Neural mechanisms underlying speech perception in children with speech sound disorders

Cummings, Alycia ¹ and Wu, Ying ²

¹ Department of Communication Sciences & Disorders, Idaho State University – Meridian, Meridian, ID

² Swartz Center for Computational Neuroscience, University of California San Diego, San Diego, CA

Conflict of Interest Statement

The authors do not have any financial or non-financial relationships relevant to the content of this manuscript.

Funding Statement

This research was supported by NIH grant numbers R15DC013359 (from the National Institute on Deafness and Other Communication Disorders) and C06RR022088 (from the National Center for Research Resources) awarded to the first author.

Contact Author:

Alycia Cummings
1311 East Central Drive
Meridian, ID 83642-7991
cummalyc@isu.edu
208.373.1772

Running Head: Neural mechanisms of speech perception

ABSTRACT

Purpose: The underlying mechanisms of speech sound disorder (SSD) are unknown.

To examine the relationship between inaccurate phonological representations and speech production ability, neural changes that occurred in conjunction with speech treatment were measured.

Method: Twenty preschool-aged children with SSD participated in a traditional speech treatment program targeting a single sound (/ɪ l f θ s k/) for 19 sessions. Event-related potential (ERP) recordings were completed before and after treatment, with approximately three months between sessions. During ERP recordings, two oddball stimulus sets, each containing four consonant-vowel (CV) syllables: one standard (ba/treated sound) and three deviants (ba/treated sound, da, one other CV) were presented. ERP responses indexing speech sound encoding (auditory P1/P2) and speech sound discrimination (mismatch negativity, MMN) were measured.

Results: Children's speech discrimination responses to treated sounds shifted from a positive mismatch response (PMR) pre-treatment to a MMN response post-treatment. Pre-treatment treated sound P1/P2 latencies negatively correlated with the amount of change in speech production accuracy.

Conclusions: Children's speech discrimination responses changed from using developmentally immature neural networks pre-treatment to using more mature networks after treatment, suggesting that children's phonological representations of treated sounds became more fine-grained and/or detailed after treatment. Interestingly, children's pre-treatment neural responses correlated with treatment outcomes, providing initial evidence for a biomarker of speech production ability.

1. INTRODUCTION

Children with speech sound disorders (SSD) have difficulty producing and correctly using some sounds of their target language. Some of these children also have difficulty perceiving and categorizing these speech sounds. It is presently unknown what underlying mechanisms might account for the problems that children with SSD encounter during communication. One possible explanation is that children with SSD cannot produce speech sounds correctly because they have poorly specified phonological representations, which are the result of inaccurate speech sound perception. Thus, speech sound production errors may stem from imprecise speech perception and its resulting incorrect phonological representations.

Speech sound perception and phonological representations. Accurate speech perception is a complex process (Aslin & Smith, 1988). For example, auditory sensory information must first be detected, and then transformed into a neural representation of the event, with meaning eventually being attached to the auditory input. More specifically, phonological (i.e., speech sound) representations are formed by decoding of the speech signal, which requires, in part, the extraction and sequencing of phonetic features from the auditory signal (Scott & Wise, 2004). Being able to accurately perceive speech sounds allows for the accurate formation of phonemic categories, and more importantly, the accurate identification of words. Thus, it is important to form detailed phonological representations so that accurate speech production can occur.

It has been claimed that the speech errors children with SSD make may be due to knowledge deficits at the level of phonemic detail and/or at the level of phonemic contrasts (Rvachew & Jamieson, 1995). Specifically, it has been proposed that speech sound perception problems arise, at least in some cases, from faulty representation (i.e., memory traces) of the speech signal in the central auditory processing centers (Kraus, 2001). It is quite likely that children with SSD may

have difficulty creating phonological representations due to their inaccurate perception of speech sounds (Chaney, 1988; Macken, 1980; McGregor & Schwartz, 1992). While stable perceptual representations of speech sounds would allow children to form detailed phonological representations that can be generalized across experiences, unstable neural encoding of speech could affect children's ability to process rapidly changing acoustic information that differentiates phonemes (Carr, Tierney, White-Schwoch, & Kraus, 2016). This unstable neural encoding could lead to the creation of fuzzy (i.e., less detailed) phonological representations that do not allow children with SSD to accurately discriminate between sounds that share similar articulatory features (e.g., voicing, articulatory placement, and/or articulatory manner). More specifically, if children with SSD have fuzzy phonological representations, their ability to accurately discriminate one sound from another may be impaired. This suggests that if children do not have an appropriately detailed underlying phonological representation to access during speech production, speech production errors would likely occur. Thus ultimately, children's speech production abilities might be highly dependent on their speech perception abilities (Scott, 2012).

