Neurophysiological correlates of word recognition in noise in adults and children

by

Nicholas K. Walker

B.A., University of Texas at Dallas

M.S., Montana State University

M.A., University of Colorado, Boulder

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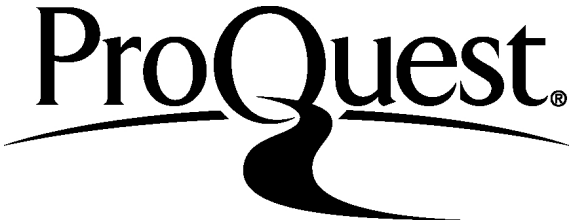

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Neurophysiological correlates of word recognition in noise in adults and children

Written by Nicholas K. Walker

Has been approved for the Department of Speech, Language, and Hearing Sciences

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Phillip M. Gilley, Ph.D.

_____

Christine Yoshinaga-Itano, Ph.D.

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Neurophysiological correlates of word recognition in noise in adults and children

Thesis directed by Professor Phillip M. Gilley

Abstract

During auditory processing of linguistic information listeners must overcome many challenges in order to interpret the meaning of the spoken language. Speech is rarely delivered without noise, and the auditory system must rapidly interpret the incoming speech signal. In this study I examined the effects of multitalker babble on speech perception in typical adults and children during a word recognition task. Continuous EEG was collected from 64 scalp channels while participants completed the tasks. EEG for each participant were analyzed using a continuous wavelet transform (CWT) using a fast-Fourier convolution. CWTs were computed at 256 log-spaced frequencies ranging from 3Hz to 100Hz. Intertrial phase clustering was computed across conditions. ITPC profiles were computed separately for noise and non-noise conditions, and statistical tests were performed for comparison.

Adults and children both had significant effects of noise on the N1 component, while neither had effects of noise on the P3 component amplitude suggesting that early components are more important for speech perception in noise. Phonotactic probability had an effect on the P3 component at the left temporal electrode site. While word frequency and noise had an effect on the earlier N1 component. The adults showed a significant effect of noise versus quiet in ITPC of the alpha and theta frequency ranges following stimulus onsets at temporal channel regions,

which may suggest a slight right hemisphere dominance for processing in noise. Children show a similar pattern; however, the ITPC for children are not significant at the group level in the left hemisphere, which suggests that phase clustering is developing from right to left. This suggests that children are still developing consistent processing mechanisms and that they may have more temporal jitter in the phase onset of alpha and theta oscillations. These results highlight the importance limiting noise for speech perception and processing in typically developing children to improve auditory speech perception, and likely have implications for children with language learning difficulties depending on how there auditory system develops.

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REVIEW OF THE LITERATURE

INTRODUCTION

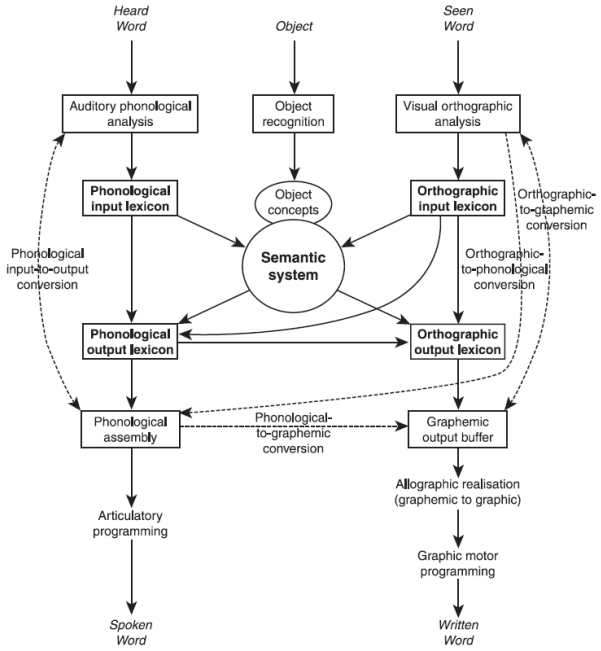
Language processing is a complicated process that is not fully understood. Models are continually being developed to help describe how the brain processes incoming linguistic information, and how the brain generates linguistic output. No model of language is completely accurate as seen by the fact that there are no complete computer models of language, and there appears to be a trade-off between simplicity and accuracy. Many models focus on single word processing, while others try to model syntax and phrase level processing adding more complexity to the underlying model. All domains of language develop simultaneously with some domains becoming more established early on in development.

The first hurdle faced by a child in learning language is learning what sounds are important for language and what sounds are environmental noises. This process involves taking a complicated speech stream and learning the different sounds of the language. Children 3 months of age appear to categorize phonemes from all the different languages as distinct speech sounds. This ability to distinguish all the different phonemes is then refined so that the child categorizes phonemes based on his/her native language (see Cheour et al. 1998; Ruvera-Gaxiola, Klarman, Garcia-Sierra, & Kuhl 2005). As the sound system becomes established, the child begins to learn to separate the word boundaries in the speech signal that is necessary to begin developing semantic representations of words.

During development, the processing abilities of the linguistic system grow and expand as the child learns abstract concepts and more complicated grammatical structures. Early in development children learn concrete words that they do not typically combine with other words. As the child approaches 18 months of age the child begins making two word phrases, and then

moves to three and four word phrase. It seems that a grammatical system is added onto a single word processing system very early in development.

There are a myriad of areas that could be impaired and could lead to language disorders during development based on current developmental models of speech processing. If one area in a model of speech perception and production delevops atypically it could lead to potential language learning issues. A simple model of single word processing was presented by Whitworth, Webster and Howard (2005), which they adapted from Patterson and Shewell (1987) shows many potential areas for problems in populations with language disorders.



From a developmental perspective of this model the child when learning spoken language must first begin to take the "heard words" and develop the auditory phonological analysis system, before developing a phonological input lexicon and so on. The development of these different systems would follow the path of the arrows in this system. A system in this model would only need to begin to develop before the next system could begin to develop.

The question in an individual with language impairment is "Where is the problem occurring in this model?", and if the impairment is developmental then question becomes "Where is the developmental difficulty arising in this model?".

SPECIFIC LANGUAGE IMPAIRMENT (SLI)

It appears that children learn language with incidental exposure and for the most part without direct instruction. Most children are able to overcome the noise in speech signals and extract the meaningful information necessary to learn language. These children then begin to use their linguistic knowledge to continue to grow and expand their linguistic abilities. Barring neurological problems, hearing loss, structural problems, and other frank disorders the vast majority of children will learn language with little difficulty. There are, however, a group of children who do not easily learn language despite having typical language exposure, and have no known causal factors for their difficulties. These children do not have hearing loss, a known neurological disorder, or a structural disorder, and have a non-verbal IQ in the typical range. Yet these children have significant defects compared to their peers in expressive language, or both receptive and expressive language. This disorder has been termed Specific Language Impairment (SLI), since it seems that only language development is affected. Specific Language Impairment is also a developmental disorder and is not simply a language development delay (Leonard, 1998).

Research over the past half century has focused on describing the deficits seen in children with SLI and developing hypotheses that may explain why these children struggle with language as well as developing treatment methods. In terms of the model presented above, these children with SLI could have problems in the development of one or more domains described in

the model. Adding more complexity to developing a hypothesis of SLI is the fact that the developing brain is very plastic and able to adapt to the challenges it faces. This new model incorporates a hypothesis that makes it possible that a domain like auditory phonological analysis may be impaired initially, and then be remediated, but after already leading to deviations in the development of other domains further down the processing chain. Understanding the cause of SLI would have broad implications for treatment of SLI and other language disorders, as well as, for theories of language. Unfortunately, there is still a large amount of mystery regarding the cause of SLI.

AUDITORY PROCESSING DISORDERS (APD)

Children with APD like children with SLI have normal hearing thresholds. Children with APD have difficulty understanding oral language, following directions, understanding speech in noise, and discriminating rapidly occurring stimuli (Jerger & Musiek, 2000); all of which children with SLI also have difficulty (McArthur & Bishop, 2001; Ziegler, Pech-Georgel, George, & Lorenzi, 2011). One of the common tests in studying and diagnosing APD is a dichotic listening task (Jerger & Musiek, 2000) and non-word repetition tasks (Moore, Ferguson, Edmondson-Jones, Ratib, & Riley, 2010). Dichotic listening and non-word repetition are both similar to working memory tasks on which children with SLI perform poorly (Archibald & Gathercole, 2007; Gathercole & Baddeley, 1990; Graf Estes, Evans, & Else-Quest, 2007). Moore et al. (2010) examined auditory processing in 1469 school age children in schools in Great Britain, and found a large amount of variability in attention, memory and auditory processing scores in children. The authors did not reach a conclusion about how to best diagnose APD. The authors commented that a consensus on the definition of APD still needs to be reached, and

better measures with higher test-retest reliability need to be developed to diagnose the disorder. It is still an open question if SLI and APD are truly separate disorders, or if they fall on a continuum with the same neurological basis.

COMORBIDITY OF IMPAIRMENTS

Making matters more complicated is the fact that SLI is often comorbid with auditory processing disorders (APD) and specific reading disorder (SRD):(also referred to as dyslexia). Sharma, Purdy and Kelly (2009) examined comorbidity of language impairments, APD, and SRD in children with suspected auditory processing disorders. The authors found that of the 52 children who had a language impairment; five children (~10%) had only a language impairment, seven children (~13%) had a comorbid auditory processing disorder, eight children (~15%) had comorbid SRD, and 32 children (~62%) had both SRD and APD based on current diagnostic criteria. Similarly, McArthur, Hogben, Edwards, Heath, and Mengler (2000) found that 51% of children with SLI had comorbid SRD, and 55% of children with SRD had SLI. These findings of comorbidity of SLI, SRD and APD present some challenges for research, and have some possible implications for hypotheses of SLI. The first challenge that is presented is how to determine the clinical population used in studies of SLI. If only children without comorbid APD and SRD are used in a study then the population is very limited (only ~10% of children with SLI). In other words, since only 7% of children have SLI (Tomblin et al., 1997) about 0.7% of children would have SLI without comorbid SRD or APD assuming the results of Sharma et al. (2009) extend to the general population. It may also be that there is the same underlying neurological bases for a couple or all three of these disorders and that different children have different behavioral manifestations depending on the individual plasticity of a child's brain and

the environment that the child is exposed to from birth. Changes in experimental methods, particularly neuroimaging, may aid in separating potential differences in pathophysiology, and additionally if researchers begin to provide fuller descriptions of clinical groups with individual participant data it may be easier to parse out different pathophysiologies based on behavioral data and experimental results across studies. While these ideas sound good on paper, they increase the cost of doing research in practice.

PATHOPHYSIOLOGY

There is not a cohesive and agreed upon understanding of the pathophysiology (potentially pathophysiologies) of SLI. The underlying and biological mechanism behind the disorder is difficult to determine. One barrier that stands in the way of the discovery of the pathophysiology of SLI is that there is, as of yet, no animal model of the disorder. This is not surprising as there is no animal model of language, and, therefore no model of language disorders in animals. If, however, SLI is caused by a disorder in a system for which an animal model can be created then it may be possible to construct a low level model of the disorder based on that component system. If auditory processing, working memory, speed of processing or attention is the underlying cause of the disorder then it may be possible to create an animal model of the disorder. The issue in research is how one separates an attention deficit from a speed of processing deficit from a working memory deficits, etc. For example, one common task is stimulus identification (i.e. can the child identify what stimuli s/he heard). These types of tasks rely not only on auditory processing, but also attention, speed of processing, and working memory. Tasks given to children have a hard time separating these components (memory, speed of processing, language, and auditory perception). The aforementioned component areas have all

been proposed as possible causes of SLI. Additionally, linguistic knowledge has also been proposed as the cause of the disorder. Proponents of different hypotheses each rely on different types of deficits seen in the disorder to provide evidence for their view of the disorder. This paper will review current neuroimaging research and then review three of the more common hypotheses of SLI: auditory processing hypotheses including both temporal processing hypotheses and immature auditory development hypotheses, working memory or phonological memory hypotheses, and attention based hypotheses.

NEUROIMAGING STUDIES

The hope of neuroimaging research is to provide a deeper understanding of the etiology of the disorder and to find biomarkers of the disorder. A variety of techniques have been used to examine potential causes of the disorder including structural imaging using magnetic resonance imaging (MRI) and diffusion tensor imaging (DTI), a technique that uses a special type of MRI scan, and functional imaging using fMRI and EEG. Each technique has its own set of advantages and disadvantages. DTI and MRI can only look at the brain structure, with DTI examining white matter tracts, and MRI examining primarily grey matter. fMRI and EEG examine the engagement of different brain regions. fMRI has better spatial resolution than EEG, but worse temporal resolution and a much higher cost. The number of studies using neuroimaging has been limited in this population, but can be divided based on the imaging technique used and if the study examines structural or functional differences.

Magnetic resonance imaging (MRI)

MRI is a neuroimaging method that can visualize the brain structure of participants to look for anatomical anomalies in clinical populations. MRI studies in children with SLI have found inconsistent results. Plante, Swisher, Vance, and Rapcsak (1991) conducted an MRI study comparing the brain structures of boys with SLI and boys with typical development. The boys in the study ranged in age from 4;2 to 9;6. The authors reported typical perisylvian fissure measures. In the children with SLI they found the right perisylvian fissure was larger than or equal to the length of the left perisylvian fissure. Volume measures also showed that the children with SLI had larger right perisylvian fissures than controls, however, Plante et al. (1991) did not find any difference between the groups in left perisylvian volume.

Gauger, Lombardino, and Leonard (1997) examined the brain morphology of the pars triangularis and the planume temporalis in children with SLI and typically developing children. The authors found that children with SLI had "narrower right hemispheres"(p. 1276), a smaller volume of the left pars triangularis, and as a group were less likely to have the leftward asymmetry of the pars triangularis, planume temporale compared to control children. These results are in line with Plante et al. (1991), and may suggest that children with SLI have atypical development of language areas in the brain.

Trauner, Wulfeck, Tallal, and Hesselink (2000) found that a large percentage (34%) of children with language impairments had atypical MRI profiles in cortical and/or subcortical white matter. The authors concluded that this might suggest a common developmental mechanism of delayed or different white matter tract development resulting in inefficient processing of information. Additional studies have shown that many children with SLI have a high incidence of polymicrogyria in the samples studied (de Vasconcelos Hage et al., 2006; Guerreiro et al., 2002) which may also suggest atypical white matter tract development in many

children with SLI. Soriano-Mas et al. (2009) conducted voxel-wise comparisons of gray and white matter in children with SLI and typically developing children. They found that children with SLI had increased gray matter volumes in the right perisylvian fissure (BA 22), and the occipital petalia (BA 18). When age was considered in the analysis the younger children with SLI had increased gray matter volumes in the entorhinal areas bilaterally, the temporopolar cortex, the caudate nuclei, the motor precentral cortex, and the left precuneus. The authors also found increased white matter volumes in the younger children with SLI in the middle temporal gyri and the right medial frontal cortex. These increased volumes in the younger children with SLI were not seen in the older children with SLI suggesting that the volumes may normalize as children age.

Additionally, there has been a DTI study on children with SLI. Kim et al. (2006) found that children with SLI ages 30-70 months had less fractional anisotropy in the region of the genu of the corpus callosum which may suggest that children with SLI have deviant white matter tract development in the corpus callosum. The authors suggest that there is delayed development of white matter tracts in children with SLI.

The aforementioned MRI and DTI studies all seem to suggest abnormalities in the white and/or gray matter development of children with SLI compared to typical children in areas that are known to be important for language. There is, however, a paucity of MRI studies. Studies have used small sample sizes and been able to find gross differences in structure, but only one study has had enough statistical power to conduct a whole brain analysis (Soriano-Mas et al., 2009). The MRI results need to be replicated on a larger scale to determine if there are different neurological development patterns in subgroups of children with SLI. All the studies mentioned

did find children with SLI who did not show abnormal MRI profiles compared to peers, which may suggest that some children with SLI have normal neurological development.

Functional MRI (fMRI)

There has been little research studying children with SLI using fMRI. One study looked only at a family with a strong history of language impairments (Hugdahl et al., 2004), which may have results that do not extend to children with SLI in general. Additional studies using fMRI tend to focus on "non-linguistic" aspects of the disorder including processing capacity, memory, and task switching.

Weismer, Plante, Jones, and Tomblin (2005) investigated verbal working memory in children with SLI in order to test if non-linguistic regions of the brain contribute to poorer performance. The authors used an encoding and recognition paradigm for their verbal working memory task. They found that the adolescents with SLI were less accurate, but did not have significantly slower reaction times than controls. The fMRI results indicate that the two groups were activating the same regions during the task; however, the degree of the response between the groups did differ. Also, there was not a slower response observed in the fMRI data. Regional differences in the HRF response were seen in precentral sulcus region, and the insular portion of the IFG. The SLI group showed less correlation between the STG, frontal, and parietal regions of interest. The correlational analysis may not have much meaning without statistical comparisons, and further validation samples.

