and control participants.

I hypothesize that due to the underlying phonological deficits, individuals with SLI/dyslexia could lack the advantage of the existing long-term memory trace observed in non-impaired individuals. Furthermore, based on Huotilainen et al. (2001), I predict that: 1) In control participants, speech sounds show faster memory trace development (earlier and larger MMN response) than non-speech sounds (i.e., 'few' repetition: speech#non-speech and 'many' repetitions: speech#non-speech), 2) The SLI and dyslexic participants, however, lack this advantage of the existing trace (i.e., 'few' repetitions: speech#non-speech and 'many' repetitions: speech#non-speech).

2) **Memory trace formation for consonants.** The current experiment uses speech and acoustically complex non-speech (sine wave speech, SWS) in CVC stimuli where the critical change is in the final *consonant*. The aim is to investigate if the results by Huotilainen et al. (2001) are replicable in using consonants and complex non-speech stimuli. However, because there were no 'familiarity' effects in the previous study (Chapter 7), the present experiment investigates the processing of speech vs. non-speech across all four (High120, High220, Low120, low220) stimulus types.

In short, the rationale of the current experiment and paradigm is as follows: once the target (deviant) sound is presented it becomes the new standard. This standard is then repeated either 'few' (from two to three) or 'many' (from four to five) times (see Figure 7.2 in Chapter 7) before the next deviant appears. Interestingly, Huotilainen and colleagues (2001) reported that memory trace formation is faster for native language prototypes than for non-prototypes. In other words, the number of standards that are required to produce a prominent MMN depends on the linguistic status of the stimuli.

8.2 Method

8.2.1 Participants

Same participants as in previous study (Chapter 7) took part in this study.

8.2.2 Data analysis

In the present experiment, the data from the previous study (Chapter 7) is re-analyzed by classifying the EEG responses to the deviant stimuli into two exclusive categories on the basis of their presentation frequency, that is, a deviant occurring after either 'few' or 'many' repetitions of the standard stimulus.

In other words, the experimental task and stimuli in the current experiment are identical with the MMN paradigm reported in the previous chapter, i.e., the same stimuli (speech and non-speech) and the same paradigm (roving-standard, SOA 800 ms, total 2160

stimuli; see Huotilainen et al. 2001; Shestakova et al. 2002; Baldeweg et al. 2004) as reported in Chapter 7 were used in the EEG recording.

To recap, in the roving-standard paradigm all four stimuli ([bɒt], [bot/d], [bod/t], [bɔ:d]) are standards and deviants. In other words, each of the four deviants becomes a standard stimulus thus avoiding the need for control conditions (i.e., presenting deviants in isolation or reversing the presentation order) required by the standard oddball paradigms. The same procedure is applied to the SWS stimuli presented in a separate block (see description in Chapter 7).

In the data analysis, those standards immediately following a deviant are removed from the average following the procedure by Huotilainen et al. (2001) and Shestakova et al. (2002). Therefore, only the third stimulus of each new sequence is considered as standard.

A total of 480 deviants (240 deviants in both the 'few' and 'many' categories) and 660 standard stimuli were presented. The standards immediately following a deviant were removed from the analysis. EEG recording procedures are reported in Chapter 7. Because no clear and identifiable peak could be detected in some conditions, ERP data analyses were done on mean amplitudes from the deviant – standard difference waves in three 50 ms time-windows (i.e., 230-280, 280-330, 330-380 ms, respectively, see Figures 8.1-8.4, see also Peltola et al. 2003). The analysis time-windows were placed around the amplitude maxima of the grand average waveform. Seven electrodes (Fz, Fcz, Cz, F3, F4, C3, C4) were chosen for initial statistical analyses [repeated measures ANOVA: Electrode (7), Time (3), Repetition (2), Mode (2), Group (3)]. Similarly to the previous study (Chapter 7), if no interactions between group/mode and electrode position (i.e., topographical differences) were identified analyses were conducted on a single central electrode that showed the most significant MMN amplitudes (Fcz).

8.3 Results

Grand-averaged MMN waveforms (at Fcz) are represented in Figures 8.1 and 8.2 (speech and non-speech) and the corresponding difference waves in Figures 8.3 and 8.4 (speech and non-speech).

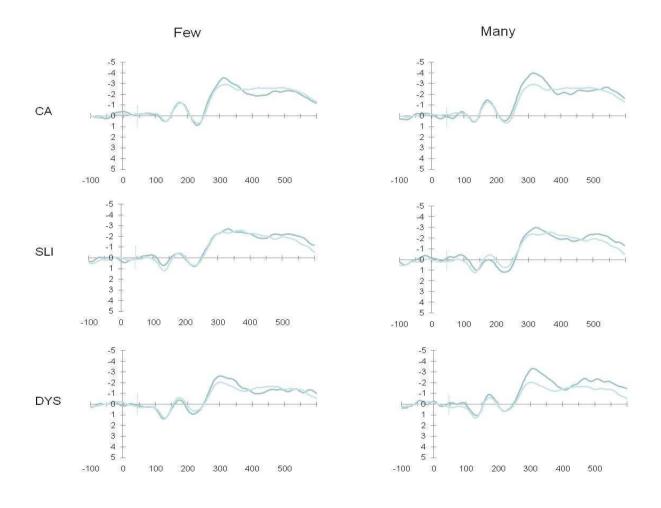


Figure 8.1. Grand-averaged ERP waveforms at Fcz for standard (light blue) and deviant (dark blue) in CA, SLI and dyslexia groups for 'few' and 'many' repetitions of standards for speech stimuli.