While no published research has examined underlying perceptual mechanisms implicated in SSD, studies involving children with developmental language disorders, reading disorders, and/or childhood apraxia of speech have identified atypical electrophysiological discriminatory responses to speech sounds (Froud & Khamis-Dakwar, 2012; Kraus et al., 1996; Sharma et al., 2006; Uwer, Albrecht, & von Suchodoletz, 2002). One potential deficit that children with SSD might share with children with developmental language disorders and reading disabilities is an impairment in phonological processing (Pennington & Bishop, 2009). Thus, it is plausible that the underlying neural responses representing the discrimination of speech sounds in children with SSD could also be atypical in nature. The present study examines how event-related

potential (ERP) measures, such as the P1/P2 and MMN, may be useful in differentiating brain activity supporting speech sound encoding and discrimination.

Speech sound encoding: P1/P2. The adult P2 (or child P1/P2)¹ reflects sound content feature encoding (Čeponienė, Torki, Alku, Koyama, & Townsend, 2008). The magnitude of the P2 elicited by speech syllables has been shown to increase substantially in adults after perceptual training of previously non-discriminable syllables (Tremblay & Kraus, 2002; Tremblay, Kraus, McGee, Ponton, & Otis, 2001). These changes in waveform morphology were interpreted as increases in neural synchrony and strengthened neural connections (Tremblay et al., 2001).

Increases in P1/P2 amplitude have also been found in children with learning impairments who completed a commercial auditory training program (Hayes, Warrier, Nicol, Zecker, & Kraus, 2003). Hayes and colleagues (2003) proposed that the increased robustness of the P1/P2 responses in the trained children was possibly due to an increase in synaptic strength of existing excitatory synapses, increased myelination of neuronal axons, and/or the recruitment of additional neurons to existing generators (Buonomano & Merzenich, 1998; Klingberg et al., 2000; Klinke, Kral, Heid, Tillein, & Hartmann, 1999). Thus, the P1/P2 appears to be a good index of speech perception training outcomes. Given that traditional speech treatment inherently includes speech perception in the process of learning to produce sounds correctly, the P1/P2 has the potential to also be a general index of speech treatment outcomes.

¹ The auditory N1 is not present in children's ERP waveforms until 7-9 years of age (Čeponienė, Rinne, & Näätänen, 2002). Until the N1 is recorded in young children, the P1 and P2 peaks cannot be distinguished from each other (Čeponienė, Alku, Westerfield, Torki, & Townsend, 2005). However, using independent component analysis, Čeponienė and colleagues (2005) were able to demonstrate that P2-like activity is present in a later portion of children's P1 peak. The P1 and P2-like activities were distinguished by differences in scalp distribution and responsivity to stimulus salience. Therefore, it appears that children's P1 represents a correlate of adult P1 and P2 peaks and will hereafter be referred to as the P1/P2.

Speech sound discrimination: MMN. The MMN is an attention-independent neurophysiological response elicited by an acoustically different (deviant) stimulus when presented in a series of homogenous (standard) stimuli (Näätänen, Paavilainen, Rinne, & Alho, 2007). Thus, the MMN is an index of automatic change detection in the brain and is thought to reflect stimulus discrimination and sensory memory (Sams, Paavilainen, Alho, & Näätänen, 1985). The MMN is sensitive to language-specific speech sound representations (Kraus, McGee, & Koch, 1998; Näätänen, 2001; Näätänen et al., 1997; Phillips et al., 2000; Winkler et al., 1999). Indeed, the MMN has been used to demonstrate the amount of detail in adult phonological representations (Cornell, Lahiri, & Eulitz, 2013; Cummings, Madden, & Hefta, 2017; Eulitz & Lahiri, 2004; Lahiri & Reetz, 2010). Thus, the MMN could be a useful measure when examining the phonological representations of children with SSD: The size of the MMN could be indicative of the detail stored in a phoneme's phonological representation. That is, the larger the MMN, the more detailed the phonological representation. In support of this interpretation, children with better phoneme processing abilities demonstrated larger MMN responses than children scoring lower on a phoneme processing test (Linnavalli, Putkinen, Huotilainen, & Tervaniemi, 2017).