Dibbets, Bakker, and Jolles (2006) examined task-switching in children with SLI compared to typical children. In this study, children with SLI did not show a larger performance cost of a switch versus non-switch condition. The authors also did not find different patterns of

activation in children with SLI compared to typical peers. The use of only six children with SLI and seven control children likely resulted in an underpowered experiment meaning that there may have been significant differences in the imaging data that were not detected due to the low power.

These fMRI studies suggest that there may be a difference in the way that children with SLI engage brain regions during working memory tasks, but neither study showed a difference in the regions that were activated. fMRI studies that explicitly examine SLI have been rare, and have had a limited number of subjects for comparisons. With more statistical power and more studies it may be possible to examine theories of SLI, and search for potential brain regions responsible for the poorer language skills seen in these children.

ELECTROENCEPHALOGRAPHY (EEG)

EEG has been used relatively more extensively in children with SLI compared to other neuroimaging techniques. EEG experiments in children with SLI have relied on well-established ERP components, for example the MMN (Ahmmed, Clarke, & Adams, 2008; Benasich et al., 2006; Bishop, Hardiman, & Barry, 2010, 2011; Davids et al., 2011; Friedrich, Weber, & Friederici, 2004; Korpilahti, 1995; Rinker et al., 2007; M Sharma et al., 2006), the P1-N1-P2 (Bishop & McArthur, 2004; Gilley, Sharma, Dorman, & Martin, 2006; McArthur, Atkinson, & Ellis, 2009, 2010; McArthur & Bishop, 2004b; Ors et al., 2002), the P3 (Evans, Selinger, & Pollak, 2011; Shaheen, Shohdy, Abd Al Raouf, Mohamed El Abd, & Abd Elhamid, 2011; Weber-Fox, Leonard, Wray, & Tomblin, 2010), the N400 (Ceponiene, Cummings, Wulfeck, Ballantyne, & Townsend, 2009), and the P600 (Fonteneau & van der Lely, 2008).

The MMN response

The MMN response is a heavily used response with children with SLI. The majority of EEG research in the area of SLI has relied on the MMN and related responses. The MMN is an early auditory response to a change in presented auditory information. The classical MMN is elicited when a deviant stimulus occurs within a train of frequently occurring stimuli. The deviant stimulus typically differs on some auditory dimension. The amount of difference needed to elicit the MMN is approximately the amount of difference needed for the individual to be able to discriminate the stimuli on behavioral measures (Näätänen, Paavilainen, Rinne, & Alho, 2007). The MMN is typically evaluated by subtracting a participant's average waveform for deviant stimuli from his/her average waveform for the standard stimuli, and is a negative going peak between 100-225 ms (the typically assumed MMN window) (Cowan, Winkler, Teder, & Näätänen, 1993).

The neurological source of the MMN and the cognitive system responsible for the MMN is important for interpreting differences between groups in the MMN. There is a strong relationship with memory systems and the generation of the MMN. Sensory memory and memory comparison processes have been proposed as primary systems responsible for the generation of the MMN. Evidence for the importance of sensory memory in the generation of the MMN has been seen in a variety of studies.

The MMN occurs during the same time scale of the N1 and P2 response (Alho, Woods, Algazi, Knight, & Näätänen, 1994), which may mean that the MMN is generated simply by a difference in the activation of the auditory pathway. However, the topography of the MMN and the existence of frontal source components (Alho et al., 1994), as well as animal studies (Javitt, Steinschneider, Schroeder, & Arezzo, 1996; Pincze, Lakatos, Rajkai, Ulbert, & Karmos, 2001,

2002) suggest that the MMN is separated from the classic auditory components. It has been shown that if there is a long time period (more than a few seconds) between the stimuli then an MMN will not be generated suggesting the MMN is the result of a sensory memory decay (Mäntysalo & Näätänen, 1987). Additionally the frequently occurring stimuli must be well established in memory, requiring a few repetitions, before a deviant stimuli can elicit an MMN (Näätänen, 1990). It is possible that attention could be generating the MMN (see Näätänen et al., 2007; Näätänen, 1990), however, it has been shown that overt attention to the task is not necessary for the generation of the MMN (Näätänen, Paavilainen, Tiitinen, Jiang, & Alho, 1993). Näätänen et al. (1993) showed that the MMN was still present when participants were both asked to respond to a deviant stimuli (attended condition), and when they were told to ignore the auditory stimuli (unattended condition). The authors did not find any difference in MMN amplitude or latency in the attend condition compared to the unattended condition when the deviant stimuli differed in the frequency of the standard stimuli. They did, however find a decrease in MMN amplitude when the deviant differed in intensity from the standard stimuli.

Näätänen (1991) proposed that there are two components that contribute to the generation of the MMN: those that are "informational (computational) and activational or energetical" (p. 482). This model suggests that there are components that are related to the memory formation and comparison, which generates the MMN and components that are modulatory and enhance the amplitude of the MMN. In a review of the MMN, Näätänen et al. (2007) suggested that the frontal sources of the MMN may be related to passive attention shifts to the deviant stimuli. Based on the available research, it is evident that differences in the MMN amplitude may be related either to the informational and activational components, energetical components, or both systems. The MMN differences may reveal differences in preconscious

discrimination, but it is still not clear what subcomponent of the system is leading to group differences.

The MMN provides a measure of auditory perception in children with SLI, and serves as a non-behavioral measure of auditory perception. The MMN has been used to try to determine the sources of differences seen in behavioral experiments for auditory theories of SLI. As mentioned earlier, the component system that is responsible for the MMN could be memory, attention or auditory processing, which is important to consider when making conclusions about the MMN and SLI. The MMN has been used by an increasing number of labs in recent years to examine auditory processing skills in SLI. Korpilahti (1995) found that children with SLI ages seven to thirteen had decreased MMN amplitudes in response to tones (500 Hz and 530 Hz). The author chose these tones because in prior research these tones have been observed to elicit an MMN in all normal adolescents tested. The author did not find that the MMN amplitude predicted poor performance on auditory discrimination tests, which may suggest that the differences in neural activation are not leading to significant behavioral differences. However, the results also may indicate that the MMN is more sensitive to group differences. Rinker et al. (2007) examined the MMN responses in children with SLI ages seven to eleven compared to normal IQ-matched children in response to tones (700 Hz and 750 Hz). The authors found no group differences in the obligatory P1 and N250 responses between the groups. They were able to observe the early and late MMN components that the authors term the MMN1 and MMN2 in control children and were not able to observe them in the children with SLI. They did not find significant group differences for the MMN1 or MMN2 amplitudes at frontal sites. They did find group differences in amplitude for posterior electrode sites. This is in contrast to Korpilahti (1995) who found group differences at the F5 electrode. In contrast, Uwer, Albrecht, and Von

Suchodoletz (2002) did not find group difference between children with SLI and control subjects on the MMN responses with more salient tones (1000 Hz and 1200 Hz). The experiment used easy to detect differences and results failed to find a significant MMN. The authors did however show that there was a significant difference between children with SLI and controls in response to phoneme contrast of /da/ and /ga/ and /da/ and /ba/ for the late MMN in both expressive, and expressive/receptive SLI. The MMN results in this study were not significantly correlated with behavioral discrimination scores, which again may suggest that the processing aspect of the task may be more sensitive to group differences than the behavioral measures. Ahmmed et al. (2008) examined MMN responses for relative frequency changes to determine the amount of frequency difference required to generate an MMN in children with SLI and controls. They found that the MMN was significant in both children with SLI and the comparison group for a 5% and 10% tone contrast, but not a 2% contrast. The comparison group also had significantly larger MMN amplitudes than the children with SLI. These studies tend to show decreased amplitude of the MMN response in children with SLI, which may indicate less engagement of one or more brain regions responsible for the generation of the MMN.

It is interesting that differences are seen in the MMN response for children with SLI when using tone stimuli, and speech stimuli. Davids et al. (2011) examined the differences in MMN response for speech stimuli and similarly complex nonlinguistic stimuli in children with SLI and controls. The authors attempted to use spectrum-rotated speech to create nonlinguistic stimuli. They found that children with SLI as a group did not generate an MMN response to both the linguistic and non-linguistic stimuli, while the control group generated an MMN to both types of stimuli.

The classical MMN research seems to suggest that children with SLI have reduced amplitudes for the MMN responses if the difference between the standard and deviant stimuli is smaller, but not too small. The aforementioned differences are for similar tones and similar speech sounds, when the detectability of the stimuli differences is easier there appears to be no group differences. It is still not clear why children with SLI have reduced amplitudes in the MMN response. In an attempt to address this issue, Bishop et al. (2010) examined the frequency contributions to the MMN in children with SLI and controls. They found differences only in the late MMN component between children with SLI and controls. A time frequency analysis of single trial data revealed that the children with SLI did not have the same reduction of power in the theta band that was seen in the control group. The results of this study suggest a deviance in the MMN response and not a maturational lag as has been proposed. With all of this research in the MMN there is still little consensus on what information it provides. It may or may not provide evidence for an auditory hypothesis of the disorder. It is difficult to make strong conclusions about why children with SLI have a reduced MMN because the current theory of the MMN hypothesizes that it is generated by a variety of subsystems and there is little to no research on the actual source of the differences seen in children with SLI.. It is clear that there are some neurological differences in the processing of sounds between children with SLI and typical peers, but more focus needs to be paid to the cause of those differences.

Early Components

The traditional P1 and N1 components are thought to reflect the earliest stages of cortical auditory processing. The P1 component is typically defined as the largest positive going peak in a time window that is determined by the experimental conditions and participant characteristics.

In development the P1 response decreases in latency as children approach adulthood (Ponton, Eggermont, Kwong, & Don, 2000). The P1 is thought to be generated from a reciprocal loop from the auditory cortex and the auditory thalamus (C Liégeois-Chauvel, Musolino, Badier, Marquis, & Chauvel, 1994), and it is thought that the decrease in latency as children age is a result of increased myelination of the auditory pathway (Ponton et al., 2000). In contrast the N1 follows the P1 response and is defined as the largest negative going peak in a given time interval determined by the experimental condition and participant characteristics. The N1 develops as children age first appearing consistently in older children age 7-8 when there are long ISIs between auditory stimuli, and being consistently observable in children age 11-12 (Gilley, Sharma, Dorman, & Martin, 2005). The N1 also decreases in latency as children age and the maturation of the response is thought to reflect changes in myelination, synaptic refinement, and/or cortical fiber density. This component is thought to arise primarily from the auditory cortex, with possible contributions from frontal regions and the thalamus (Ponton et al., 2000).

In contrast the P3 is thought to be primarily related to attentional and memory components of processing (Polich, 2007). Auditory aspects of the stimuli presented can affect the P3 response, including factors such as noise level, and ease of discrimination. The P3 is elicited by an oddball paradigm in response to rare stimuli. It is typically defined to be the largest positive going peak in a given time window that is dependent on subject characteristics and the experimental paradigm. In tone discrimination tasks the P3 occurs at about 300 ms on average in a typical adult. The participant must be able to detect the change of the stimuli and like the MMN is thought to be primarily related to attention and short term memory (Polich, 2007). The sources that appear to contribute to the generation of the P3 are the lateral frontal cortex, and the posterior parietal cortex including the angular gyrus (Linden, 2005). Polich (2007) presents an

inhibition hypothesis for the P3 response. In this hypothesis it is thought that the P3 (specifically the P3a) is first generated by frontal attentional circuits showing a recognition of stimulus change. The frontal regions then send information to parietal areas for memory comparisons. The brain is then thought to inhibit other areas and focus on the new incoming information, which is what generates the P3 response. The response is a culmination of both attention and memory systems engaging in the processing of novel stimuli.

The early components have been studied to examine the auditory system development of children with SLI compared to peers. Differences in the latency of the P1 and N1 components may reflect a maturational lag in children with SLI. Research on these components in children with SLI has led to conflicting results, which may be a result of the heterogeneous nature of the disorder or the testing environment. Some studies have examined auditory processing in the context of background noise (e.g. 50 dB SPL) while others have looked at processing in quiet conditions. Listening in background noise requires attentional filtering in order to detect the meaningful sounds. It may be that some of the differences seen between studies are due to the background noise and other properties of the experimental paradigm.

Significant effects have been seen in early components of the EEG for children with SLI for both tone and speech stimuli. Ors et al. (2002) examined the N1, P2 and P3 response in children with SLI ages 10;5-14;4 using both tone (3000 Hz and 1000 Hz) and speech stimuli ("puss" and "buss" i.e. the Swedish words for kiss and bus). The authors found significant difference in the N1 amplitudes between the two groups with children with SLI having significantly larger N1 amplitudes compared to peers for tones only. The authors did not find significant latency differences of the N1 component, and they did not see any significant difference of the P2 component for both tones and speech stimuli. Tonnquist-Uhlén (1996)

examined the T complex, which is an early detection component at the temporal electrode sites, in children they classified as having severe SLI compared to age matched peers and found a significant delay in latency of the T complex at the T3 and T4 electrode sites. The author did not find any difference in the amplitudes between groups. This may suggests slower processing of auditory information in children with severe SLI.

McArthur and Bishop (2004b) assessed whether differences in the early auditory component in children were due to difference subgroups of children with the disorder. The authors split their sample of children with SLI into those with good and poor frequency discrimination scores and then examined the ERP response of both clinical groups compared to typical peers. The authors hypothesized that only children with poorer auditory discrimination scores would show differences in classical ERP measures. The authors found atypical ERPs for both groups of children with SLI, suggesting that there are auditory processing differences in children who scored in the typical range on behavioral assessments of auditory processing. The authors found that the ERPs for children with SLI were similar to the typical ERPs of younger age groups of children, which they suggest may reflect a delay in auditory pathway maturation. The authors replicated these results in an additional study (Bishop & McArthur, 2004) again finding an ERP pattern in children with SLI consistent with the ERP pattern seen in younger children.

It may be that other cognitive systems are contributing to the abnormal auditory ERPs seen in children with SLI. Children with SLI who have abnormal N1-P2 responses also have poorer non-word reading scores, which may reflect poorer phonological processing and memory and not simply passive auditory processing differences (McArthur et al., 2009). It has also been shown that with auditory discrimination training that is successful in improving behavioral

scores in children with SLI affect but does not normalize the N1-P2 components (McArthur et al., 2010). This training aimed at improving the tone discrimination ability of children with SLI and SRD, and was successful to that end. The training, however, did not normalize the ERP responses of children who improved behaviorally. The early stages of processing appear to not be the contributing factor to the improved behavioral performance.

The early components of the auditory potential have been used primarily to assess auditory theories of SLI or to establish norms for children with SLI. The P3 components have been looked at more in a context of a generalized slowing or processing capacity theory of SLI. P3 component results have also differed between studies. Ors et al. (2002) examined the P3 response and found longer P3 latency in children with SLI for both tones and speech stimuli, and significantly lower P3 amplitudes for speech stimuli only. In contrast, Evans et al. (2011) found no latency differences in children with SLI for visual and auditory P3 responses using speech for the auditory condition and face stimuli for the visual condition within the context of a 1-back and 2-back task. Evans et al. (2011) did find that children with SLI had reduced amplitudes for both the auditory and visual P3 conditions as a main effect. No significant interaction was reported that if present would have suggested that the children with SLI had an amplitude reduction for the harder condition as compared to the control group. Shaheen et al. (2011) examined the auditory P3 in the context of an oddball paradigm and found that children with SLI had reduced P3 amplitudes and slower P3 latencies compared to the control group. Increased P3 latency is correlated with ease of processing and classification speed (see Azizian, Freitas, Watson, & Squires, 2006; Comerchero & Polich, 1999; Polich, 2007) and the slower P3 latency in children with SLI appears to be in line with slower reaction times seen in children with SLI (Kail, 1994; Windsor & Hwang, 1999). The slower P3 latency may be due to differences in attentional or

memory processing and not a generalized slowing of processing. The decreased P3 amplitude may reflect difficulty in updating working memory systems for more fine-grained difference in stimuli (Ors et al., 2002) or it may reflect difficulties in both processing capacity and working memory (Evans et al., 2011).

Early visual components have also been examined in children with SLI and comorbid RD. In an oddball detection condition, children with SLI and RD showed reduced amplitude in early visual components, including the P150 and P350, and a reduced latency of the P230 response during target identification (Neville, Coffey, Holcomb, & Tallal, 1993). These same children also showed a delayed N150 response to standard visual stimuli compared to control children. Neville et al. (1993) also examined the N400 component in a reading task, and found that children with SLI and RD had increased N400 amplitudes for open class words, particularly in posterior electrode sites. Anomalous sentence endings resulted in a delayed N400 response, which may suggest slower processing of linguistic information.