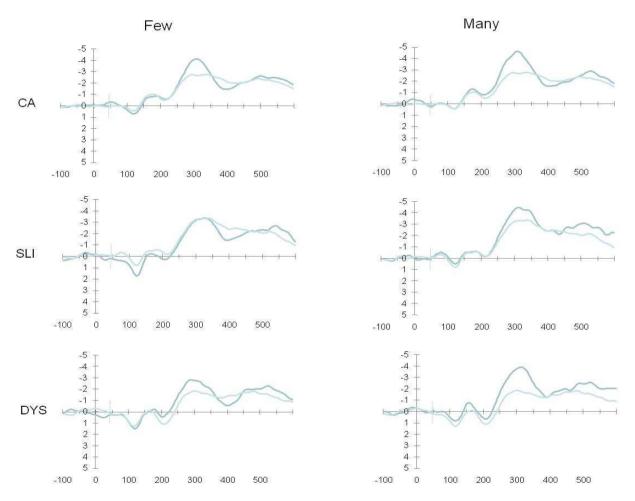


Figure 8.2. Grand-averaged ERP waveforms at Fcz for standard (light blue) and deviant (dark blue) in CA, SLI and dyslexia groups for 'few' and 'many' repetitions of standards for SWS stimuli.

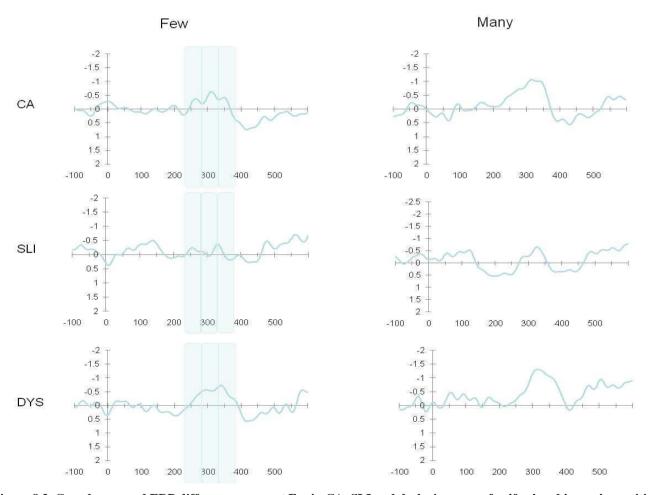


Figure 8.3. Grand-averaged ERP difference waves at Fcz in CA, SLI and dyslexia groups for 'few' and 'many' repetitions of standards for speech stimuli. The three time-windows for statistical analyses are highlighted in the left panel.

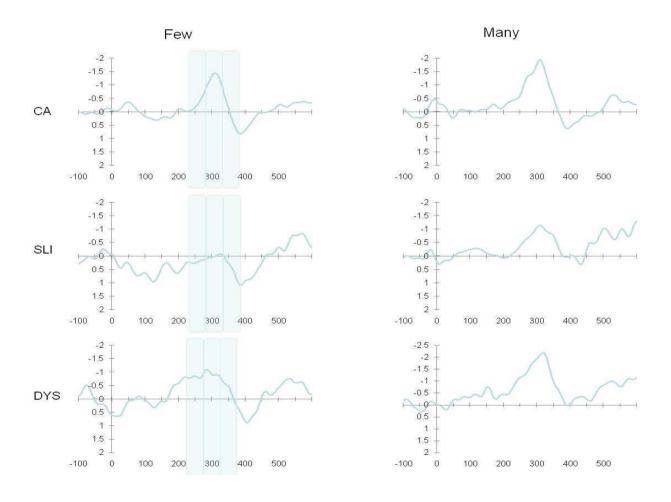


Figure 8.4. Grand-averaged ERP difference waves at Fcz in CA, SLI and dyslexia groups for 'few' and 'many' repetitions of standards for sws stimuli. The three time-windows for statistical analyses are highlighted in the left panel.

The repeated-measures ANOVA showed statistically significant main effects of **Repetition** (many>few), **Time** $(T_2>T_3=T_1)$ and **Electrode** (Fz, Fcz, Cz, F3, F4, C3, C4) (see Table 8.1 for statistical details).

Table 8.1: Main effects of Repetition, Time, Electrode and Group (see text for more details).

	df	F	p	η_p^2	3	Mean (µV)		6 CI Upper
Repetition	1,31	12.336	.001	.285		4 /		
Few						-0.31	-0.54	-0.08
Many						-0.62	-0.83	-0.41
Time	2,62	14.292	<.001	.316				
T_1						-0.33	-0.52	-0.14
T_2						-0.69	-0.93	-0.44
T_3						-0.38	-0.59	-0.16
Electrode	6,186	4.832	.001	.135	.646			
Group	2,31	1.700	.199	.099				

Furthermore, the following interactions (with factor "Electrode") were significant: **Electrode*Repetition** [F(6,186)=3.693, p=.009, η_p^2 =.106, ϵ =.612] and **Electrode*Time** [F(12, 372)=4.634, p=<.001, η_p^2 =.130, ϵ =.439] (see Appendix G: Figures A and B for details). This interaction between electrode location and time-window/repetition was due to posterior electrodes (C3, Cz, C4) not eliciting effects of similar magnitude to frontal/central electrodes. As stated above, when no interaction between electrode location and mode/group are found, the analyses are conducted at a single electrode site showing most reliable MMN amplitude (Fcz).