The response to a stimulus mismatch is more variable in developmental populations. For example, in young children, responses are often either not a clear negativity or are fully positive in polarity (Cheour et al., 2002; Maurer, Bucher, Brem, & Brandeis, 2003). Positive mismatch responses (PMR) have been associated with neural development (Mueller, Friederici, & Männel, 2012). Specifically, different neural networks could be involved in the PMR and MMN, with superficial neural networks being recruited to produce the MMN while deep cortical neurons might generate the PMR (Kral & Eggermont, 2007; Ponton, Eggermont, Khosla, Kwong, & Don, 2002; Ponton, Moore, & Eggermont, 1999). PMR responses have often been reported in children

who have or are at-risk-for dyslexia (Volkmer & Schulte-Körne, 2018). Given that dyslexia is phonologically based, these results suggest that children with poorer or less detailed phonological representations, such as children with SSD, might demonstrate developmentally immature PMR to speech sounds. Thus, if there is a developmental trajectory of stimulus mismatch responses, children would first demonstrate a PMR that gradually shifts in polarity to eventually result in a large MMN. The transition from PMR to MMN might occur much earlier in typically developing children, as compared to children with impaired phonological processing.

The present study.

No previous study has used electrophysiological methods to examine neural change occurring in conjunction with speech treatment (i.e., behavioral) changes in children with SSD. The present study examined children's neural responses reflecting auditory encoding (P1/P2) and attention-independent discrimination (MMN) of syllables containing phonemes that children with SSD could and could not produce correctly. Two questions were asked: 1) Do neural changes occur in conjunction with speech treatment? 2) Can pre-treatment neural activity be correlated with post-treatment speech production abilities?

2. METHODS

2.1 Participants.

Twenty children with speech sound disorders (SSD) (7 male, 13 female; mean age: 58.75 months, range: 44-84 months) participated in the study. All participants had normal vision and hearing within normal limits as determined by a standard audiometric screening (American National Standards Institute, 1991). The children with SSD met the following criteria (Table 1): previous diagnosis of SSD by a certified speech-language pathologist; residency in a

monolingual English-speaking household; an oral-peripheral mechanism exam completed within normal limits (Robbins & Klee, 1987); nonverbal cognitive skills meeting or exceeding the normal range (standard scores above 85) as assessed by the Brief IQ Screener on the *Leiter International Performance Scale – Revised* (Leiter-R) (Roid & Miller, 1997); receptive vocabulary skills meeting or exceeding the normal range (standard scores above 85) as assessed by the *Peabody Picture Vocabulary Test – 4th edition* (PPVT-4) (Dunn & Dunn, 2007); and a reduced consonant inventory, excluding a minimum of three target English sounds from their respective phonemic inventories (Gierut, Simmerman, & Neumann, 1994). This study was approved by the university internal review board and a parent of each participant signed informed consent in accordance with the university human research protection program.

< Table 1 about here >

2.2 Speech Treatment.

The speech treatment procedures used in the present study are described in detail elsewhere (Cummings, Hallgrimson, & Robinson, 2018). Briefly, a single-subject multiple baseline design, in which every child served as his/her own control, was used (McReynolds & Kearns, 1983; McReynolds & Thompson, 1986). Prior to the beginning of treatment, and at the end of treatment, three speech probe assessments were administered to measure system-wide phonological change: the Goldman-Fristoe Test of Articulation, 2nd edition (Goldman & Fristoe, 2000), the 256-word *Assessment of English Phonology* (AEP) (Barlow, 2003), and the *Little PEEP: Shorter Protocol for the Evaluation of English Phonotactics* (PEEP; Barlow, 2012).

Each child received treatment of one word-initial singleton consonant: /ɪ l ʃ θ s k/. Treated phonemes were chosen based on each child's stimulability levels (Miccio, Elbert, & Forrest, 1999) and pre-intervention phonemic inventory (Gierut et al., 1994). Thus, not every child was

missing the same phoneme. The /ɪ/ was the treated sound for eight children, /l/ for three children, /ŋ/ for five children, /θ/ for two children, and /s/ and /k/ for one child each. The dose form of treatment was drill play. All sessions were 50 minutes in duration and typically occurred twice a week for 19 treatment sessions. Speech treatment involving the target sound was conducted over approximately three months for each child.