In order to assess attention, Shafer and her colleagues have conducted studies of speech and non-speech sound processing in a variety of noise and no noise contexts. In meaningful story and non-story contexts, children with SLI show a reverse asymmetry left and right temporal sites when processing the word "the" (Shafer, Schwartz, Morr, Kessler, & Kurtzberg, 2000). Comparing ERP responses in active versus passive tasks (i.e. tasks requiring a response vs. task where child is watching a silent film), children with and without SLI show a negative shift in the attend condition similar to the Nd response seen in adults (Shafer, Ponton, Datta, Morr, & Schwartz, 2007). The children with SLI had a delayed latency of this negativity compared to control children. This study may suggest that children SLI have deficits in attentional filtering.

AUDITORY HYPOTHESES OF SLI

Auditory processing is the first component of processing in models of auditory speech processing. As a result, auditory processing is a reasonable place to begin searching for the pathophysiology of the disorder. It is not surprising that auditory theories of SLI were some of the first theories of the disorder. They also are some of the most controversial hypotheses of the disorder. Eisenson (1968) proposed that children with SLI have particular difficulties with temporal sequencing of auditory information, and she also suggested that there might also be a lag in cerebral maturation in children with SLI. Two of the auditory-based hypotheses of SLI are based on these propositions. The first hypothesis is that temporal sequencing is the primary cause of SLI. The second hypothesis is that children with SLI have a delayed maturation of the auditory cortex specifically.

In the 1970s Tallal and her colleagues began to assess the temporal processing of children with language impairments. They extended the work of Lowe and Campbell (1965) who found that children with language impairments had difficulty with temporal sequencing of auditory information. In a series of papers her lab found that children with language impairments had deficits in determining temporal order of tones presented with short ISIs, and completing a same different task with short ISIs (Tallal & Piercy, 1973). The authors found that as the ISI increased all children performed at or near the ceiling level. Poor performance on a temporal judgment task may not translate to poor speech perception, so Tallal and Piercy next looked at speech sound processing. They found that children with language impairments performed poorly on same different tasks, and temporal order judgment tasks using /bɑ/ and /dɑ/ stimuli (Tallal & Piercy, 1974). They also looked at the vowels /ɛ/ and /æ/, but found no significant difference between children with language impairments and controls. Based on these findings, Tallal and Piercy

(1974) proposed that it is the rapid formant transition of the consonant stimuli resulted in the poorer performance of the language impaired children on that task and not the vowel task. They suggested that children with SLI have a primary deficit in temporal processing deficit of auditory information based on these findings. Tallal and Piercy (1975) then showed that if they lengthened the formant transitions that children with language impairments performed similarly to controls. From theses series of studies the authors claimed that the linguist deficits were "…secondary to an impaired rate of processing auditory information (Tallal & Piercy, 1975, p. 73)."

Researchers then began to look at the possibility of improving rapid auditory processing in children with SLI using computer games (Merzenich et al., 1996; Tallal et al., 1996). Merzenich et al. (1996) showed that children could improve in their rapid auditory processing with training. Tallal et al. (1996) showed that when paired with language training children with SLI improved in receptive language scores and rapid auditory processing. It is not clear from these studies if improving rapid auditory processing has any effect on improving language skills in children with SLI.

In the 1990s researchers continued to look into other aspects of auditory processing. Researchers have shown that children with SLI have slower reaction times when identifying a rare tone in a series of standard tones (Neville et al., 1993), and that children with SLI have difficulty identifying a stimuli when it is presented with a backward masker, but perform relatively normal levels with forward and simultaneous maskers (Wright et al., 1997). Longer tones presented with masking noises did not show differences between children with SLI and controls. Based on these two studies it appears that children with SLI have difficulty dealing with rapidly occurring masking and with identifying rapidly presented tones.

The temporal auditory processing deficit hypothesis has been the subject of intense challenges (see Bishop, Carlyon, Deeks, & Bishop, 1999; McArthur & Bishop, 2001; Rosen, 2003). Experimental, theoretical, and replication issues have questioned this hypothesis. One issue has to do with the experimental paradigm of using same or different tasks and temporal judgment tasks. As Coady, Kluender, and Evans (2005) pointed out, the poorer performance on temporal order judgment tasks could also be the result of successive and rapidly occurring stimuli and not the rapidly changing formant transitions of the individual stimuli. Bishop et al. (1999) proposed that poor performance on these auditory perceptual tasks could be the result of the testing method, and could reflect other cognitive deficits such as poor attention, difficulty adapting to task demands and/or slower learning of the task. It has also been argued that if impaired rapid auditory processing is the cause of the disorder than it should be impaired in all children with SLI (Bishop, Carlyon, et al., 1999; Rosen, 2003), which is not the case for children with SLI (Bishop, Bishop, et al., 1999) or children with specific reading disorder (SRD) (Ramus, 2003). Bishop, Bishop, et al. (1999) also did not replicate Wright's finding of poorer detection thresholds for backward masking, which may be due to the control group characteristics. Helzner, Champlin and Gillam (1996) showed that children with SLI had the same threshold as controls for detecting tones in noise with brief gaps, but that children with SLI took longer to reach their thresholds due to having more errors, which according to the authors suggests that these children may have attentional deficits instead of temporal processing deficits. McArthur and Bishop (2001) pointed out that across studies not all participants with SLI demonstrate temporal auditory processing deficits, and proposed that perhaps a subgroup of children with SLI have temporal processing deficits while others do not. This idea is supported by Rosen (1999) who found that children with "grammatical SLI" did not show auditory processing deficits.

Researchers have also found deficits in other areas of auditory processing that do not appear to be a temporal processing issue. McArthur and Bishop (2004a, 2004b) in a pair of studies examined frequency discrimination in children with SLI and found that children with SLI as a group had poorer frequency discrimination compared to controls at initial testing (McArthur & Bishop, 2004b), and that these differences had persisted at retesting 18 months later (McArthur & Bishop, 2004a). It was only a subset of children with SLI, who had poor frequency discrimination in these studies, which the authors proposed may be due to a different pathophysiology of the disorder in that group of children. The authors also found a strong negative correlation with frequency discrimination thresholds and non-word repetition such that as thresholds increased non-word repetition scores decreased (McArthur & Bishop, 2004b). The authors suggest that this does not correspond to what is expected in a temporal auditory processing account of SLI, and proposed that immature cortical maturation may provide a better explanation of the disorder.

The hypothesis that children with SLI have a delayed auditory maturation has received new life based on auditory findings in the EEG literature as well as behavioral findings. This theory has largely come from McArthur and Bishop (Bishop & McArthur, 2004, 2005; McArthur & Bishop, 2004b). McArthur and Bishop (2004b) found that children with SLI had age inappropriate ERPs compared to age matched peers on an MMN based auditory task examining frequency discrimination in the P1-N1-P2 range. The P1-N1-P2 in this study had delayed latency and more resembled the ERPs of younger controls. This result held for all children in the study with SLI regardless if they had normal frequency discrimination scores. In a follow-up study the authors suggested that based on the time frequency analysis of the MMN that there is a deviance in the theta band waves in children with SLI that is not seen

developmentally in controls suggesting a deviance of auditory processing and not a delay (Bishop et al., 2010).

The auditory theories of SLI are supported by a large amount of EEG research. These theories correctly predict that there would be deviances in the ERP components discussed. However, the differences in the ERP components could also be due to other cognitive systems, and not auditory processing per se. It is possible that the same phenomena used as evidence from the EEG literature may also be due to memory or attentional systems differences, which may better explain why there are also differences in visual processing in some children with SLI.

PHONOLOGICAL PROCESSING AND MEMORY THEORIES

Gathercole and Baddeley (1990) proposed that the phonological loop of working memory may have a significant effect on language development, and if impaired may result in language impairments. This idea is based on a model of working memory that proposes that working memory is comprised of three components: a central executive, which determines what needs to be remembered, a phonological loop which acts as a verbal rehearsal mechanism for remembering items, and a visual-spatial sketchpad that helps to remember visual aspects of what is being remembered. Gathercole and Baddeley (1990) found that children with SLI had difficulties with both nonword repetition and serial recall of words. The authors propose that phonological working memory plays a significant role in the development of higher cognitive skills (e.g. vocabulary, language comprehension, and reading). Word learning involves the simultaneous learning of the sound sequence and semantic information of the word. Difficulties with learning the sound sequences of new words may impact a child's ability to learn new words.

Evidence for this hypothesis of SLI has come from a variety of phonological tasks, as well as, looking at children with RD compared to children with SLI.

Research has shown that phonological awareness and memory are important to developing reading skills. In children with SLI there is a high rate (about 51%) of reading disorders (McArthur et al., 2000), which may suggest that there is a similar underlying pathophysiology for both children with SLI and children with SRD. Research has confirmed that children with SLI have deficits on a variety of phonological processing tasks. Children with SLI have been shown to have poorer performance on both non-word repetition and sentence repetition tasks than both typically developing children and reading impaired children without language impairments (Kamhi, Catts, Mauer, Apel, & Gentry, 1988; Kamhi & Catts, 1986). Children with SLI have shown deficits in sound blending, invented spelling, deletion, and word repetition tasks compared to normal children (Leitão, Hogben, & Fletcher, 1997).

The primary task to examine phonological working memory in children with SLI that has been used is non-word repetition. Children with SLI perform worse at complex non-word repetition than typical controls and even compared to children with sensorineural hearing loss (Briscoe, Bishop, & Norbury, 2001). Graf Estes, Evans, and Else-Quest (2007) conducted a meta-analysis of studies examining non-word repetition in children with SLI. They found that deficits in non-word repetition were consistent across the age groups used in studies. Aspects of the non-word lists in studies appear to have a significant effect on performance. Children with SLI seem to perform worse on lists that have non-words that are less word like, and lists that are composed of longer non-words.

Archibald and Gathercole (2007) suggested that the poor performance on non-word repetition may have a more complicated relationship with short-term memory (STM). The

authors examined performance on non-word repetition and serial recall using non-words and found that children with SLI performed poorly on both tasks, but worse on the non-word repetition task. According to Archibald and Gathercole this implies that children with SLI do not simply have problems with STM, but also includes an additional factor that is related to the ability to repeat novel multiple syllable forms.

From a treatment perspective it has been shown that treating phonological awareness in SLI appears to aid in improving semantic skills (Zens, Gillon, & Moran, 2009). If phonological encoding or memory is the source of their semantic difficulties, it makes sense that improving their phonological skills might improve semantic skills in children with SLI.

ATTENTION BASED HYPOTHESES

Attentional hypotheses of the disorder have received little direct study as a potential source of the language difficulties experienced by children with SLI. Modern theories of attention may provide new ways to assess attention in children with language learning impairments. Shafer et al.(2007) proposed that deficits in passive attention to speech information at a young age may contribute to the language impairments in children with SLI. This idea is based on Jusczyk's (1997) theory of spoken language development, which stressed the importance of early attention to the relevant and important cues of speech in the development of language. It may be that the early automatic selective attention in children with SLI to the most meaningful components of speech is impaired, which could lead to poorer language skills. There have been a limited number of studies to examine attentional based hypotheses, however, studies that have looked at auditory processing, and auditory memory require attentional resources (even if only passive attention).

Chikkerur, Serre, Tan, and Poggio (2010) proposed a Bayesian theory of attention in the visual system. The visual system is concerned with answering two questions: "what" and "where". The system wants to identify the object and where the object is in space. Historically these questions have been identified as separate streams: a dorsal ("where") stream and a ventral ("what") stream. In order to determine the location and identification of an object, the brain uses features of the object and spatial priors (i.e. information about where items were located in the past). The system uses both feed-forward modulation and feedback modulation to refine the predictions of the visual scene. In this model if the features of the object in the visual scene are what the system predicts and expects then the model will identify the object. When the prediction does not match the object features, a feedback portion of the model will enhance the response of the system, as the expectations of the model are updated and the system attempts determine the object and location. Additionally, if a particularly important or meaningful stimulus were introduced to the system the feedback portion would enhance the processing of the stimuli to increase the efficiency of the processing.

Hochstein and Ahissar (2002) proposed a similar model of visual attention composed of feedback and feed-forward loops. In the Hochstein and Ahissar (2002) theory of visual attention lower levels act to feed processing information to higher centers which then can feedback into the system to refine the processing information. The feedback components in this model are related to the refinement of visual processing if more details are required to fully process the object. This model aims to identify how the distinct features of the object are combined into what is perceived as a whole object. In this model initial stimulus processing is automatic and feed-forward. If visual scrutiny is then initiated at a higher level of processing, then the feedback mechanism engages in order to refine the visual representation of the object.

Similarly, the auditory system attempts to determine what is being heard and where the stimulus is in space. It is possible that a combination of a feedback and feed-forward system would exist in the auditory system as well. If the auditory system detects the expected features then the system would determine the heard stimulus and origin. The lower centers of the auditory system act in a largely feed-forward manner, as in the visual system (i.e. the auditory brainstem, and cochlea are tuned to processes certain frequencies, and types of stimuli). The feedback components of the auditory system in an extension of these models would rely on the identification of important auditory features, and the importance of the incoming stimuli. If a child had an impairment in either the feed-forward mechanism or the feedback mechanism, then he/she would have difficulty using the features of incoming information to identify stimuli, and enhancing the processing of meaningful stimuli. These problems could result in inefficient encoding of incoming linguistic information, which could result in inefficient retrieval of linguistic information.

CONCLUSION

Neuroimaging applications to SLI are in the beginning stages. Studies on children with SLI using structural neuroimaging techniques seem to point to abnormal white and grey matter development in children with SLI in the left hemisphere near the auditory and language regions near the superior temporal gyrus (de Vasconcelos Hage et al., 2006; Gauger et al., 1997; Kim et al., 2006; Plante et al., 1991; Trauner et al., 2000). Functional imaging studies indicate differences in auditory processing of children with SLI, which may be related to memory, simple auditory processing, and/or attentional mechanisms (Ahmmed et al., 2008; Bishop et al., 2010; Davids et al., 2011; Korpilahti, 1995; Shafer, Morr, Datta, Kurtzberg, & Schwartz, 2005; Uwer

et al., 2002). The theories of SLI discussed also provide some possible areas of interest in regards to a neurological basis of the disorder. In an auditory theory it may simply be differences in the auditory pathway that are leading to SLI. In a memory-based theory, problems may be arising from areas related to memory encoding or memory retrieval, which may include frontal areas, language areas, and/or temporal regions. In an attention-based theory of SLI possible areas of processing differences could be in the auditory pathways, and/or top-down processing pathways that modulate the auditory pathway. If a particular region of difference in processing function can be identified in children with SLI, then it may be possible to look at gene expression and other methods to determine if there is a medical treatment for the condition based on the area/s of the brain that is initiating and causing the disorder.

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CHAPTER 2:

NEUROPHYSIOLOGICAL CORRELATES OF WORD RECOGNITION IN NOISE IN ADULTS

INTRODUCTION

During auditory processing of linguistic information listeners must overcome many challenges in order to interpret the meaning of the spoken language. Speech is rarely delivered without noise, and the auditory system must rapidly interpret the incoming speech signal. Spectral properties and temporal envelope both appear to be important for speech processing. Recent research has focused on the importance of the temporal envelope in speech processing. Giraud and Poeppel (Giraud & Poeppel, 2012) suggest that theta band frequencies in the EEG signal are reflective of temporal envelop processing.

Temporal envelope is clearly important in speech processing, however, speech perception is often made complicated by noise in the environment. In noise the brain must rely on other features of the stimuli and top-down modulation of the auditory system to interpret the incoming auditory information. Additionally, word frequency and phonological pattern frequency have been shown to have an impact on speech perception and word recall, and may impact both bottom up and top down processing of auditory information (Coady & Aslin, 2003; Coady, Mainela-Arnold, & Evans, 2013). Taken together, it is possible that the impacts of a noisy environment may be exacerbated when the speech information becomes less certain.

Initial research in our laboratory suggests that when listening to speech sounds, EEG frequency activations differ between typical children and children with language learning problems. Further differences in these EEG frequencies may provide insight into mechanisms that differentiate bottom-up auditory processes from top-down control during speech perception (Gilley, Walker, & Sharma, 2014).

By examining the EEG frequency domain, one may be able to separate functional networks by separating the observed ERP into its frequency components. Low theta oscillations

(4-8 Hz) have been shown to be important for auditory perception (Ghitza & Greenberg, 2009; Ghitza, 2011; Mesgarani & Chang, 2012) and memory consolidation and retrieval (Klimesch, 1999; Nyhus & Curran, 2010). Gamma (30-80 Hz) activity has been implicated in local area processing and in selective attention (Brosch, Budinger, & Scheich, 2002; Kaiser, Ripper, Birbaumer, & Lutzenberger, 2003).