The subsequent analysis at Fcz showed statistically significant main effects of **Repetition** (2: few vs. many) and **Time** (3: T_1 , T_2 , T_3). The main effect of **Group** (3: CA, SLI and DYS) approached statistical significance (see Table 8.2 for statistics and Table 8.3 for mean amplitudes).

For **Repetition** it was shown that 'many' repetitions of standard elicited larger MMN responses than 'few' repetitions of standard. Pairwise comparisons for **Time** revealed that T_2 differed significantly both from T_1 and T_3 (p<.001) but T_1 and T_3 did not differ from one another (p=.460). For **Group**, the post hoc analysis (LSD) showed that the SLI group differed significantly from the dyslexia group (p=.034) but not from the control group (p=.119). The control and dyslexia group did not show statistically different responses (p=.515).

Table 8.2: Main effects of Repetition, Time and Group (see text for more details).

	df	F	p	η_p^2	3	Mean (μV)		6 CI Upper
Repetition	1,31	19.180	<.001	.382		- \f- /		
Few						-0.31	-0.62	-0.01
Many						-0.79	-1.02	-0.56
Time	2,62	13.667	<.001	.306	.902			
T_1						-0.35	-0.59	-0.11
T_2						-0.86	-1.17	-0.55
T_3						-0.44	-0.71	-0.17
Group	2,31	2.604	.090	.144				
CA						-0.64	-1.06	-0.23
SLI						-0.17	-0.61	0.26
DYS						-0.84	-1.27	-0.40

Furthermore, the interactions of **Mode*Repetition** [F(1,31)=4.769, p=.037, η_p^2 =.133], **Mode*Time** [F(2,62)=6.707, p=.006, η_p^2 =.178, ϵ =.739], and **Repetition*Time** [F(2,62)=9.193, p=<.001, η_p^2 =.229, ϵ =.958] were statistically significant (see Figure 8.5).

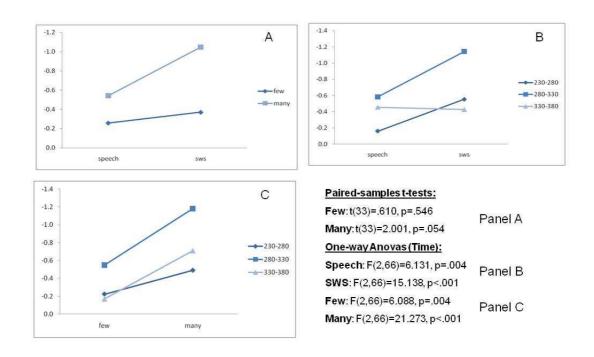


Figure 8.5 Interactions for Mode*Repetition (A), Mode*Time (B) and Repetition*Time (C).

For the **Mode*Repetition** interaction, a paired sample t-test showed that speech and non-speech elicit (marginally) different responses only after 'many' repetitions of standard (see Figure 8.5, Panel A). One-way ANOVA for **Time** (Panel B) and follow-up pairwise comparisons (LSD) for the **Mode*Time** interaction showed that in speech the MMN response was largest in the two later time-windows (T_1 - T_2 : p=.001 T_1 - T_3 : p=.045 and T_2 - T_3 : p=.287, NS.) whereas, in SWS, the MMN response was largest at the second time-window (T_1 - T_2 : p<.001 T_1 - T_3 : p=.446, NS and T_2 - T_3 : p<.001). Finally, One-way ANOVA for **Repetition*Time** interaction showed that T_2 > T_1 = T_3 after 'few' repetitions (T_1 - T_2 : p=.006 T_1 - T_3 : p=.686, NS and T_2 - T_3 : p=.003) whereas T_2 > T_3 > T_1 after 'many' repetitions (T_1 - T_2 : p<.001 T_1 - T_3 : p=.076, and T_2 - T_3 : p<.001).

Moreover, the interaction **Repetition*Time*Group** approached statistical significance $[F(4,62)=2.376, p=.062, \eta_p^2=.133]$ (see Table 8.3 for mean amplitudes). The follow-up tests (repeated measures ANOVA) for each group separately revealed significant main

effects of **Repetition** [F(1,11)=8.579, p=.014, η_p^2 =.438] and **Time** [F(2,22)=8.898, p=.001, η_p^2 =.447] in the CA group. In the SLI group, the main effect of **Repetition** [F(1,10)=4.007, p=.073, η_p^2 =.286] and **Repetition*Time** interaction [F(2,20)=3.421, p=.053, η_p^2 =.255] approached statistical significance. Finally, in the dyslexia group the main effects of **Repetition** [F(1,10)=7.880, p=.019, η_p^2 =.441] and **Time** [F(2,20)=3.573, p=.047, η_p^2 =.263] and the **Repetition*Time** interaction [F(2,20)=12.132, p<.001, η_p^2 =.548] were all statistically significant.

Table 8.3. Mean amplitudes of difference waves in three time-windows for CA, SLI and DYS at Fcz electrode. Values are in μV (SD).