Speech production accuracy data were derived from the phonetic transcriptions of the pre- and post-treatment speech probes (Table 2). Two production accuracy values were calculated for each child at both times of evaluation: 1) the treated consonant in untreated words and 2) all consonants in untreated words (i.e., a percent consonants correct (PCC) analysis. Trained listeners used the International Phonetic Alphabet (IPA) to narrowly transcribe all speech samples using the *PHON* computer transcription and data analysis program (<https://www.phon.ca/phontrac>) (Rose & MacWhinney, 2014). Each child's speech samples were reliability-checked by a second transcriber; these speech samples were compared for point-by-point consonant agreement that was required to reach an agreement threshold of 85%.

< Table 2 about here >

2.3 ERP Stimuli.

Syllables (consonant + /a/) were pronounced by a male North American English speaker. The syllables were digitally recorded in a sound isolated room (Industrial Acoustics Company, Inc., Winchester, UK) using a Beyer Dynamic (Heilbronn, Germany) Soundstar MK II unidirectional dynamic microphone and Behringer (Willich, Germany) Eurorack MX602A mixer. All syllables were digitized with a 16-bit AD converter at a 44.1 kHz sampling rate. The average intensity of all the syllable stimuli was normalized to 65 dB SPL. As initially recorded, the syllables varied slightly in duration, due to the individual phonetic make-up of each

consonant. Syllable duration was minimally modified (by shortening the vowel duration) so that all syllables were 375 ms in length. Each syllable token used in the study was correctly identified by at least 15 adult listeners. Formant frequency measurements of /ba/ and the treated sound CV syllables are presented in Table 3.

< Table 3 about here >

All participants completed both pre-treatment and post-treatment ERP studies, with the recording dates being as close as possible to the onset and completion of each child's treatment program. All participants heard two oddball stimulus sets, each containing four English speech consonant-vowel (CV) syllables. Two of the syllables were the same for all participants: "ba" (/ba/) and "da" (/da/). The third and fourth CV syllables varied in the children with SSD, as they contained sounds the children could not produce (e.g., "ra" (/ɹa/) or "tha" (/θa/)). One of these syllables contained each child's selected treatment sound (i.e., phoneme) target /ɪ l f θ s k/.

In one stimulus set, /ba/ served as the standard syllable, with three other CV syllables serving as deviants (e.g., /ɹa/, /la/, /da/). In the second stimulus set, each individual child's treated sound CV syllable served as the standard (i.e., /ɹa/, /la/, /tʃa/, /θa/, /sa/, /ka/), with the other three syllables being deviants (e.g., /ba/, /la/, /da/). The two stimulus sets were presented back to back in the same session, with the order of the sets randomized across participants. Since just the treated sound and /ba/ syllables served as both standards and deviants (in opposite stimulus sets), only responses to these syllables will be addressed further.

2.4 ERP Stimulus Presentation.

The stimuli were presented in blocks containing 237 standard stimuli and 63 deviant stimuli (21 per deviant), with five blocks being presented to each participant. Each block lasted approximately 6 minutes and the participants were given a break between blocks when

necessary, which was typically 2-3 breaks per session. Within the block, the four stimuli were presented using an oddball paradigm in which the three deviant stimuli (probability = 7% for each) were presented in a series of standard stimuli (probability = 79%). Stimuli were presented in a pseudorandom sequence and the onset-to-onset inter-stimulus interval varied randomly between 600 and 800 ms. The syllables were delivered by stimulus presentation software (Presentation software, www.neurobs.com). The syllable sounds were played via two loudspeakers situated 30 degrees to the right and left from the midline 120 cm in front of a participant, which allowed the sounds to be perceived as emanating from the midline space. The participants sat in a sound-treated room and watched a silent cartoon video of their choice. The recording of the ERPs took approximately one hour.