Models of speech perception attempt to extract auditory features from the interaction of observed functional networks. A variety of neuroimaging techniques have been used to examine the oscillatory dynamics of speech perception in humans and animals. Inferior frontal gyrus (IFG) has been shown to have a wide range of response frequencies from 1-200 Hz (Flinker et al., 2015) or 1-72 Hz (Giraud & Poeppel, 2012). The left superior temporal gyrus (STG) has been shown to be particularly active with theta and gamma oscillations (Liégeois-Chauvel, Musolino, Badier, Marquis, & Chauvel, 1994; Liégeois-Chauvel, Lorenzi, Trébuchon, Régis, & Chauvel, 2004). The inferior temporal gyrus (ITG) appears to be particularly responsive with gamma activity. During speech perception these areas are interacting with visual, memory and motor regions through oscillatory networks to process and interpret the auditory information (Giraud & Poeppel, 2012). Beta frequency bands (~16 Hz) also appear to be important for sensory gating of incoming information and appear to be important for the extraction of relevant features from sensory stimuli while suppressing irrelevant information (Hong, Buchanan, Thaker, Shepard, & Summerfelt, 2008).

Speech is typically processed in noise often in the presence of other speakers. The brain must separate the signal of interest from the background noise. Decreases in prefrontal cortex white and grey matter thickness have been associated with increased difficulty for processing in speech in noise (Wong, Ettlinger, Sheppard, Gunasekera, & Dhar, 2010). Additionally, increases

in Blood Oxygenation Level Dependent BOLD response in the precuneus and superior temporal region also predict improved perception in multitalker babble (Wong, Uppunda, Parrish, & Dhar, 2008). It is not clear how multitalker babble will affect the time frequency decomposition of the EEG signal during speech perception.

In this study we examine the effects of multitalker babble on speech perception in typical adults. Multitalker babble is a realistic noise that is encountered in a variety of environments. White noise has been shown to have a larger impact than multitalker babble on speech perception in adults, but it does not provide a realistic listening environment (Tun & Wingfield, 1999). Additionally, multitalker babble noise has been shown to delay brainstem processing in both typical adults and children and children with reading impairments (Anderson, Skoe, Chandrasekaran, & Kraus, 2010).

Time frequency analysis provides a tool for examining the interaction of the auditory system with other cognitive systems (memory, attention, and language). Oscillatory frequencies in the observed EEG signal are thought to reflect functionally connected networks of brain regions or local processing within a single region. We are interested in what components are important for speech perception in quiet and noise, and what systems are interacting during speech perception.

This study aims to provide additional information on the functional aspects of oscillatory networks by examining brain sources of time frequency components via the inverse wavelet transform. We use time frequency analysis with a spectral principle component analysis to determine task relevant oscillatory frequencies and the relative timing of those frequencies. We then apply the inverse wavelet transform to the data to project back into the original channel space. Oscillatory networks are generated by cells within a local brain region and by networks of

functionally connected regions. In order to extract local networks of oscillatory sources we applied independent components analysis (ICA) to the inverse wavelet transform at the selected frequencies. We used a principal components analysis (PCA) to the time frequency domain to reduce noise that is not task relevant and to identify peak frequencies that are important for task responses.

METHODS

Fifteen adults (5 male) aged 18-29 participated in this study. All participants were monolingual English speakers and had no history of neurological impairments, typical hearing, and typical or corrected to normal vision. All participants provided informed, written consent as approved by the University of Colorado Institutional Review Board, and were compensated with a gift card equivalent to $10 per hour for their participation.

Stimuli consisted of CVC words and non-words (20 words, and 20 non-words) matched on phonotactic probability. Phonotactic probability refers to the frequency at which the component phonemes in a word occur together. Words were grouped into four word classes grouped by word frequency and by phonotactic probability: high word frequency and high phonotactic probability (HF/HP), high frequency and low phonotactic probability (HF/LP), low word frequency and high phonotactic probability (LF/HP), and low word frequency and low phonotactic probability (LF/LP). Word classes were then matched on phonotactic neighborhood density to account for effects of neighborhood density, or the number and frequency of words that share considerable phonological overlap. All stimuli were recorded from the same female speaker with repeated productions for matching quality. Sound files were edited in Audition™ to match total length of auditory signal to 350 ms by extending or decreasing the vowel length by

adding or removing cycles of the vowels. The sound files were then normalized to match the average volume of each file. Multitalker babble was also used as a variable of interest and are from Shafer et al. (Schafer & Thibodeau, 2006; Schafer et al., 2012). The noise file was normalized so that the signal to noise ratio of the stimuli to noise was 6 dB SNR.

Participants completed an instruction set where a recording of the female speaker provided them with the words and gave an auditory example sentence that used the word. Participants then participated in two short practice sets where they practiced identifying the words: one set in quiet and one in noise. The practice session had each word and non-word used in the experiment presented a single time and participants were instructed to respond every time they heard a word. Accuracy feedback for correct responses was provided on a television screen at the fixation cross. Participants then completed the four experimental sets.

The stimuli were presented in four blocks presented in random order. The blocks consisted of 250 stimuli (words and non-words) with words presented as rare stimuli at a rate of ~30% (29%<probability<31%). Stimuli lists were constructed in Matlab using random permutations with the conditions that two words did not occur in succession, and that all stimuli were used at least once. Stimuli were presented with an ISI of 1000 ms. Participants were instructed to respond using a button box every time they heard a word.

Participants were seated in a comfortable chair in a sound-attenuated room. Stimuli were presented over insert earphones with an average loudness of 65 dB SPL and the noise when presented was at 59 dB SPL. Sound levels were calibrated using a Larson-Davis System 824 Sound Level Meter.

Continuous EEG was recorded from 64 sintered Ag/Ag-Cl electrodes embedded in an elastic cap (Neuroscan QuikCaps) arranged according to the extended 10–20 system. A separate

bipolar channel was used to monitor ocular activity during the session. The EEG was referenced online to a vertex electrode and offline to the average reference. EEG was sampled at 1000 Hz, and filtered from 0.1 to 100 Hz offline. Electrode impedances were kept below 10kOhms.

Data Analysis

EEG data were segmented into 1650 ms epochs around stimuli with a 200 ms prestimulus interval. Any bad channels were deleted prior to analysis. The EEG was down-sampled to 250Hz. Ocular and single channel artifacts were removed using ICA as implemented in EEGLAB (Delorme & Makeig, 2004). Eye blinks were considered to be components that were correlated with the eye channel at 80% and had a frontal electrode distribution. Components with the maximum component weight at a single channel being 3 standard deviations above any other channel were considered single channel noise and rejected. Epochs were average referenced and baseline corrected to the prestimulus interval.

ERP waveforms at selected channel groups (PZ, T7, and T8) were assessed using a repeated measures ANOVA (1x8) with single degree of freedom tests. Channel groups were created as the average of the identifying channels and direct neighbors. The PZ channel group consisted of CPZ, POZ, P1, and P2; the T7 channel group consisted of T7, FT7, C5, and TP7; and the T8 channel group consisted of T8, FT8, C6 and TP8. Based on pilot analyses and results from this data set, we found that the auditory N1 peak over the temporal electrodes and cognitive P3 peak over the parietal electrodes revealed consistent differences across study conditions. Therefore, we limited the subsequent analyses to these peaks. N1 amplitude and P3 amplitude were assessed by taking the average amplitude in a specified time window (90-260 ms for N1,

375-900 ms of P3) based on the grand average ERP at the PZ electrode group, which contains both the N1 and P3 response.

Concatenated epochs for each participant were analyzed through a continuous wavelet transform (CWT) using a fast-Fourier convolution (see Torrence & Compo, 1998). CWTs were computed at 256 log-spaced frequencies ranging from 3Hz to 100Hz. Edge effects were avoided by removing 100 ms at the beginning and end of each epoch after the CWT and by analyzing the data at frequencies above 4 Hz. Wavelet power was computed using element-wise conjugate multiplication: $power = X.* conjugate(X)$ which is equivalent to $power = |X|.^2$, but faster computationally. Intertrial phase clustering (ITPC) was computed across conditions using: $ITPC = \left|mean\left(exp(i * k)\right)\right|$, where k is the vector of phase angles at a single time frequency point across trials (Cohen, 2014). ITPC is a scale from 0 to 1 with 0 meaning the phase angles are uniformly distributed and 1 meaning the phase angles are identical from trial to trial.

ITPC significance is strongly related to the number of trials available for analysis with the statistical power increasing dramatically after 50 trials. ITPC is Rayleigh distributed and critical values can be found using equation 2.1.

$$ITPC_{crit} = \sqrt{\frac{-ln(p)}{trials}} \qquad (2.1)$$

The average number of trials for any participant in noise or quiet was 150 following rejection. Using a p value of 0.001 the critical value for this study is 0.2146 and for a p-value of 0.01 the critical value is 0.1752.

Reaction times and accuracy were analyzed using separate repeated measures ANOVAs. Reaction times were transformed using a natural log of the reaction time in order to not violate normality assumptions of the ANOVA. Accuracy data were transformed using an arcsine

transform to not violate the normality assumption of the ANOVA. All within subject factors were tested with single degree of freedom tests using contrast codes in order to avoid violations of the homogeneity of variance assumptions of ANOVA, and to provide an easy to interpret analysis.

Source analysis

A spectral-temporal principle component analysis (PCA) of the concatenated trials and channels in the time-frequency power domain was used to select frequencies that were consistently active across channels. Each frequency of interest was then selectively filtered and used as the basis for modeling the brain source waves. These peaks were determined by taking the difference between the raw frequency spectrum and the PCA filtered spectrum averaged across all channels. Areas of the spectrum with the smallest difference are the areas that are being enhanced by the filter. A group average difference frequency spectrum was then computed using individual participants' average z-score frequency difference spectrums to find consistent frequency bins that are enhanced across participants. Frequencies of interest for each subject were considered as peak frequencies (log scale) closest to the mean peak frequency of interest of the group. Individual source waves were then computed via the inverse wavelet transform of the CWT scales within $1/6^{th}$ of an octave below the peak frequency and $1/6^{th}$ of an octave above the frequency of interest. Each set of source waves for each participant was then subjected to extended infomax ICA. Independent components that accounted for 10% of the variance or more were retained for the brain source analysis.

Brain source analysis was completed by reconstructing the current source densities (current density reconstruction, or CDR) for each source wave using standardized low-resolution

brain electromagnetic tomography (sLORETA). sLORETA solutions were constrained to a 3-shell realistic head model (scalp, skull, cortex) constructed with the boundary element method (BEM) from the averaged adult MRI (Montreal Neurological Institute) using Curry 6.1 (Compumedics-Neuroscan). Electrode positions were co-registered to the BEM based on standard 10-20 electrode positions. CDRs for each source wave were then computed using custom code in Matlab to implement the sLORETA algorithm. CDR solutions were then vector normalized and averaged across participants. Final group mean CDRs were then limited to the upper 5% of the current net (i.e., at a power threshold of 95%).

RESULTS

The repeated measure ANOVA showed that there were no significant effects of word frequency, phonotactic probability, or noise on overall response accuracy of word recognition. There is a significant interaction effect of word frequency and phonotactic pattern frequency on reaction times (FIGURE 2.1) with participants responding faster to high frequency and high probability words and low frequency and low probability words compared to high frequency and low probability words and low probability and high frequency words ([HF/HP & LF/LP] < [HF/LP & LF/HP], $p= 0.000001$, $F(1,14)= 65.5729$).

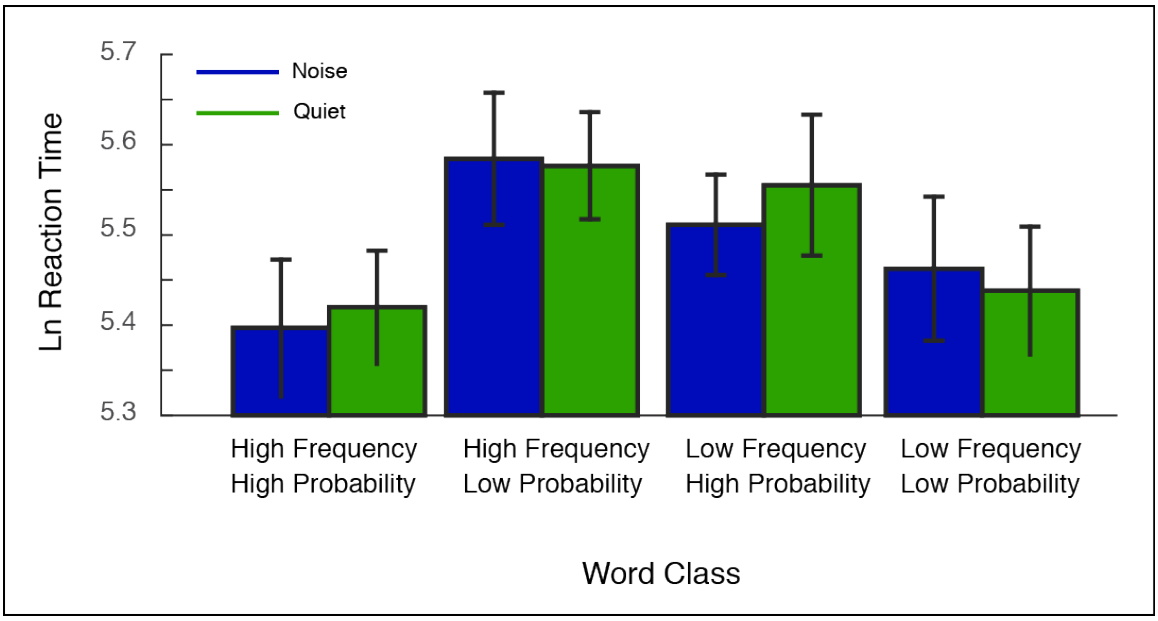

FIGURE 2.1: Average natural log reaction times: Reaction times were not significantly different in noise and in quiet. There is an interaction effect of word frequency and phonotactic probability with adults responding faster to high frequency and high probability words, and low frequency and low probability words.

There was a significant effect of noise versus quiet (FIGURE 2.2) on N1 amplitude at the PZ electrode group ($p= 0.0246$, $F(1,14)= 6.3432$) and the T8 electrode group ($p=.0006$, $F(1,14)=19.2259$). There was a significant effect of phonotactic probability on the N1 amplitude at the T7 electrode group ($p= 0.0294$, $F(1,14)= 5.8806$). The N1 is an index of early auditory speech perception, and differences may indicate differences in engagement of processing regions contributing to the N1. There were no significant effects of noise, phonotactic probability, or word frequency on the P3 response at any electrode group.

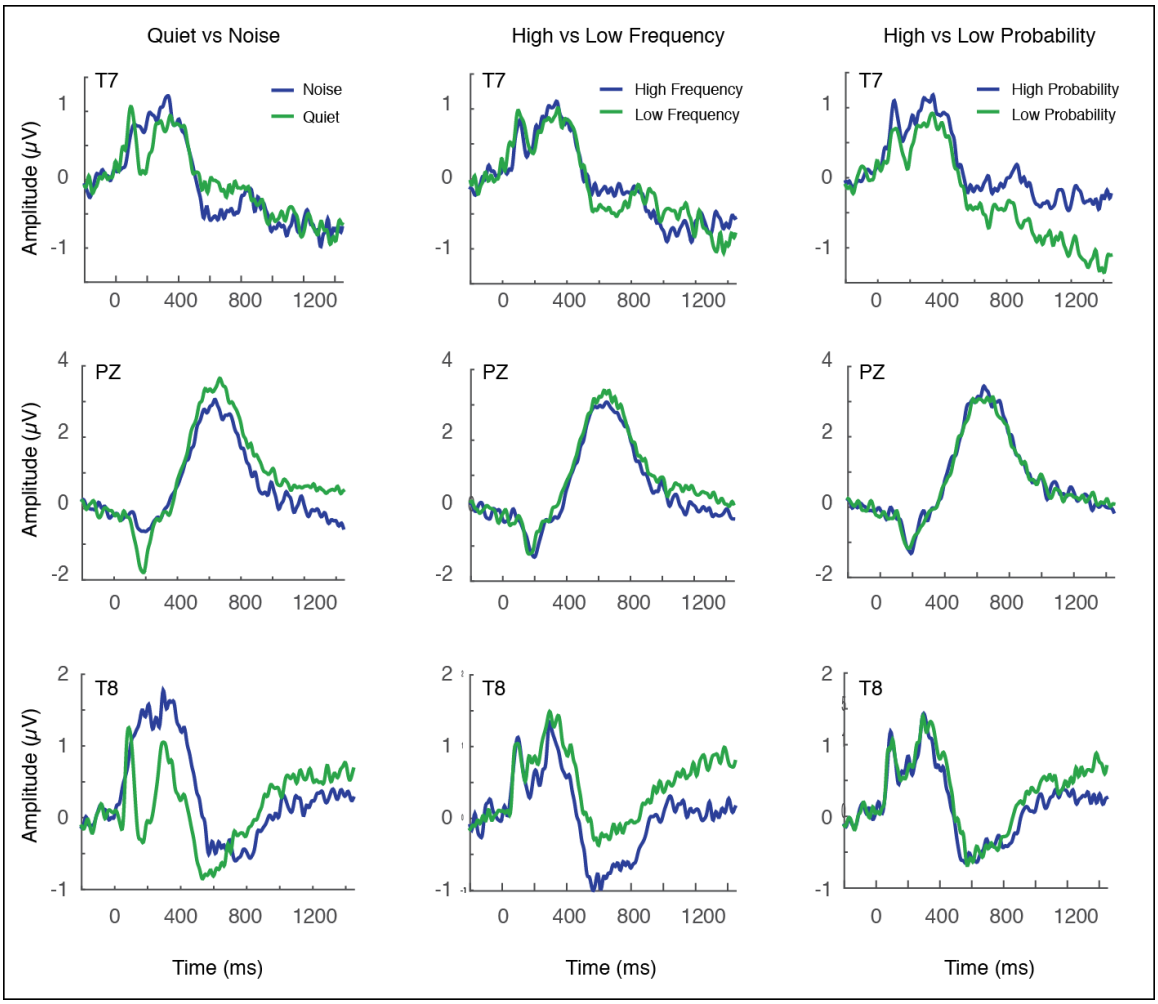

FIGURE 2.2: GROUP average ERP waveforms: Wave forms at each electrode group showing condition-level differences for noise versus quiet (left column), high versus low phonological pattern frequency (middle column), and high versus low phonotactic probability (right column). X-axes represent time in milliseconds from the onset of the stimulus. Y-axes represent ERP amplitude in microvolts. There is a significant effect of noise on the N1 response at the PZ and T8 electrode groups.