	SPEECH				SWS					SPEECH	SWS			
		Few			Many	_		Few			Many		Total	Total
	T_1	T ₂	T ₃	T_1	T_2	T ₃	T_1	T_2	T ₃	T_1	T ₂	T ₃		
CA	16	13	23	51	92	69	29	-1.24	18	82	-1.68	57	49	80
	(1.00)	(1.02)	(1.25)	(.70)	(.90)	(1.13)	(.48)	(1.00)	(.93)	(.74)	(.92)	(.88)	(1.00)	(0.83)
SLI	-0.12	47	06	.29	38	18	.21	.01	.31	43	-1.00	63	09	25
	(1.49)	(1.15)	(1.21)	(1.05)	(1.03)	(1.25)	(1.08)	(1.35)	(1.28)	(.95)	(.84)	(1.07)	(1.20)	(1.10)
DYS	16	08	52	27	-1.11	-1.04	83	94	34	-1.16	-1.97	-1.16	61	-1.07
	(.83)	(.79)	(.80)	(.89)	(1.25)	(1.16)	(1.37)	(1.22)	(1.31)	(1.48)	(1.82)	(1.53)	(0.95)	(1.46)
Total	15	36	27	17	81	64	30	74	07	81	-1.55	78	40	71
	(1.09)	(1.14)	(1.09)	(.93)	(1.08)	(1.19)	(1.09)	(1.28)	(1.18)	(1.10)	(1.29)	(1.18)	(1.09)	(1.19)

As mentioned above, the **Repetition*Time** interaction was extremely significant in the dyslexia group and approached significance in the SLI group but not in the CA group (p=.995) (see Appendix F Figure C for details). This interaction is due to both SLI and dyslexia groups eliciting larger MMN amplitudes after 'Many' repetitions of standards in later time-windows (i.e., in T₃: 330-380ms) whereas in the CA group the MMN response was most prominent in the second time-window (T₂: 280-330ms).

Overall, all groups elicited larger MMN amplitudes for deviants after 'many' repetitions of standard (i.e., main effects of **Repetition**). However, the SLI group showed only a statistical trend for this effect. CA and dyslexia groups showed statistically significant effects for **Time** (standard error in the parenthesis) [**CA**: **T**₁: -.44 μ V(.20), **T**₂: -1.07 μ V(.26), **T**₃: -.42 μ V(.22); LSD: **T**₁-**T**₂: p=.002; **T**₂-**T**₃: .004; **DYS**: **T**₁: -.61 μ V(.21), **T**₂: -1.14 μ V(.27), **T**₃: -.77 μ V(.23); LSD: **T**₁-**T**₂: p=.007] both groups eliciting the largest response in the second time-window that differed from both the first and second time-windows in the CA group and from the first time-window in the dyslexia group.

Because the second time-window (T_2) elicited the largest response in the control group, a further comparison was conducted in this time-window. Repeated measures ANOVA for T_2 only showed significant main effects of **Repetition** and **Mode**. The **Group** main effect approached significance (see Table 8.4 for statistics). Furthermore, the **Repetition*Mode** interaction was also statistically significant [F(1,31)=4.212, p=.049, $\eta_p^2=.120$].

Table 8.4: Main effects of Repetition, Mode and Group in T₂.

	df	F	p	η_p^{-2}	Mean (µV)	95% CI Lower Upper
Repetition	1,31	27.868	<.00	1 .473	4 /	
Few					-0.54	-0.95 -0.18
Many					-1.18	-1.48 -0.87
Mode	1,31	6.848	.014	.181		
Speech					-0.58	-0.93 -0.22
SWS					-1.14	-1.53 -0.74
Group	2,31	2.576	.092	.143		
CA					-1.07	-1.60 -0.54
SLI					-0.36	-0.91 0.19
DYS					-1.14	-1.70 -0.59

The Post Hoc analysis (LSD) revealed that the SLI group differed significantly from dyslexic group (p=.049) and the difference between SLI and control group approached significance (p=.068). The CA group did not differ from dyslexic group (p=.845).

The **Repetition*Mode** interaction was caused by speech and non-speech differing significantly after 'many' repetitions [t(33)=2.862, P=.007; **Speech:** -0.81 μ V (1.08); **SWS:** -1.55 μ V (1.29)] but only marginally so after 'few' repetitions [t(33)=1.949, P=.060; **Speech:** -0.36 μ V (1.14); **SWS:** -0.74 μ V (1.28)].

8.4 Discussion and conclusion

To summarize the results, first of all, the mismatch response was generally larger after 'many' repetitions of standards both with speech and SWS stimuli. However, in the SLI group this effect was only marginally significant. Secondly, the SLI group showed a trend in eliciting generally smaller MMN responses than dyslexic and control groups. Thirdly, the MMN response was most prominent in the medial time-window (i.e., 280-330 ms). Finally, somewhat surprisingly, contrary to previous findings of Huotilainen and colleagues (2001), speech and non-speech did not differ overall. Moreover, speech and non-speech did not differ after 'few' repetitions of standards whereas they differed after 'many' repetitions of standards. In other words, the early long-term memory trace facilitation for speech sounds was not observed in the present study (see Section 8.1). This finding could be due to two factors: 1) the memory trace formation and facilitation effects are less prominent for consonants (Sharma et al., 1993; Shafer et al. 2004) or 2) this particular speech contrast was more difficult to perceive since the difference occurred syllable-finally and was masked by the preceding vowel. Lastly, in the speech condition, the MMNs were, overall, more distributed in time in that the speech sounds elicited MMNs also in the later time-window (330-380 ms). Time and repetition analyses showed that the control group elicited identical MMN responses after 'few' and 'many' repetitions as a function of time (many>few) whereas in the SLI and dyslexia groups the MMN responses after 'many' repetitions were delayed or sustained longer.