2.5 EEG Recording and Averaging.

Sixty-six channels of continuous EEG (DC-128 Hz) were recorded using an ActiveTwo data acquisition system (Biosemi, Inc, Amsterdam, Netherlands) at a sampling rate of 256 Hz. This system provides “active” EEG amplification at the scalp that substantially minimizes movement artifacts. The amplifier gain on this system is fixed, allowing ample input range (-264 to 264 mV) on a wide dynamic range (110 dB) Delta- Sigma ($\Delta\Sigma$) 24-bit AD converter. Sixty-four channel scalp data were recorded using electrodes mounted in a stretchy cap according to the International 10-20 system. Two additional electrodes were placed on the right and left mastoids. Eye movements were monitored using FP1/FP2 (blinks) and F7/F8 channels (lateral movements, saccades). During data acquisition, all channels were referenced to the system’s internal loop (CMS/DRL sensors located in the centro-parietal region), which drives the average potential of a subject (the Common Mode voltage) as close as possible to the Analog-Digital Converter reference voltage (the amplifier “zero”). The DC offsets were kept below 25 microvolts at all

channels. Off-line, data were re-referenced to the common average of the 64 scalp electrode tracings.

Since the two data sets (treated sound and /ba/) were collected on the same day (pre- or post-treatment), the data were combined for processing. Data processing followed an EEGLAB (Delorme & Makeig, 2004) processing pipeline described in detail here:

https://scn.ucsd.edu/wiki/Makoto%27s_useful_EEGLAB_code#Example_of_batch_code_to_preprocess_multiple_subjects_.2801.2F27.2F2017_updated.29. Briefly, data were high-pass filtered at 0.5 Hz using a pass-band filter. Line noise was removed using the CleanLine EEGLAB plugin. Bad channels were rejected using the trimOutlier EEGLAB plugin and the removed channels were interpolated.

Source level contributions to channel EEG were decomposed using Adaptive Mixed Model Independent Component Analysis (AMICA) (Palmer, Makeig, Kreutz-Delgado, & Rao, 2008) in EEGLAB (<http://www.scn.ucsd.edu/eeglab>). Non-artifact independent component (IC) scalp topographies were modeled as projections of single equivalent dipoles and clustered on the basis of dipole locations. On average, the pre-treatment individual data contained 642 (SD = 192) treated sound standard syllable epochs (i.e., trials), 713 (SD = 153) /ba/ standard syllable epochs, 94 (SD = 27) treated sound deviant syllable epochs, and 77 (SD = 25) /ba/ deviant syllable epochs. The post-treatment individual data contained on average 712 (SD = 186) treated sound standard syllable epochs, 796 (SD = 85) /ba/ standard syllable epochs, 96 (SD = 11) treated sound deviant syllable epochs, and 85 (SD = 21) /ba/ deviant syllable epochs.

2.6 ERP Measurements.

Two data analysis strategies were used in the present study: 1) traditional MMN and P1/P2 mean amplitude and peak latency repeated measure ANOVA analyses using averaged data and 2) correlation analyses.

2.6.1 Mean amplitude and latency measurements of averaged data: MMN and P1/P2. In an oddball paradigm, the MMN is typically examined by subtracting the standard ERP response from the deviant response in difference waves. The dual stimulus set nature of the present study allowed for the creation of “same-stimulus”, or identity, difference waveforms. These difference waves were created by subtracting the ERP response of a stimulus serving as the standard from that of the same stimulus serving as the deviant, across stimulus sets. For example, the ERP response for /ba/ as the deviant was subtracted from the ERP response for /ba/ as the standard (of the reversed stimulus set) (Cornell et al., 2013). The creation of identity difference waveforms eliminates the potential confound that variations in ERP morphology may result from acoustic stimulus differences, since the same stimulus is used to elicit both the standard and deviant responses.

Given that the MMN is typically maximal over fronto-central midline electrode sites (e.g., Fz, FCz, and Cz) (Näätänen, Teder, Alho, & Lavikainen, 1992), these three electrodes were selected for the mean MMN amplitude analyses. Each maximal peak latency was first measured in the grand averaged waveforms using the ERPLAB Toolbox (Luck & Lopez-Calderon, 2012) across the 100-400 ms time window. Peak latencies elicited by the treated sound syllable and /ba/ stimuli were averaged across the three electrodes to determine the “center” latency of each peak. The “center” latency was then used to align a 100 ms window (50 ms on either side) to measure the mean amplitudes for all electrodes and stimulus types. This meant the MMN mean amplitude window was 250-350 ms. Responses elicited by the treated sound and /ba/ syllables

were examined separately using Time (Pre-treatment, Post-treatment) x Electrode (Fz, FCz, Cz) repeated measure ANOVAs.