Intertrial phase clustering reveals significant effects in the T7, T8 and PZ channel groups following the stimulus onset in quiet conditions in the theta range for T7 and PZ and in the alpha and theta range for T8 (FIGURE 2.3). The phase clustering is not significant at the p=0.01 level in noise for any electrode group. The red and orange coloring are the areas of significant phase clustering and occur following stimulus onset in the task in a similar timeframe to the N1 effects observed.

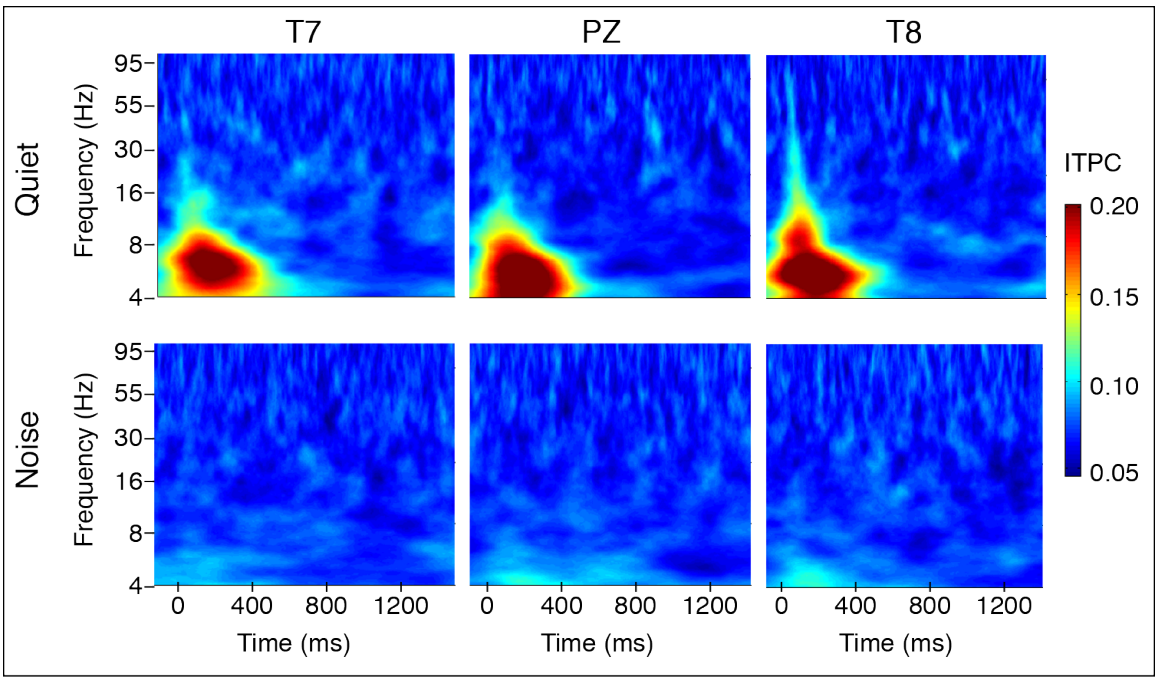

FIGURE 2.3: Intertrial phase coupling at three electrode groups: ITPC in quiet and noise at three electrode groups. For each plot, the x-axis represents time in milliseconds from the stimulus onset, the y-axis represents log-spaced frequency in Hertz, and color represents the ITPC. Significant activations are seen in the theta frequency bands after stimulus onset at all electrode groups. T8 also has Significant ITPC values in the alpha range.

Average CWT power spectra reveal strong alpha (8-12 Hz) and theta (4-8 Hz) bursts near stimulus onset and strong beta bursts (12-19 Hz) during the latter part of the epoch (FIGURE 2.4). Alpha and theta appear to be playing a role in the early processing phases of the stimuli, while beta is stronger near the reaction from the participant. These bursts correspond to the PCA enhanced frequencies. The power spectra also correspond to the sources from the source analysis.

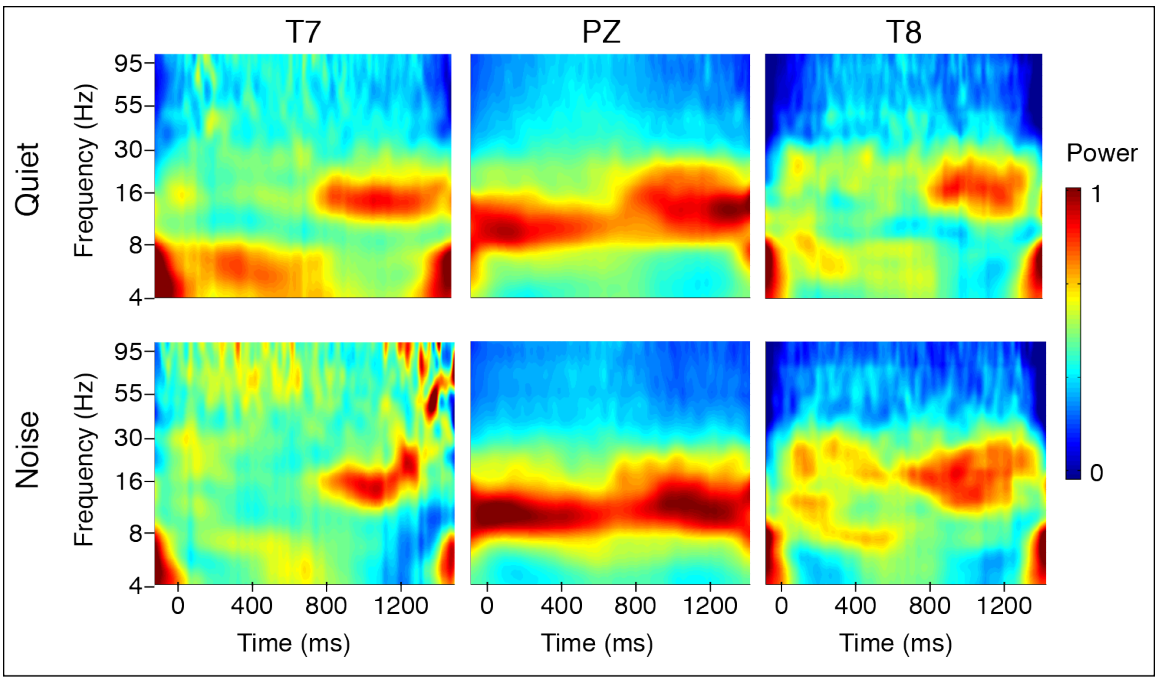

FIGURE 2.4: Average time-frequency power: Average time-frequency power at three channel groups in quiet and in noise. For each plot, the x-axis represents time in milliseconds from the stimulus onset, the y-axis represents log-spaced frequency in Hertz, and color represents the At T7 there is a slight increase in gamma activity following stimulus onset in noise, and a slight increase in theta activity in quiet. The central electrodes are dominated by alpha and beta activity in both quiet and noise. T8 is characterized by a slight increase in alpha and beta activity following stimulus onset in noise.

The source analysis also shows activation of the 5Hz frequency band in the right temporal lobe and thalamus, while 8 and 13 Hz frequency bands were generated by the precuneus and cingulate cortex. The beta frequency bands were localized to the right temporal lobe. Higher frequencies were too variable between participants and did not come out as significant (FIGURE 2.5) which may be to a lack of power or to participant variability.

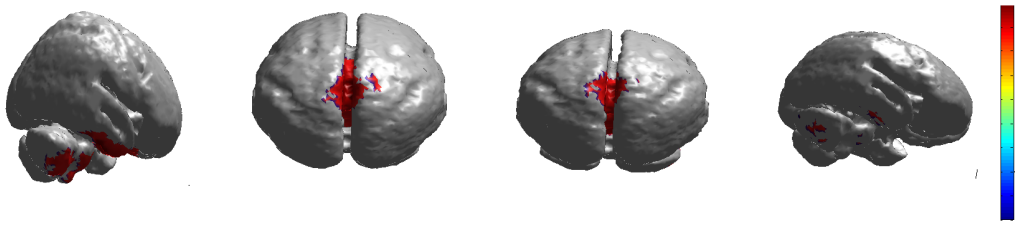

FIGURE 2.5: Mean current density reconstructions for all participants. CDR sources are overlaid on the surface of the cortical shell used in the source model. Color represents t-values from the solution, with red indicating the upper 5th percentile. The sources from left to right are 5Hz, 8Hz, 13 Hz, and 19 Hz. Low theta and beta frequencies appear in the right temporal lobe with low theta also having some thalamic activity present. The 8Hz and 13 Hz sources both are localized to the precuneus and posterior cingulate gyri.

DISCUSSION

Reaction times to the different word classes provide interesting insight into the processes involved in word identification. Adults show faster reaction times to HF/HP and LF/LP words compared to HF/LP and LF/HP words. This suggests that words that are the most common and least common in both dimensions are the fastest to be identified. One explanation for this finding is that the decision making process for identifying a word from a group of non-words involves multiple, parallel contributions to a probabilistic decision. If a word occurs frequently and has phonological properties that occur frequently (HF/HP), then the cumulative probability of identifying that word reaches a decision criterion more quickly. However, such an account does not sufficiently explain the faster detection when those same features occur infrequently (LF/LP). It is possible that the faster reaction times to LF/LP stimuli reflect a task-level strategy for word identification. In this task, subjects were informed of the words to listen for before the task began. Those words with uncommon features will tend to "stand out" from the other words; that is, they appear more salient as a target for the task. Such prior knowledge of those now-salient features may act as a primer for working memory during the task, thus speeding reaction times. In other words, during this type of task, the listener is primed for those words with uncommon features.

The N1 ERP responses revealed significant effects of noise during auditory processing. The N1 response shows effects of noise at central and right hemisphere electrode sites, while phonotactic probability had a significant effect on left hemisphere electrode sites. The N1 response timing is in line with the timing of the significant intertrial phase cluster of the theta frequency bands in quiet across all electrode groups. There was also significant phase clustering in the alpha frequency range for the right hemisphere electrode group, which may indicate more

engagement in the right hemisphere in the coordination of processing regions and/or attentional refinement of the auditory processing.

The N1 effects described in our results corroborate previous reports of the N1 being sensitive to temporal uncertainty and to background noise. For example, the N1 decreases in amplitude and increases in latency when the temporal following rate of a stimulus increases rapidly (Gilley et al., 2005, 2006). Further, the authors of that study reported that these changes to stimulus rate follow a developmental trajectory that coincides with age related changes in language processing. In adults, the N1 amplitudes appear to be sensitive to uncertainty of the temporal following rate. When comparing responses to predictable sequences of speech sounds, the N1 was larger than when listeners heard random, unpredictable sequences, and those changes were more pronounced over the right hemisphere (Cochell & Gilley, 2012).

Based on the N1 response and the increased ITPC in the right hemisphere it is possible that the right hemisphere is more influenced by noise on the speech signal. If the right hemisphere is engaged for tracking and following the temporal envelop of a stimulus, then it is possible that the noise acts to de-synchronize these tracking responses. In turn, such de-synchronization might decrease the contribution from this part of the network to the overall decision criterion for identifying a target stimulus. Similarly, the reduction of N1 amplitudes over the left hemisphere may suggest that the left hemisphere is sensitive to phonotactic probabilities, with or without competing noise. The noise condition in this study is at a loud volume and it is possible that clustering would continue to be seen in environments with fluctuating noise levels or reduced noise levels.

The source analysis also point to the right hemisphere being dominate in the perception of this task as sources; the low frequency theta and the low frequency beta sources were

localized in part to the right temporal lobes. The higher frequencies could not be localized in the group average due to participant variability; however the higher frequencies are likely still important in the task perception. The low frequencies appear to be carrying most of the power in this study.

This right hemisphere clustering and the N1 results described above might be in line with the notion that the right hemisphere is more adaptive to slow temporal features, and the left hemisphere is more responsive to fine structure (Giraud & Poeppel, 2012; Giraud et al., 2007; Jamison, Watkins, Bishop, & Matthews, 2006). Theta frequency activity has been shown to be increased in the right auditory cortex and is thought to phase lock with the speech envelop (Giraud & Poeppel, 2012). The source analysis and ITPC analysis in this study suggest that theta and alpha are more active in the right hemisphere and at a more consistent phase from trial to trial. Further, when listening in background noise, phase clustering becomes less consistent which affects both temporal envelope following and probabilistic decision processes.

Noise can have a negative impact on speech perception and it appears that noise used in this study may have a larger impact in the right hemisphere than the left hemisphere. It would be interesting to examine the effects of fluctuating noise and noise at lower volumes on the responses measured in this study to see if there is a continuum of phase clustering as noise is added to the signal or if there is a cut-off SNR at which the brain is no longer able to phase cluster.

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CHAPTER 3:

NEUROPHYSIOLOGICAL CORRELATES OF WORD RECOGNITION IN NOISE IN CHILDREN

INTRODUCTION

Understanding the underlying mechanisms of auditory speech and language processing has important implications for the diagnosis and treatment of language learning impairments in children. Identifying and treating language impairment in children is complicated by the fact that specific language impairment (SLI) is often comorbid with auditory processing disorders (APD) and specific reading disorder (SRD: also referred to as dyslexia). Sharma, Purdy and Kelly (2009) examined comorbidity of language impairments, APD, and SRD in children with suspected auditory processing disorders. The authors found that of the 52 children who had a language impairment; five children (~10%) had only a language impairment, seven children (~13%) had a comorbid auditory processing disorder, eight children (~15%) had comorbid SRD, and 32 children (~62%) had both SRD and APD based on current diagnostic criteria. Similarly, McArthur, Hogben, Edwards, Heath, and Mengler (2000) found that 51% of children with SLI had comorbid SRD, and 55% of children with SRD had SLI.

These findings of comorbidity of SLI, SRD and APD present some challenges for research, and have some possible implications for hypotheses of SLI. The first challenge is how to determine the clinical population used in studies of SLI. If children without comorbid APD and SRD are used in a study then the population is very limited (only ~10% of children with SLI). In other words, since only 7% of children have SLI (Tomblin et al., 1997) about 0.7% of children would have SLI without comorbid SRD or APD assuming the results of Sharma et al. (2009) extend to the general population. It may also be that there is the same underlying neurological bases for a couple or all three of these disorders and that different children have different behavioral manifestations depending on the individual plasticity of a child's brain and

the environment that the child is exposed to from birth. Changes in experimental methods, particularly neuroimaging, may aid in separating potential differences in pathophysiology.

Neuroimaging applications to language impairments have provided some insights into potential causes of differences. Studies on children with language impairments using structural neuroimaging techniques seem to point to abnormal white and grey matter development in children with language impairments in the left hemisphere near the auditory and language regions near the superior temporal gyrus (de Vasconcelos Hage et al., 2006; Gauger et al., 1997; Kim et al., 2006; Plante et al., 1991; Trauner et al., 2000). Functional imaging studies indicate differences in auditory processing of children with language impairments, which may be related to memory, simple auditory processing, and/or attentional mechanisms (Ahmmed et al., 2008; Bishop et al., 2010; Davids et al., 2011; Korpilahti, 1995; Shafer et al., 2005; Uwer et al., 2002).