In short, these results do not fully confirm the predictions outlined in section 8.1. However, as in the previous study in Chapter 7, the SLI group tended to show more variation, less prominent MMN elicitation and less prominent repetition effects than

controls and dyslexics. Moreover, both the SLI group and dyslexic group showed longer MMN responses after 'many' repetition of standards. These results could be further explored by using vowels (where MMN results have been more prominent) and a wider range of standard repetitions (e.g., as in Baldeweg et al. 2004).

9. General discussion and conclusion

In this chapter I recapitulate the results of the three experiments in this thesis. After establishing that I discuss how they relate to the initial research questions in Chapter 5. Lastly, I will present some directions for the future investigations of the issues raised in this thesis.

Cue weighting and CP in SLI and dyslexia

- Duration was the main cue for syllable final consonant voicing and first formant frequency (F1) was not used by CA, SLI or DYS groups. The SLI group, however, weighted the duration cue less heavily than other groups.
- Non-matched adults used both the duration and frequency cue in determining the syllable final consonant voicing.
- The individual data analyses revealed that 23% of the SLI group and 11% of the DYS group were outliers on the duration cue condition, and 15% of the SLI group and 9% of the DYS group were outliers on the F1 cue condition. These results indicate that a subgroup of individuals with SLI (and dyslexia) may have incorrectly weighted phonological representations.
- On measures of Categorical Perception (boundary and slope) the groups, overall, did not differ from one another in the boundary measure. Of the SLI group, however, 31 % performed poorly on this measure. In the slope measure, SLI group showed shallower categorization functions than both DYS and CA groups. Moreover, individual data analysis showed that 12% of the SLI group performed poorly on this slope measure. These results could indicate that dyslexia is not caused by a CP deficit. Moreover, a CP deficit is more likely to accompany SLI. However, the present study suffered from few drawbacks. Firstly, the current sample consisted of older individuals with language impairments. Secondly, the dyslexia group was a high functioning group (see participant

details in Chapter 6 and 7). Therefore, the issue of a CP deficit as a core deficit in dyslexia and/or SLI is only properly addressed by, firstly, investigating younger individuals to account for the developmental changes and, secondly, by investigating less compensated individuals.

Auditory processing in SLI and dyslexia

- In behavioural discrimination speech was generally easier to discriminate than non-speech. These results could indicate that familiar stimuli (or stimuli that have linguistic labels) are easier to discriminate despite speech being acoustically more complex than non-speech. Further research, however, is needed.
- The three groups (CA, SLI and DYS) did not differ overall in their discrimination sensitivity. These results indicate that an underlying auditory processing deficit is unlikely to cause SLI or dyslexia.
- Individual data analyses, however, showed that 36% of the SLI and 18% of the DYS group were outliers on speech discrimination and 18% of SLI and 9% of the DYS group were outliers on non-speech discrimination. These results indicate that a subgroup of individuals with SLI and dyslexia may have underlying auditory or speech processing deficits.
- There was a high correlation between speech and non-speech discrimination in SLI and DYS groups but not in the CA group indicating that genuine 'mode' effects were only observed in the CA group.
- N1 and MMN amplitudes and latencies did not differ between groups. However, one must be cautious in interpreting the current results because of the large age distributions in the experimental groups. One must bear in mind that there are significant age-related changes especially in the N1 component of the auditory evoke potentials (e.g., in its morphology and latency) and these maturational changes seem to continue well into the second decade of life (see e.g., Sharma, Kraus, McGee & Nicol, 1997). Therefore, grouping together individuals who are between 14-25 years of age will most likely

camouflage any such effects (e.g., differential maturation of auditory function between groups) (see also McArthur & Bishop, 2005).

- The SLI group failed to elicit a significant MMN response to several experimental conditions.
- MMN amplitude and latency measures were not correlated with behavioural discrimination sensitivity indicating that by focusing attention to the stimuli the listener is able to make use of different acoustic cues than those used in the preattentive (automatic) condition.

Memory trace formation in SLI and dyslexia

- Speech and non-speech did not differ overall.
- 'Many' repetitions of standard stimuli elicited larger MMN responses. In the SLI group, however, the effect of repetition ('few' vs. 'many') approached statistical significance.
- The SLI group elicited smaller MMN responses than the DYS group.
- In the SLI and DYS groups the MMN response sustained longer than in the CA group after 'many' repetitions.
- Faster memory trace formation was present in the non-speech stimuli than in the speech stimuli indicating that early long-term memory trace facilitation for speech sounds does not take place in consonants. These results are in contradiction with previous studies using vowels (Huotilainen et al. 2001).

These were the main results from the three experiments conducted in this thesis. The results are presented with the original questions (see Chapter 5) below:

1) Is SLI associated with persistent auditory deficits and how many individuals with SLI still show these deficits in (early) adulthood? *Approximately* 10-30% of

individuals with SLI still show auditory processing problems in some auditory tasks in (early) adulthood.

- 2) Is dyslexia associated with persistent auditory deficits and how many individuals with dyslexia still show these deficits in (early) adulthood? Approximately 9-20% of individuals with dyslexia still show auditory processing problems in some auditory tasks in (early) adulthood.
- 3) If SLI and dyslexia are associated with auditory problems, are the auditory deficits different in SLI and dyslexia? *Yes*.
- 4) Is the auditory deficit specific to speech? *No*.
- 5) Is the input-processing deficit a consequence of inability to focus on relevant acoustic cues?
 - *Yes*. The SLI group weighted the duration cue less than dyslexics and controls. Moreover, consonant perception was less categorical in young adults with SLI than in controls or dyslexics.
- 6) Is the input-processing deficit a consequence of general inability to discriminate sounds?
 - *Not really*. Larger proportion of individuals with SLI, however, was outliers on speech and non-speech discrimination than of controls and dyslexics. However, high correlation between these measures indicates that the 'deficit' is not necessarily specific to speech. Moreover, the SLI group failed to elicit significant MMN response to several experimental conditions indicating that auditory problems can be present in some individuals with SLI.
- 7) Is the input-processing deficit a consequence of deficient memory trace formation (e.g., slower encoding of auditory trace)?