As every participant might not demonstrate a clear and measurable MMN peak (Cummings et al., 2017), mean amplitude measurements of the auditory P1/P2 ERP peak in the standard and deviant waveforms were taken as an additional measure of neural change occurring in conjunction with speech treatment. Three regions of interest (ROI) were defined and electrodes for each ROI were defined. Left hemisphere ROI channels were F5, FC5, and C5; midline ROI channels were Fz, FCz, and Cz; right hemisphere ROI channels were F6, FC6, and C6. The P1/P2 mean amplitude was measured across a 100 ms window (100-200 ms post-syllable onset) for both the treated sound syllable and /ba/ for all nine electrodes. P1/P2 peak latency was measured during the same time window. The most positive peak within the time window was chosen automatically by the ERPLAB Toolbox (Luck & Lopez-Calderon, 2012). Once the amplitude and latency values for each electrode were measured, the median P1/P2 mean amplitude and peak latency values for each ROI were then calculated separately for each subject for both the pre-treatment and post-treatment assessments.

The P1/P2 amplitude and latency responses were examined in separate Time (Pre-treatment, Post-treatment) x Syllable Type (Standard, Deviant) x Laterality (Left, Midline, Right) repeated measure ANOVAs. As with the MMN, responses elicited by the treated sound and /ba/ syllables were examined separately. Partial eta squared (η^2) effect sizes are also reported for all significant effects and interactions. When applicable, Geisser-Greenhouse corrected p-values are reported.

2.6.2 Correlation analyses. Correlation analyses were completed to identify brain-behavior relationships occurring in conjunction with speech treatment. That is, relationships between pre-treatment ERP amplitude and latency measures and the amount of change in treated sound

accuracy from pre- to post-treatment (Table 2) were of interest, as they were considered potential indices of treatment success.

3. RESULTS

3.1 Averaged ERP Results

Only significant main effects and interactions are reported.

3.1.1 MMN Mean Amplitude Analysis. The MMN amplitude elicited by the treated sound standard syllable changed significantly from pre- to post-treatment $F(1,19) = 5.242$, $p < 0.04$, $\eta^2 = .216$ (Table 4, Figure 1). While children's pre-treatment responses during the MMN time window were positive (i.e., a PMR), a MMN response developed post-treatment. No significant MMN amplitude changes were observed for /ba/.

< Figure 1 about here >

< Table 4 about here >

3.1.2 P1/P2 Mean Amplitude Analysis. When evaluating the treated sound P1/P2 amplitudes, a significant difference in syllable type was observed $F(1,19) = 4.476$, $p < 0.05$, $\eta^2 = .191$ (Table 5, Figure 1). Specifically, the treated sound deviant stimulus P1/P2 amplitude was greater than that of the standard stimulus. No significant P1/P2 amplitude effects were observed for /ba/.

< Table 5 about here >

3.1.3 P1/P2 Peak Latency Analysis. The P1/P2 latency response elicited by /ba/ was significantly earlier post-treatment than it was pre-treatment ($F(1,19) = 5.461$, $p < 0.04$, $\eta^2 = .223$) (Table 6). Moreover, the /ba/ standard syllables elicited earlier P1/P2 responses than did the /ba/ deviants ($F(1,19) = 13.748$, $p < 0.002$, $\eta^2 = .420$). No significant P1/P2 latency effects were observed for the treated sound.

< Table 6 about here >

3.2 Correlation Analyses

3.2.1 P1/P2 latency - Treated sound accuracy change. Change in treated sound production accuracy correlated with the peak latency of the pre-treatment treated sound deviant midline P1/P2 responses ($r(20) = -0.571, p < .01$)². Thus, children with earlier P1/P2 peak latencies prior to beginning treatment demonstrated greater change in their treated sound production accuracy at the end of treatment (Figure 2). Neural responses elicited by /ba/ did not correlate with treated sound accuracy change.

< Figure 2 about here >

4. DISCUSSION

Children with speech sound disorders (SSD) participated in event-related potential (ERP) oddball paradigms prior to beginning a speech treatment program and at the conclusion of that treatment. Children's responses to two speech syllables were of interest. One syllable, /ba/, contained a consonant that all children could accurately produce prior to beginning treatment. The second syllable contained the consonant (e.g., /l tʃ θ s k/) that was targeted during each child's individually-designed speech treatment program.