Prior work has shown developmental differences in the cortical evoked response in typical children having delayed latency of P1 and N1 responses as they develop (Gilley et al., 2005, 2006; A. Sharma et al., 2005). Children with language impairments also show differences in CAEP responses from their typical peers. These differences in early CAEP responses are likely due to myelination and cortical development. Functional networks also continue to develop as children age including attention and memory systems. While many studies have shown differences in children with language impairments and their peers, it would be nice to understand the developing processing system and its trajectory in order to better understand differences in children with language impairments.

Difficulties in auditory perception in children with language impairments have been shown extensively(e.g. see Bishop et al., 1999; Bishop, Hardiman, & Barry, 2010, 2012; Kraus et al., 1996; Tallal & Piercy, 1974; Wible, Nicol, & Kraus, 2004); however there is little

consensus in what is causing the observed processing deficits. Time-frequency decomposition of EEG data is a promising method for identifying contributions from different oscillatory processes, and has been used to examine differences in children with language impairments, dyslexia, and auditory processing disorders. Bishop et al. (2010) examined the frequency contributions to the mismatch negativity (MMN) response in children with SLI and controls using a time-frequency analysis of single trial data. Their analysis revealed a reduction of theta power in the control group that was not observed in children with SLI, suggesting that theta frequencies (4-8 Hz) may be important for difference detection in the auditory system.

Initial research in our laboratory suggests differences in EEG frequency activations in typical children and children with language learning problems when presented with speech sounds. Specifically, we have shown de-synchronization of oscillatory EEG activity in the beta frequency range (13-30 Hz) in children with language learning problems (Gilley et al., 2014), and decreased theta (4-8 Hz) and gamma (30-50 Hz) power in children with listening difficulties when listening in background noise (Gilley, Sharma, & Purdy, in review). The effects observed in these different frequency bands may provide insight into mechanisms that differentiate bottom-up sensory processes from top-down modulatory processes that extract the signal of interest during speech perception (Gilley et al., 2014).

The development of oscillatory neural networks in the gamma frequency range (30-80 Hz) continues into adulthood and may reflect maturation of GABAergic neurotransmission and cortical network development (Uhlhaas, Roux, Rodriguez, Rotarska-Jagiela, & Singer, 2010). Resting state oscillations with higher gamma power have been positively correlated with higher cognitive and language skills in infants and children (Benasich, Gou, Choudhury, & Harris, 2008). Lower frequency activity also changes developmentally with an increase in alpha (8-12

Hz) power and a decrease in theta (4-8 Hz) and delta (2-4 Hz) power as children grow into adulthood (Klimesch, 1999).

Language learning occurs through repeated exposure to auditory and visual signals of language. Phonotactic probability and word frequency have been shown to affect word retention and processing in children with SLI differently than typical peers (Coady & Aslin, 2004; Coady, Evans, & Kluender, 2010; Coady et al., 2013). As children are exposed more and more to frequently occurring words and phonological patterns it is possible that they develop more efficient neural mechanism to process those words and sounds that are most common to their language (Jusczyk, 1997).

In order to better understand the differences between children with language learning problems and typical peers it is important to understand the developmental trajectory of processing information. In this study we examined functional networks for speech processing and word identification in typically developing, school-aged children to better understand the underlying contributions to auditory language processing during development.

METHODS

Eleven children aged (7-14) participated in this study. All participants were monolingual English speakers and had no history of neurological impairments, typical hearing, and typical or corrected to normal vision. All parents provided informed, written consent and children provided informed written assent as approved by the University of Colorado Institutional Review Board, and were compensated with a gift card equivalent to $10 per hour for their participation.

Stimuli consisted of CVC words and non-words (20 words, and 20 non-words) matched on phonotactic probability. Phonotactic probability refers to the frequency at which the

component phonemes in a word occur together. Words were grouped into four word classes grouped by word frequency and by phonotactic probability: high word frequency and high phonotactic probability (HF/HP), high frequency and low phonotactic probability (HF/LP), low word frequency and high phonotactic probability (LF/HP), and low word frequency and low phonotactic probability (LF/LP). Word classes were then matched on phonotactic neighborhood density to account for effects of neighborhood density, or the number and frequency of words that share considerable phonological overlap. All stimuli were recorded from the same female speaker with repeated productions for matching quality. Sound files were edited in Audition™ to match total length of auditory signal to 350 ms by extending or decreasing the vowel length by adding or removing cycles of the vowels. The sound files were then normalized to match the average volume of each file. Multitalker babble was also used as a variable of interest and are from Shafer et al. (Schafer & Thibodeau, 2006; Schafer et al., 2012). The noise file was normalized so that the signal to noise ratio of the stimuli to noise was 6 dB SNR.

Participants completed an instruction set where a recording of the female speaker provided them with the words and gave an auditory example sentence that used the word. Participants then participated in two short practice sets where they practiced identifying the words: one set in quiet and one in noise. The practice session had each word and non-word used in the experiment presented a single time and participants were instructed to respond every time they heard a word. Accuracy feedback for correct responses was provided on a television screen at the fixation cross. Participants then completed the four experimental sets.

The stimuli were presented in four blocks presented in random order. The blocks consisted of 250 stimuli with words presented as rare stimuli at a rate of ~30% (29%<probability<31%). Stimuli lists were constructed in Matlab using random permutations

with the conditions that two words did not occur in succession, and that all stimuli were used at least once. Stimuli were presented with an ISI of 1000 ms. Participants were instructed to respond using a button box every time they heard a word.

Participants were seated in a comfortable chair in a sound-attenuated room. Stimuli were presented over insert earphones with an average loudness of 65 dB SPL and the noise, when presented, was at 59 dB SPL. Sound levels were calibrated using a Larson-Davis System 824 Sound Level Meter.

Continuous EEG was recorded from 64 sintered Ag/Ag-Cl electrodes embedded in an elastic cap (Neuroscan QuikCaps) arranged according to the extended 10–20 system. A separate bipolar channel was used to monitor ocular activity during the session. The EEG was referenced online to a vertex electrode and offline to the average reference. EEG was sampled at 1000 Hz, and filtered from 0.1 to 100 Hz offline. Electrode impedances were kept below 10kOhms.

Data Analysis

EEG data were segmented into 1650 ms epochs around stimuli with a 200 ms prestimulus interval. Any bad channels were deleted prior to analysis. The EEG was down sampled to 250Hz. Ocular and single channel artifacts were removed using ICA as implemented in EEGLAB (Delorme & Makeig, 2004). Eyeblinks were considered to be components that were correlated with the eye channel at 80% and had a frontal electrode distribution. Components with the maximum component weight at a single channel being 3 standard deviations above any other channel were considered single channel noise and rejected. Epochs were average referenced and baseline corrected to the prestimulus interval.

ERP waveforms at selected channel groups (PZ, T7, and T8) were assessed using a repeated measures ANOVA (2x8) with single degree of freedom tests. Channel groups were created as the average of the identifying channels and direct neighbors. The PZ channel group consisted of CPZ, POZ, P1, and P2; the T7 channel group consisted of T7, FT7, C5, and TP7; and the T8 channel group consisted of T8, FT8, C6 and TP8. P3 amplitude and N1 amplitude were assess by taking the average amplitude in a specified time window based on the grand average ERP at the PZ electrode group (90-260 ms for N1, 375-900 ms of P3).

Concatenated epochs for each participant were analyzed with a continuous wavelet transform using a fast-Fourier convolution (see Torrence & Compo, 1998). CWTs were computed at 256 log-spaced frequencies ranging from 3Hz to 100Hz. Edge effects were avoided by removing 100 ms at the beginning and end of each epoch after the CWT and by analyzing the data at frequencies above 4 Hz. Wavelet power was computed using element wise conjugate multiplication: $power = X.* conjugate(X)$ which is equivalent to $wer = |X|.^2$, but faster computationally. Intertrial phase clustering (ITPC) was computed across conditions using: $ITPC = \left|mean\left(exp(i * k)\right)\right|$, where k is the vector of phase angles at a single time frequency point across trials (Cohen, 2014). ITPC is a scale from 0 to 1 with 0 meaning the phase angles are uniformly distributed and 1 meaning the phase angles are identical from trial to trial.

ITPC significance is strongly related to the number of trials available for analysis with the statistical power increasing dramatically after 50 trials. ITPC is Rayleigh distributed and critical values can be found using equation 3.1.

$$ITPC_{crit} = \sqrt{\frac{-ln(p)}{trials}} \qquad (3.1)$$

The average number of trials for any participant in noise or quiet was 150 following rejection. Using a p value of 0.001 the critical value for this study is 0.2146 and for a p-value of 0.01 the critical value is 0.1752.

Reaction times and accuracy were analyzed using separate repeated measures ANOVAs. Reaction times were transformed using a natural log of the reaction time in order to not violate normality assumptions of the ANOVA. Accuracy data were transformed using an arcsin transform to not violate the normality assumption of the ANOVA. All within subject factors were tested with single degree of freedom tests using contrast codes in order to avoid violations of the homogeneity of variance assumptions of ANOVA, and to provide an easy to interpret analysis.

Source analysis

A spectral-temporal principle component analysis (PCA) of the concatenated trials and channels in the time-frequency power domain was used to select frequencies that were consistently active across channels. Each frequency of interest was then selectively filtered and used as the basis for modeling the brain source waves. These peaks were determined by taking the difference between the raw frequency spectrum and the PCA filtered spectrum averaged across all channels. Areas of the spectrum with the smallest difference are the areas that are being enhanced by the filter. An average group difference frequency spectrum was then computed using the individual participant average z-score frequency difference spectrums to find consistent frequency bins that are enhanced across participants. Frequencies of interest for each subject were considered as peak frequencies (log scale) closest to the mean peak frequency of interest of the group. Individual source waves were then computed via the inverse wavelet

transform of the CWT scales within $1/6^{th}$ of an octave below the peak frequency and $1/6^{th}$ of an octave above the frequency of interest. Each set of source waves for each participant was then subjected to extended infomax ICA. Independent components that accounted for 10% of the variance or more were retained for the brain source analysis.

Brain source analysis was completed by reconstructing the current source densities (current density reconstruction, or CDR) for each source wave using standardized low-resolution brain electromagnetic tomography (sLORETA). sLORETA solutions were constrained to a 3-shell realistic head model (scalp, skull, cortex) constructed with the boundary element method (BEM) from the averaged adult MRI (Montreal Neurological Institute) using Curry 6.1 (Compumedics-Neuroscan). Electrode positions were co-registered to the BEM based on standard 10-20 electrode positions. CDRs for each source wave were then computed using custom code in Matlab to implement the sLORETA algorithm. CDR solutions were then vector normalized and averaged across participants. Final group mean CDRs were then limited to the upper 5% of the current net (i.e., at a power threshold of 95%).

RESULTS

Children showed an interaction effect of phontactic probability and word frequency on reaction times ($F(1, 10)=13.58$, $p=0,0042$), with participants responding faster to high frequency and high probability words and low frequency and low probability words compared to high frequency and low probability words and low probability and high frequency words ([HF/HP & LF/LP] < [HF/LP & LF/HP]. There were no effects of noise on reaction time or word frequency (FIGURE 3.1). The children had an overall accuracy of 50%. There were no significant effects of phonotactic probability or word frequency on accuracy.

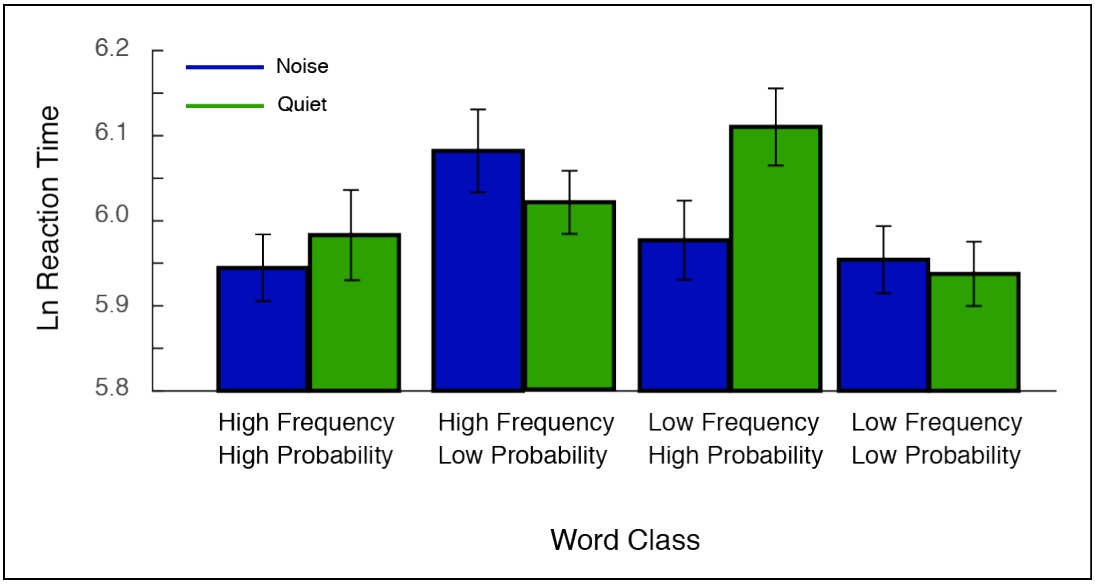

FIGURE 3.1: Average natural log reaction times. Reaction times were not significantly different in noise and in quiet. There is an interaction effect of word frequency and phonotactic probability with children responding faster to high frequency and high probability words, and low frequency and low probability words.

The T7 electrode group showed a significant effect of word frequency on N1 amplitude in children ($F(1,10)= 7.3512$, $p=0.0219$). At the T8 electrode group and PZ electrode group there was a significant effect of noise on N1 amplitude for both adults and children (T8: $F(1,10)= 18.35$, $p=0.0016$, PZ: $F(1,10)= 4.95$, $p=0.0502$). Differences in N1 amplitude may reflect differences engagement of early auditory processing networks.

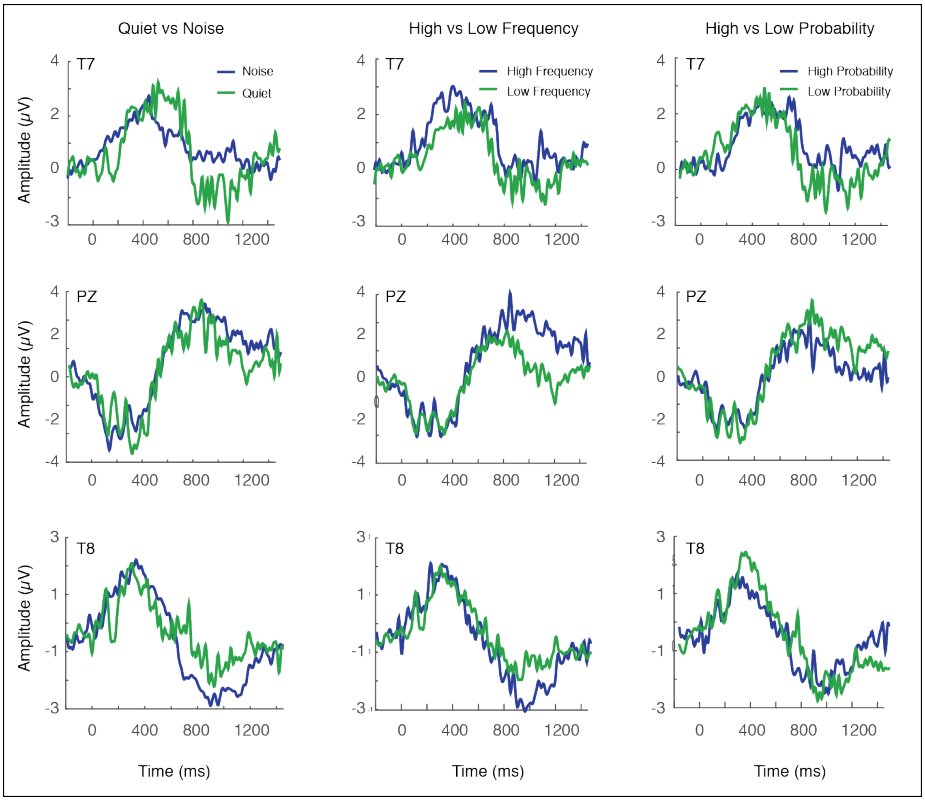

FIGURE 3.2 GROUP average ERP waveforms: Wave forms at each electrode group showing condition-level differences for noise versus quiet (left column), high versus low phonological pattern frequency (middle column), and high versus low phonotactic probability (right column). X-axes represent time in milliseconds from the onset of the stimulus. Y-axes represent ERP amplitude in microvolts. There is a significant effect of noise on the N1 response at the PZ and T8 electrode groups.