- *Could be*. Overall, SLI group elicited smaller MMN responses than dyslexic group. Moreover, SLI group showed only a statistical trend for standard stimuli repetition effects indicating slower memory trace encoding in (some) individuals with SLI.

Together these results reveal a complex pattern of behaviour. None of these measures, as such, differentiate SLI and dyslexia groups from controls in a straightforward manner. Neither do the present results lend support to the single-source models of SLI and dyslexia (see Chapter 4). In summary, the results showed that SLI/dyslexia are not poor in discriminating sounds with rapid formant transitions (whether speech or non-speech) as suggested by Tallal and colleagues (Tallal et al. 1996). Individuals with SLI (or a subgroup of them), however, may have incorrectly weighted phonological representations (Sussman, 1993, Shafer et al. 2004) and slower memory trace formation than individuals without oral language deficits. The individual data analyses also revealed that there was more variation (in all experiments) and more outliers in the SLI group than in the control or dyslexic groups. This may indicate that a small subgroup of the individuals with SLI may demonstrate auditory deficits. However, these data do not rule out the possibility that underlying auditory processing deficits may have been present during the childhood in all individuals with language impairment and resolved later in life (Bishop & Snowling, 2004). Therefore, further research is warranted.

For future research, in investigating cue-weighting, it is essential that synthetic, semi-synthetic and natural tokens embedded in real words and non-words in a more engaging task are included. Moreover, because the vocalic environment affects the acoustic realisation of consonants, several different vocalic environments should be included. Moreover, because consonants themselves differ in their acoustic realisation, several consonantal contrasts with different place and manner of articulations should be included.

In investigating speech and non-speech discrimination, the complexity of speech and non-speech tokens should be matched. However, it is challenging to create equally complex non-speech tokens that 1) contain the crucial features of the speech signal, 2) do not acoustically differ from the speech stimuli and, 3) do not sound like speech. The two most feasible options are to use either sine-wave speech analogues in speech and non-speech modes or to use distorted speech (e.g., noise-vocoded speech) (Remez et al. 1981). Moreover, in investigating memory trace formation, more sensitive standard repetition measures should be used (e.g., see Baldeweg, Klugman, Gruzelier, & Hirsch, 2004).

Moreover, stimulus selection is of vital importance. Behavioural data show that the difference between results obtained from vowels and consonants is striking. For example, effects of categorical perception are generally stronger for consonants than for vowels whereas other phenomena, such as magnet effect, are more prominent in vowels (see Chapter 4). Moreover, the internal structure of speech sound categories contains large amount of variation –and this is especially apparent in vowels. In other words, category boundaries and prototype judgements (e.g., goodness ratings) for vowels vary greatly between listeners (see Chapter 4 and Aaltonen et al. 1997 for behavioural and MMN study). In consonants, on the other hand, the surrounding vowel environment is of great importance for the acoustic realisation of a particular consonant. In short, contradicting results from different experiments may, at least partially, be due to stimulus selection because the acoustic and auditory properties of vowels and consonants are different.

10. Appendices

Appendix A_1 : Study participation details:

ID	Group	Ex	perime	ents ¹	
		1	2	3	
S010	SLI	Y	Y	Y	
S013	SLI	Y	Y	Y	
S015	SLI	Y	Y	Y	
S017	SLI	Y	Y	Y	
S024	SLI	Y	Y	Y	
S025	SLI	Y	Y	Y	
S031	SLI	Y	Y	Y	
S032	SLI	Y	Y	Y	
S067	SLI	Y	Y	Y	
S071	SLI	Y	Y	Y	
S112	SLI	Y	Y	Y	
S116	SLI	Y	Y	Y	
S049	SLI			Y	
S117	DYS	Y	Y	Y	
S118	DYS	Y	Y	Y	
S119	DYS	Y	Y	Y	
S120	DYS	Y	Y	Y	
S121	DYS	Y	Y	Y	
S122	DYS	Y	Y	Y	
S124	DYS	Y	Y	Y	
S125	DYS	Y	Y	Y	
S126	DYS	Y	Y	Y	
S127	DYS		Y	Y	
S128	DYS	Y	Y	Y	
S129	DYS	Y	Y	Y	
AO74	CA	Y	Y	Y	
A076	CA	Y	Y	Y	
A082	CA	Y	Y	Y	
A083	CA	Y	Y	Y	
A089	CA	Y	Y	Y	
A091	CA	Y	Y	Y	
A093	CA	Y	Y	Y	
A094	CA	Y	Y	Y	
A099	CA	Y	Y	Y	
A100	CA	Y	Y	Y	
A101	CA	Y	Y	Y	
A102	CA	Y	Y	Y	

A102 CA Y Y Y

Experiments: 1 = EEG (Exps 2 and 3), 2 = Discrimination (Exp 2), 3 = Cue weighting (Exp 1)

Appendix A₂: Background screening questions.

Dear [name of the participant],

I need to receive some further details of your background. On the basis of your answers, I'll try to form a group as soon as possible and will let you know immediately if you are suitable for the present experiment.