P3:

The participants did not show significant effects of word frequency, phonotactic probability or noise on the P3 amplitude.

ITPC:

Intertrial phase clustering was significant for adults in quiet in the theta and alpha frequency ranges at the T8 electrode group, and in the theta range at the PZ an T7 groups, but not in noise (FIGURE 3.3). Children showed a trend toward the adult pattern (Walker and Gilley, In preparation), but did not have significant ITPC at the 0.01 level for the T7 and PZ electrode group. There is a small ITPC burst at 10Hz in children that is above the p=0.01 level at the T8 electrode group.

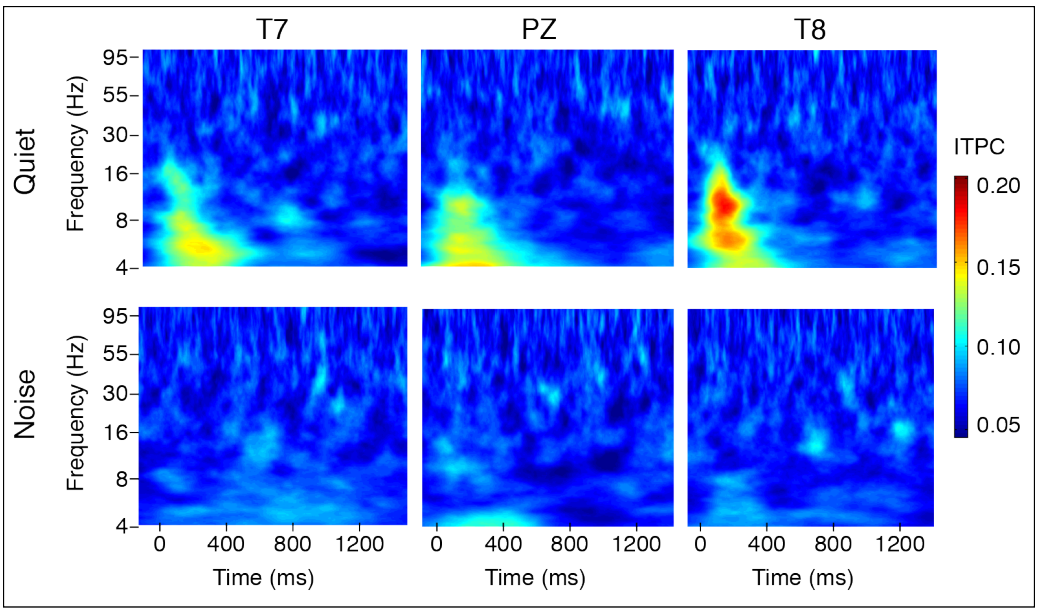

FIGURE 3.3: Intertrial phase coupling at three electrode groups: ITPC in quiet and noise at three electrode groups. For each plot, the x-axis represents time in milliseconds from the stimulus onset, the y-axis represents log-spaced frequency in Hertz, and color represents the ITPC. Significant activations are seen in the alpha frequency bands after stimulus onset at the T8 electrode group in quiet.

Power:

Children show a relative increase in alpha and beta power at the T8 electrode at the onset of the stimuli (FIGURE 3.4). They also show a relative decrease in late beta range at the T7 electrode compared to the T8 electrode group. The T7 channel group shows an enhancement of gamma activity following the onset of the stimuli. The power spectra suggest that gamma and alpha are related to early sensory processing and perception of the stumuli, while beta appears to be an early and late processing component.

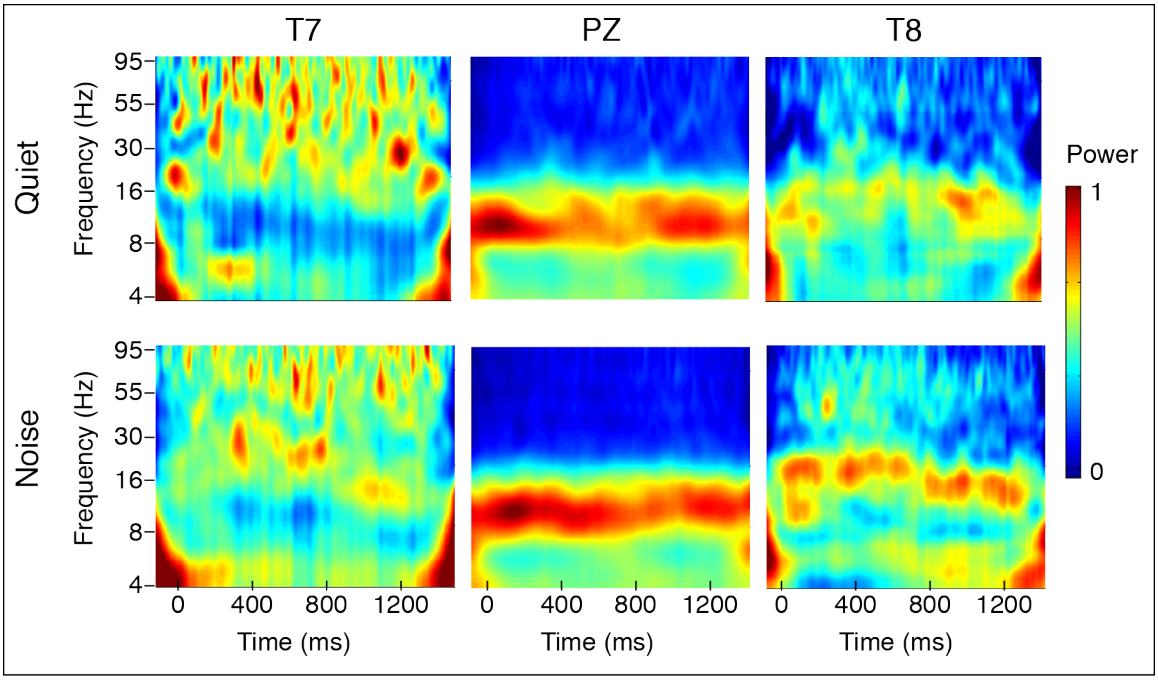

FIGURE 3.4: Average time-frequency power: Average time-frequency power at three channel groups in quiet and in noise. For each plot, the x-axis represents time in milliseconds from the stimulus onset, the y-axis represents log-spaced frequency in Hertz, and color represents the relative power. At T7 there is a large increase in gamma activity following stimulus onset in noise and quiet, and a slight increase in theta activity in quiet. The central electrodes are dominated by alpha and beta activity in both quiet and noise. T8 is characterized by a slight increase in alpha and beta activity following stimulus onset in noise with a larger increase in beta activity in noise.

The average PCA filter enhancements for the children tend to have lower frequencies in the high theta range compared to adults (7Hz vs 8 Hz), alpha range (11 Hz vs 13 Hz), and beta range (17 Hz vs 19 Hz) (see Walker & Gilley, In preparation).

Source Analysis:

The source analysis showed activation in primary thalamical regions and precuneus area (FIGURE 3.5). Sources were seen at the group level for 5 Hz, 7 Hz, 11 Hz, and 17 Hz)

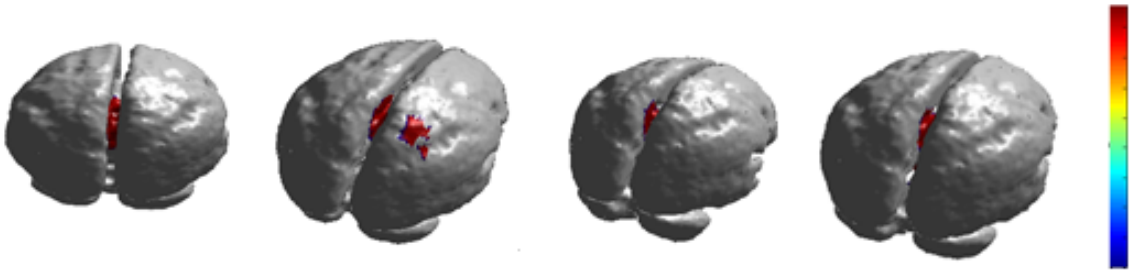

FIGURE 3.5: sLORETA solutions: Mean current density reconstructions for all participants. CDR sources are overlaid on the surface of the cortical shell used in the source model. Color represents t-values from the solution, with red indicating the upper 5th percentile. The sources from left to right are 5Hz, 7Hz, 11 Hz, and 17 Hz.

DISCUSSION

The reaction time results suggest that children are faster to identify the extreme ends of word frequency and phonotactic probability with faster recognition of high frequency and high phonotactic probability and low frequency and low phonotactic probability words compared to low frequency and high phonotactic probability words and low frequency and high phonotactic probability words. This may suggest faster recognition of words that are on these extreme ends. A similar interaction was shown by Coady et al. (2013) where they found an interaction of word frequency and phonotactic probability on word recall, although recollection was higher for all high frequency words they showed that high phonotactic probability had no impact on low frequency word recall and a positive impact on high frequency word recall. The authors were looking at encoding in a retention task, as opposed to an identification task.

In this study it may be that the unusual uniqueness of the LF\LP conditions can improve the detectability rate as these words may stand out more during the initial training due to the low occurrence of the them in terms of phonological pattern frequency and word frequency may make them "stand out" and provide participants with a strategy for identifying them. Although all the word stimuli had high familiarity scores for children the rarity of their occurrence may have impacted their perception in this task.

The largest N1 amplitude effect was driven by the noise versus quiet contrast on both the PZ and T8 electrode groups. Word frequency had a significant effect on N1 amplitude at the T7 electrode group.

The response of T7 to word frequency while T8 responded to noise may suggest that the left hemisphere is more sensitive to word pattern effects and the right hemisphere is more sensitive to noise. This is supported further by the extent of ITPC clustering in adults and

children at the T8 electrode group. Theta and alpha processing in adults appears to be more active in the right hemisphere, which would be consistent with the right hemisphere is processing more of the slow temporal structure (Giraud & Poeppel, 2012; Giraud et al., 2007; Jamison et al., 2006). Multitalker babble affects both the temporal envelop and the fine structure of the stimuli and no significant clustering is observed at any of the electrode sites of interest. In ideal listening conditions adults use phase locking to aide in processing of the incoming signal, and it appears that children in this study are trending in that direction (Walker & Gilley, In preparation). In noise additional neural processing resources appear to be aiding in the identification of the stimuli. The theta burst lines up in time with the observed N1 effect and is likely driving part of the N1 response in both children and adults.

The frequency spectrums show a late beta-band component in both noise and quiet conditions at the T7 and T8 electrode sites following gamma activity at the T7 electrode. Prior research has shown that beta band oscillations follow gamma oscillations (Haenschel, Baldeweg, Croft, Whittington, & Gruzelier, 2000). Gamma band oscillations are thought to be related to feature binding of regions of the brain that are engaged in a task during associative learning (Miltner, Braun, Arnold, Witte, & Taub, 1999; Rodriguez et al., 1999). These beta band responses may be a reflection of sensory gating(Hong et al., 2008) as they extend into the presentation of the next item and may reflect gating of the next attended item. Beta frequency differences have also been shown in children with language learning problems (LLP) in a passive listening task with children with LLP showing differences in center operating frequencies and phase amplitude coupling of the alpha and beta frequencies (Gilley et al., 2014).

The current density reconstructions for the children reveal sources in the thalamus and precuneus regions. The precuneus has been shown to be active in a prior study using a P3

paradigm with word and non-word detection (Walker & Gilley, submitted) and the precuneus region is related to episodic memory retrieval (Cavanna & Trimble, 2006) and has been shown to be active in prior studies of the P3 response (Linden, 2005). A prior study in our lab using the same experimental paradigm in adults found that adults also had low theta oscillations localized to the right temporal lobe and beta oscillations localized to the right temporal lobe (Walker & Gilley, In Preparation).

The results of this study seem to suggest that early in the response for children alpha coupling as measured at the T8 electrode group, is followed by gamma band power as seen at the T7 electrode group, and is followed by beta band activity in both the T8 and T7 electrode groups. These oscillatory networks are contributing to the processing to the word stimuli at different timescales for children in a speech perception task.

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CHAPTER 4:

WORD RECOGNTION IN NOISE IN AN ADULT AND CHILD CASE OF LANGUAGE LEARNING IMPAIRMENT

INTRODUCTION

This dissertation has focused on the neurophysiological correlates of auditory word recognition in typical adults and children in order to better understand the processes that are engaged for language. However, the central hypothesis driving this research is that networks for auditory processing function differently in persons with language learning impairments, which includes a high prevalence of comorbid disorders including specific language impairment (SLI), auditory processing disorders (APD), and reading disorders (RD). As my research career progresses, I intend to extend this work to such clinical populations, and to continue learning how these networks give rise to the language impairments observed behaviorally. In order to examine the utility of these methods in a clinical population, I tested one adult and one child with clinically diagnosed impairments, and compared their results to the group-level data reported in Chapters 2 and 3.These results highlight the complex and dynamical interactions involved in the presence of an impairment.

METHODS

One adult participant aged 18 years with a clinically diagnosed auditory processing disorder (APD) and one child aged 13 years with clinically diagnosed dyslexia were tested for this case study. The adult participant provided informed written consent prior to participating in the experiment. The child participant provided informed written assent, and the child's parent provided informed written consent as approved by the University of Colorado Institutional Review Board.

Testing protocols for each subject were coincident with the methods for their respective comparison groups as reported in Chapters 2 (adults) and 3 (children). In this case, the child

participated in the word recognition experiment (EEG) and completed a battery of tests for auditory and language processing. The adult participated in the word recognition experiment (EEG) only.

RESULTS

Typical adults and children

In order to better compare these cases, I will first summarize the similarities and differences between adults and children from the previous studies. Children and adults have similar effects of word pattern frequency and phonotactic probability on reaction times. With both children and adults reacting faster in the HF/HP and LF/LP conditions compared to the HF/LF. Children also responded slower on average than adults in the task and had less overall accuracy. Each participant's response accuracy and reaction times were similar to their peers. Of interest in this subsequent analysis was the intertrial phase coherence (ITPC) and PCA filter selection.

Intertrial phase clustering was significant for adults in quiet in the theta and alpha frequency ranges at the T8 electrode group, and in the theta range at the PZ an T7 groups, but not in noise. Children showed a trend toward the adult pattern, but did not have significant ITPC at the 0.01 level for the T7 and PZ electrode group. There is a small ITPC burst at about 10Hz in children that is above the p=0.01 level at the T8 electrode group. The average PCA filter enhancements (FIGURE 4.1) for the children and the adults show that children tend to have lower frequencies in the high theta range compared to adults (7Hz vs 8 Hz), alpha range (11 Hz vs 13 Hz), and beta range (17 Hz vs 19 Hz). Alpha frequencies tend to shift up as children age (Klimesch, 1999) which we see in these data (FIGURE 4.1).

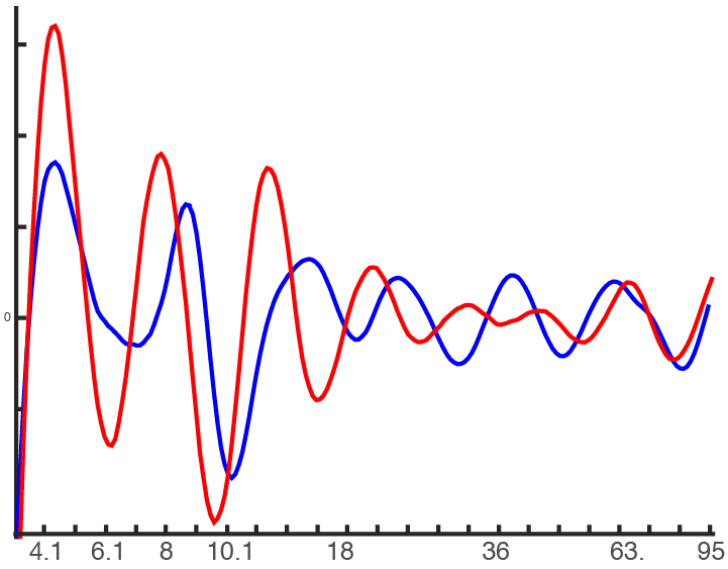

FIGURE 4.1: Children and Adult PCA Frequency Enhancements: The y-axis is relative change from zero and the x-axis is pseudo frequency from the wavelet analysis in Hz. The positive peaks represent the frequencies that are being enhanced by the PCA filter relative to the frequency spectrum.Children show frequency shifting in the PCA filter application with a lower peak in the high theta range (7Hz vs 8 Hz) in the alpha range (alpha range (11 Hz vs 13 Hz), and beta range (17 Hz vs 19 Hz).