- Do you speak American English or British English?
- Were there any other languages spoken at home as a child? If yes, indicate which languages and estimate how much (e.g. 10%, 50%, 90% of the time) and by who (mother, father, both parents, sisters...).
- Do you speak any other languages than English? If yes, indicate which languages and where and how did you learn them?
- Have you suffered from any neurological condition (e.g., epilepsy) at any point?
- Do you have (or think you have) normal hearing? Have you had any hearing problems in the past?
- Have you (or anyone in your close family) ever received speech and language therapy? If yes, indicate who in your family, when and why.

Appendix B: Instructions for identification task

INSTRUCTIONS

IDENTIFICATION EXPERIMENT

In this experiment your task is to identify the sound you hear. You will be presented one

sound or "word" at a time and your task is to press the relevant key ([bot] or [bod], i.e. z

and m on the keyboard) to indicate which "word" you heard.

The words are made with an old speech synthesizer so they may sound a bit odd from

time to time but even if you are not exactly sure, just try to guess! And do not think

about your decision too much, there are no right or wrong answers here. The best thing

is to press the key as quickly as possible according to the first impression.

There are 140 stimuli in total (there's a calculator at the bottom right hand corner of the

screen), and the experiment takes about 5-7 minutes to run.

In the practice session you will hear 15 items and you can practice pressing the buttons

already. The items are [bot] [bod] [bod] [bod] ... so every other is [bot] where you press

keys z,m,z,m,z,m... Try to listen to these sounds carefully so you will get an idea what

kind of synthetic speech you will hear. In the actual experiment, however, the sounds

may be more ambiguous.

If you have any questions, please ask!

Good luck and thank you,

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Appendix C: Instructions for discrimination task

INSTRUCTIONS

DISCRIMINATION TASK: ACTIVE

This experiment consists of two (identical) tasks, each lasting approximately 5 minutes.

Your job is to detected change in a stream of sounds and press a key () as soon as possible when ever you hear the change. The sounds are presented in the following way (where each symbol represents a sound type):

----++++/////+++////---?????///??[....]

So in this case you would press button in the following places:

----<u>+</u>+++<u>/////+++////<u>-</u>--<u>?</u>?????///<u>?</u>?</u>

Imagine that these sounds are different vowels:

iiiii eeeeoooooeeeooooiiiaaaaaiiiaa[...]

So you'd hear a change in positions:

iiiii eeeeoooooeeeooooiiiaaaaaiiiaa

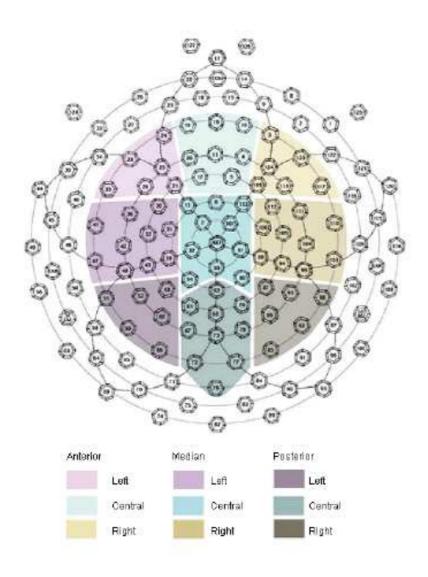
The program will not wait for you to answer so try to be as quick as possible and don't think too much; guessing is fine if you are not sure! And if you think you pressed in a wrong place, do not worry or try to correct yourself ---we have so many repetitions that it won't matter!

First you will have a short practice (32 items) after which the real experiment starts. There are 187 stimuli in the experiment in total –there's a calculator in the bottom right hand side of the screen.

If you have any questions, do not hesitate to ask.

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Appendix D: Regions of Interest (ROI, Experiment 2)



Appendix E: ROI analyses (Experiment 2)

Table A: Mean amplitude values (SE) in nine Region of Interest. AL=Anterior Left, AC=Anterior Central, AR=Anterior Right, ML=Medial Left, MC= Medial Central, MR= Medial Right, PL=Posterior Left, PC=Posterior Central, PR=Posterior Right

ROI	AL	AC	AR	ML	MC	MR	PL	PC	PR
μV	-1.9	-2.8	-2.2	-1.0	-1.9	-0.9	0.5	0.2	0.6
(SE)	(0.3)	(0.3)	(0.3)	(0.2)	(0.2)	(0.2)	(0.1)	(0.1)	(0.1)

Table B: Statistics for ROI main effects in Anterior-Posterior axis and Post Hoc (LSD) analyses.

ROI	df	\mathbf{F}	p
Anterior	2,68	31.103	<.001
Left vs. Central Left vs. Right Central vs. Right	2.0	52.012	<.001 .007 <.001
Medial Left vs. Central	2,68	52.913	< .001 .001
Left vs. Right Central vs. Right			NS* <.001
Posterior Posterior	2,68	7.332	.001
Left vs. Central Left vs. Right Central vs. Right			.002 NS* .003

^{*}p>.10

Table C: Statistics for ROI main effects in Left-Right axis and Post Hoc (LSD) analyses.

ROI		df	F	p
Left		2,68	55.294	<.001
	Anterior vs. Medial Anterior vs. Posterior Medial vs. Posterior			<.001 <.001 <.001
Centr	al	2,68	55.413	<.001
	Anterior vs. Medial Anterior vs. Posterior Medial vs. Posterior			<.001 <.001 .001
Right		2,68	60.006	<.001
	Anterior vs. Medial Anterior vs. Posterior Medial vs. Posterior			<.001 <.001 <.001

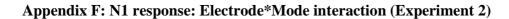
Table D: Statistics for ROI*Condition interaction and Post Hoc (LSD) analyses.