In both adults and children source analysis shows activation of the thalamus and poseterior cingulate and precuneus. Adults however, also show some activation in the right temporal lobe in the source analysis suggesting that perhaps the right temporal region is more consistently activated in adults versus children.

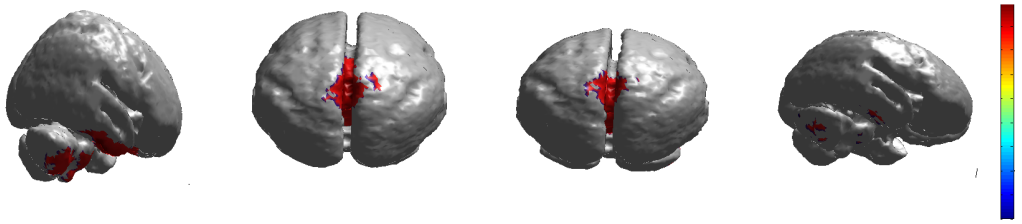

FIGURE 4.2. Source analysis for adult participants. sLORETA solutions for different frequency bands. The sources from left to right are 5Hz, 8Hz, 13 Hz, and 19 Hz. Low theta and beta frequencies source to the right temporal lobe with low theta also having some thalamical activity present. The 8Hz and 13 Hz sources both are localized to the precuneus and cingulate.

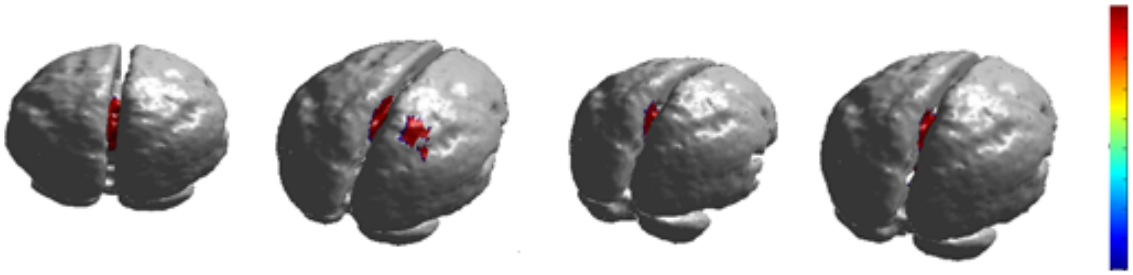

FIGURE 4.3. Source analysis for children: sLORETA solutions for different frequency bands. The sources from left to right are 5Hz, 7Hz, 11 Hz, and 17 Hz. The source analysis for all frequency bands localized to either the thalamus and/or the precuneus.

Case #1: Child with dyslexia

The first case was a thirteen-year-old child with dyslexia. His non-verbal IQ was within typical limits. He scored below average on the Frequency Pattern Test (60% in his right ear). The word classes subtest of the CELF-4 was below expected for his age (Percentile Rank 15%) and he has been diagnosed with dyslexia.

Intertrial phase clustering showed an opposite effect from his peers in that he had significant phase clustering in at the T7 electrode and less clustering at the T8 electrode site. This might suggest that he is relying more on his left auditory cortex for processing of auditory stimulation (Figure 4.4).

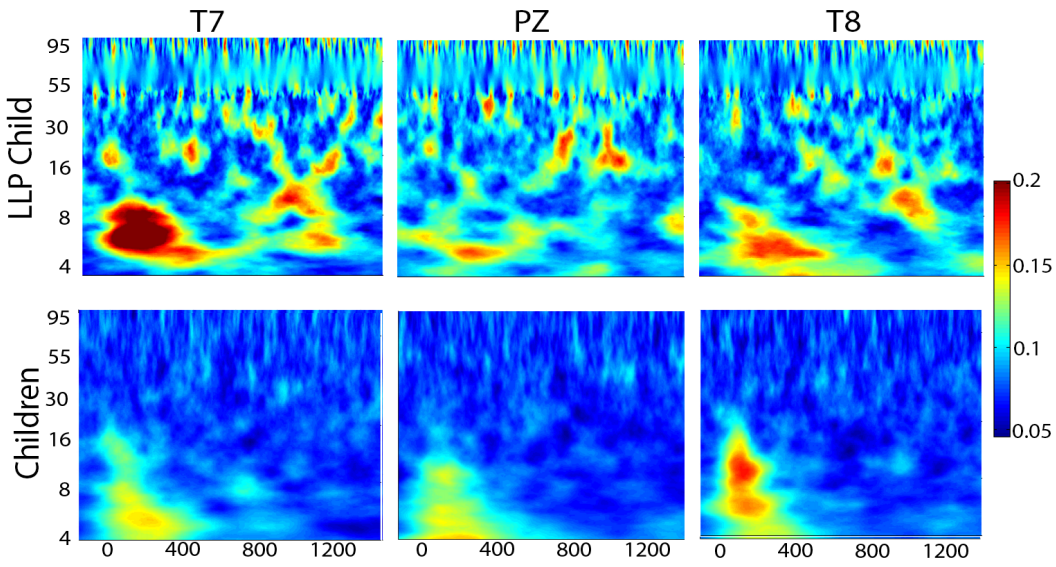

FIGURE 4.4 ITPC plots of a Child with LLP and typical children: The child with LLP shows significant phase clustering at the left T7 electrode group, while his peers show phase clustering only at the right T8 channel group.

Case #1 also shows some frequency shifting in the PCA filter enhancements. He does not show a low theta frequency peak at 5 Hz, nor does he show an alpha peak at 10 Hz (Figure 4.5). The alpha peak is still seen in typical adults, but is slightly shifted up to 13 hz. This may reflect differences in overall frequency activation from his peers.

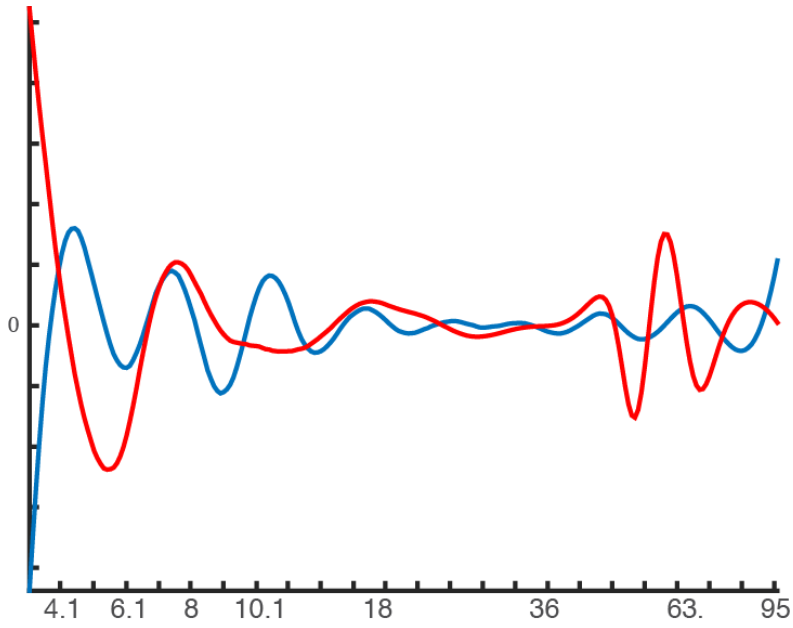

FIGURE 4.5. PCA frequency filter of child with dyslexia versus his typical peers: PCA frequency filter of child with dyslexia (red) versus his typical peers (blue). The y-axis is relative change from zero and the x-axis is pseudo frequency from the wavelet analysis in Hz. The child with dyslexia does not have a low theta peak at 4-5 Hz nor does he have an alpha peak at about 10Hz.

Case #2: Adult with auditory processing disorder

The second case is an eighteen-year-old college student who is diagnosed with auditory processing disorder. She currently uses an FM system to enhance the signal to noise ratio in her classes. Language was not assessed in this protocol. Her intertrial phase clustering (ITPC) is significant in quiet for all three electrode sites of interest; however she does not show phase clustering in the alpha range like her peers at the T8 electrode group (Figure 4.6).

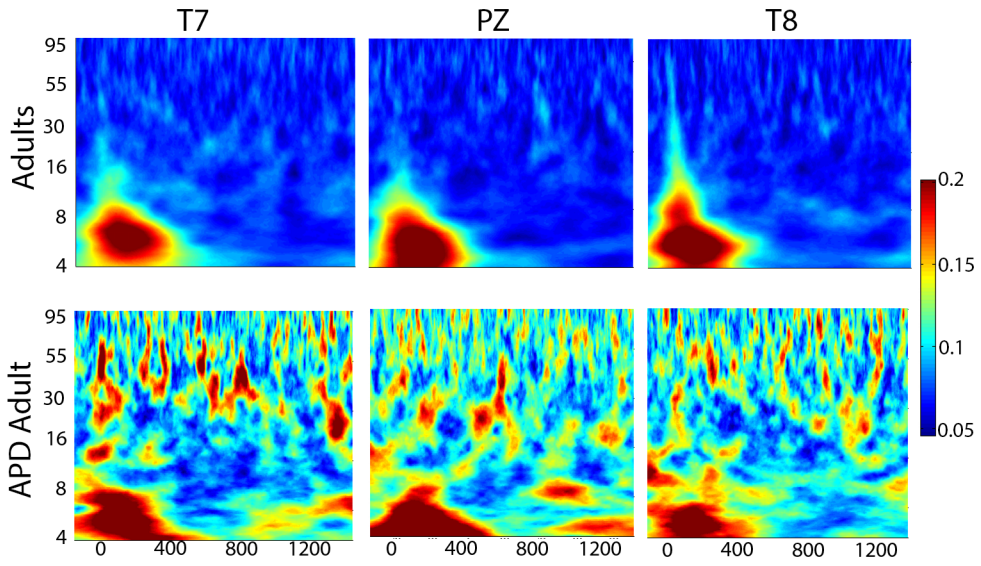

FIGURE 4.6. ITPC plots of an adult with APD versus her peers: Time is on the x-axis and relative frequency is on the y-axis on a log-scale. The adult with APD show similar patterns of phase clustering except that she does not show significant phase clustering in the alpha range after stimulus onset like her peers. She also has some significant phase clustering in the gamma frequency range with is not seen in the adult participants, but this may be do to inter-participant variability.

The adult with APD also show frequency peak shifting in the PCA filter, and is missing the low theta peak with all of her frequency peaks shifted up (or down) in the spectrum (Figure 4.7).

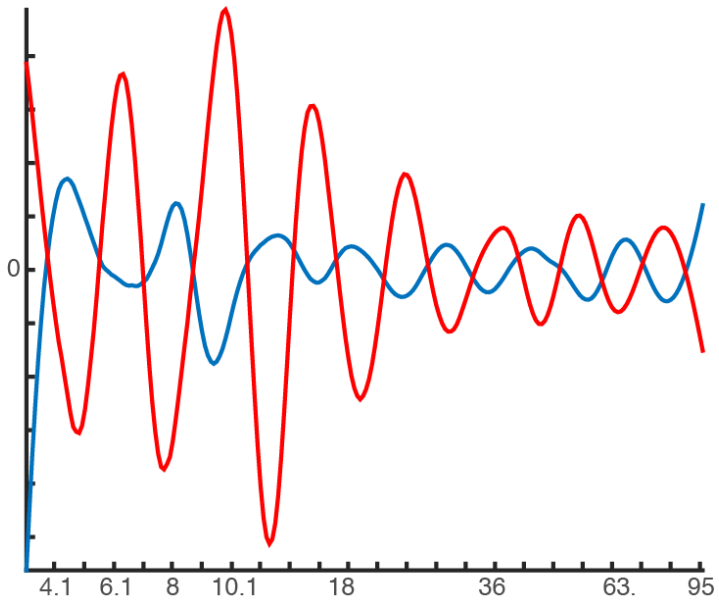

FIGURE 4.7 PCA frequency filter for an adult with APD and her peers: The y-axis is relative change from zero and the x-axis is pseudo frequency from the wavelet analysis in Hz. The positive peaks represent the frequencies that are being enhanced by the PCA filter relative to the frequency spectrum. The adult with LLP shows frequency shifting from typical adults with enhanced frequencies where typical adults have relative decreases.

DISCUSSION

The common characteristic of these two case studies is that they do not look like either the typical adults or children in their neurophysiological responses to the task. Both the adult with APD and the child with dyslexia are missing a low frequency peak in the PCA filter spectrum. Theses case studies show differences in the frequency spectrum such that the child with dyslexia is missing two of the low frequency peaks and the adult with APD showing a complete shift in oscillatory frequency peaks from the PCA.

There also appears to be some differences in the intertrial phase clustering for both the child with dyslexia and the adult with APD. The enhancement seen in the child with dyslexia may suggest atypical use of the left auditory cortex for phase clustering compared to his peers and suggest a difference in left auditory cortex processing for speech information. The adult is missing some alpha clustering compared to her peers at the T8 electrode site as well.

It has been proposed that children with LLP have differences in auditory processing and shown that they have poorer performance on dichotic listening tasks in the left ear compared to peers suggesting differences in right auditory cortex processing of dichotic information (Moncrieff & Musiek, 2002). If similar findings are shown across children with LLPs, it may suggest a difference in functional network connectivity of the left and right auditory cortices, which is in line with structural imaging studies of children with language impairments. MRI and structural imaging studies point to abnormal white and grey matter development in children with language impairments in the left hemisphere near the auditory and language regions near the superior temporal gyrus (de Vasconcelos Hage et al., 2006; Gauger et al., 1997; Kim et al., 2006; Plante et al., 1991; Trauner et al., 2000).

Taken together, these cases suggest that the underlying dynamics of language learning impairment are complex and can involve different features of processing. Coupled with our previous results (Gilley, Walker, & Sharma, 2013), the present findings also support the use of multi-dimensional EEG decomposition as a tool to assess auditory processing in these clinical populations.

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CHAPTER 5:

SUMMARY AND CONCLUSIONS

SUMMARY

Auditory speech perception is a complicated process by which the brain extracts meaningful content from the speech signal. Understanding speech processing in the typical population has the potential to aide in the development of diagnostic and treatment protocols for children with language learning problems (LLPs). In this study I examine the effects of noise, phonotactic pattern frequency, and word frequency on auditory speech perception. These factors have been shown to have a negative impact on children with language learning problems (Coady & Aslin, 2003, 2004; Coady et al., 2013; Hornickel, Zecker, Bradlow, & Kraus, 2012). The goal of this study was to examine auditory speech perception in typical adults and children so that comparisons of children with LLPs could be made in future research.

Time-frequency analysis was used to examine the effect of speech perception on the time frequency domain. We found that adults show significant intertrial phase clustering of the theta frequency range (4-8 Hz) at electrode sites near the left and right temporal lobes and in the posterior central electrode sites when processing speech in quiet. There was no significant clustering at the group level in adults in quiet. Adults also show a significant interaction effect of word frequency and phonotactic probability on reaction times, where they responded fastest to words that were high frequency and high probability and words that were low frequency and low probability.

Children also had the same interaction effect of word frequency and phonotactic probability on noise. Children showed differences in intertrial phase clustering compared to adults. Children had slight alpha rhythm intertrial phase clustering in the right electrode group examined, but no significant clustering at other frequencies or electrode sites examined. This suggests that children have a preference for relying on the right hemisphere for phase clustering

that is more prominent in adulthood. Children also show a stronger gamma frequency response in the left hemisphere compared to adults, while both children and adults have late beta responses following a word presentation.

Intertrial phase clustering appears to be important in both children and adults in the processing of speech in ideal listening environments. Alpha and theta clustering appear to be strongest in the right hemisphere. This is in line with the left and right auditory cortices processing different features of the auditory signal (Giraud & Poeppel, 2012).

Finally we applied the insights from this study to two case studies: one of a child with diagnosed dyslexia, and one of an adult with auditory processing disorder. The child with dyslexia had significant intertrial phase coherence at the left electrode group, which is the opposite of the typical children assessed. This may suggest that he shows a left auditory cortex preference for phase clustering while his peers have a right preference on average. This is in line with structural imaging studies which suggest atypical white and grey mater development in the left perisylvian region in children with language impairments (Guerreiro et al., 2002; Kim et al., 2006; Plante et al., 1991). The adult with APD showed significant phase clustering in the theta range at all electrode sites tested, but did not have significant alpha phase clustering at the right temporal electrode group assessed like her peers. She also had frequency enhancement shift as revealed by principle component analysis. Both the child with dyslexia and the adult with APD also did not have a PCA filter peak in the low theta range at about 5 Hz while both typical children and adults did.

CONCLUSION

The two experiments and the two case studies suggest that phase clustering and examining effects on theta frequency bands may be a good tool for further examining processes

that affect children and adults with language learning problems, and may reveal changes in functional connectivity for auditory speech perception in these populations.

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CHAPTER 6:

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