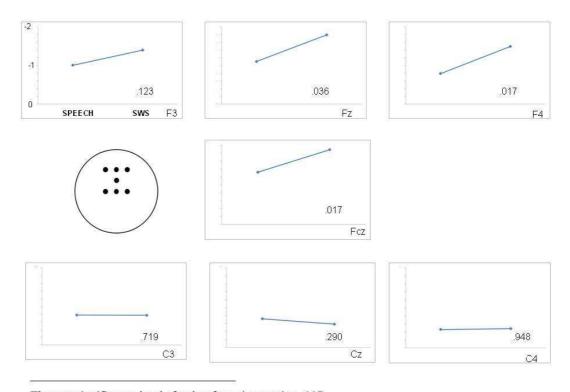
ROI	df	F	p
Anterior: Condition	1,34	52.500	<.001
Anterior*Condition	2,68	20.954	<.001
Medial: Condition	1,34	52.913	<.001
Medial*Condition	2,68	12.714	<.001
Posterior: Condition	1,34		NS*
Posterior*Condition	2,68		NS*

^{*} p>.10

Table E: Statistics for ROI*Condition interaction and Post Hoc analyses (LSD).

ROI	df	F	p
Left: Condition	1,34	30.467	<.001
Left*Condition	2,68	24.694	<.001
Central: Condition	1,34	52.913	<.001
Central*Condition	2,68	35.463	<.001
Right: Condition	1,34	21.376	<.001
Posterior*Condition	2,68	34.982	<.001





The new significance level after bonferroni correction .007

Figure A. N1 peak amplitudes (μV) for standard stimuli in speech and non-speech (SWS) in seven electrodes (the approximate location of electrodes is illustrated in the figure) and corresponding p-values from paired-samples t-test of speech *versus* SWS.

Appendix G: MMN response: Interactions (Experiment 3)

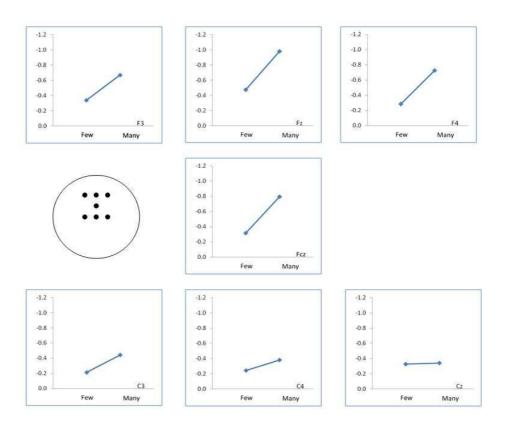


Figure A. Electrode*Repetition interaction: MMN mean amplitude (μV) for grand-averaged difference waves after 'few' and 'many' repetition in seven electrodes (the approximate location of electrodes is illustrated in the figure).

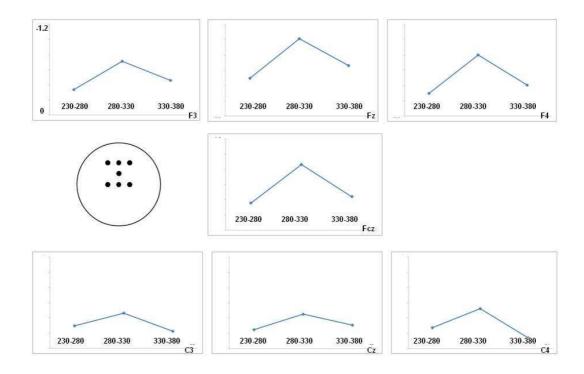


Figure B. Electrode*Time interaction: MMN mean amplitude (μV) for grand-averaged difference waves in T1 (230-280ms), T2 (280-330ms) and T3 (330-380ms) in seven electrodes (the approximate location of electrodes is illustrated in the figure).

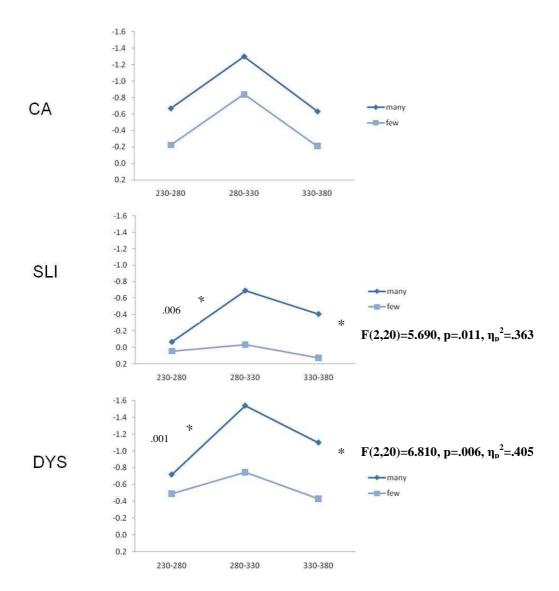


Figure C: Repetition*Time*Group interaction: MMN mean amplitudes (μV) for grand-mean difference waveforms by group (CA, SLI and dyslexia) and as a function of time, (230-280ms), T2 (280-330ms) and T3 (330-380ms). Significant contrasts indicated with an asterisk. Pairwise comparisons: LSD.

11. References

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