Do neural changes occur in conjunction with speech treatment? The children with SSD demonstrated significant changes in their mismatch responses to the treated sounds. The children demonstrated a positive mismatch response (PMR) to their treated sounds prior to beginning treatment, which shifted to a negative mismatch response (MMN) after treatment concluded. The pre-treatment PMR could indicate that children with SSD had atypical sound discriminatory

² This effect remained significant after correcting for multiple comparisons of the left, midline, and right ROIs (.05/3 = .0167).

responses to sounds that they could not produce correctly. Instead of using the superficial neural networks that elicit the MMN, deep cortical neurons were recruited to make the syllable discrimination responses (Kral & Eggermont, 2007; Ponton et al., 2002, 1999). Since axonal connectivity develops later in the superficial cortical layers, the PMR could be evidence that the children with SSD were using ontogenetically less mature perceptual discrimination mechanisms. That the PMR changed into the MMN provides evidence for neural changes occurring in conjunction with treatment. That is, the phonological representations of children with SSD might have become more consistent, fine-grained, and/or detailed with intervention (Čeponienė et al., 2008; Tremblay & Kraus, 2002).

Theories of attention and learning could be used to explain the shift from PMR to MMN (Kraus & Chandrasekaran, 2010; Kraus et al., 2014; Patel, 2011). The children's neurophysiological changes/enhancements were likely driven by the process of speech treatment. Teaching children to accurately produce their specific treated sound also necessarily directed their attention to that sound. Actively engaging with their treated sound through hundreds of word productions in treatment likely made the sound salient to the children. Subsequently, this sound-meaning correspondence improved neural responses (Kraus & Chandrasekaran, 2010; Kraus et al., 2014). Thus, the children's auditory discrimination abilities might have been enhanced by their improved attention (Fritz, Shamma, Elhilali, & Klein, 2003).

In addition, the shortened post-treatment /ba/ P1/P2 latency could be a treatment-related effect. To explain, children with SSD likely have less detailed phonological representations of the sounds that they cannot produce. However, it is a possibility that children's phonological representations of *all* sounds are not fully established. For example, typical speech sound acquisition is expected to continue up until the age of eight years (Sander, 1972), with

acquisition in children with SSD often taking much longer. Thus, even though the children with SSD could produce /b/ without issue, the underlying neural networks involved in encoding and discriminating /b/ might have still been developing in the children in the present sample. Given that the speech treatment protocol used in the present study was designed to elicit systemwide phonological change (Gierut, 2007), the change in /ba/ latency might reflect treatment outcomes. Alternatively, it is a possibility that the changes in the children's P1/P2 latencies were developmental in nature (Shafer, Yu, & Wagner, 2015). Given that approximately three months passed between ERP recording sessions, the children might have experienced neural development and changes that were unrelated to speech treatment. Including TD children in the study would have clarified whether the observed P1/P2 latency changes were due to general development or treatment.

Could pre-treatment neural activity be correlated with post-treatment speech production abilities? It is nearly impossible for speech-language pathologists (SLPs) to predict how well a child will perform in treatment, as children's outcomes can be quite varied. While behavioral predictors of speech treatment outcomes have not yet been identified, it is possible that the neural patterns children demonstrate prior to beginning treatment may indicate how well they will learn to produce a treated sound. In the present study, the P1/P2 latencies of children's responses to their treated sound negatively correlated with treated sound accuracy change. That is, the children who made more change in the production accuracy of their treated sound had earlier P1/P2 responses. With sound feature encoding represented by the P1/P2 (Čeponienė et al., 2008), children with earlier peak latencies were faster and more efficient in the encoding of the phonological features of their treated sound than those with later responses. Thus, the children who were the faster processors of phonological information had greater treatment gains.

The present study provides initial evidence that ERP responses could be indices of children's speech production ability and/or speech treatment effectiveness. At the group level, the MMN and P1/P2 latency were associated with changes occurring in conjunction with speech treatment. Specifically, the present evidence suggests that children with SSD might be recruiting developmentally immature neural networks for the processing of speech sounds, which might not allow for full and accurate processing and discrimination of phonological information. Moreover, children with SSD vary in the speed with which they encode phonological information. This processing speed is subsequently related to how much phonological change is observed after speech treatment. These findings suggest that SSD may be linked to atypical and/or inefficient neural networks involved in the encoding and discrimination of single phonemes.

Limitations. The present study provided evidence that speech treatment results in neural changes to speech sounds. That being said, one limitation concerns the treated sounds that were central to the speech treatment program. Ideally, all children would have had the same sound in error, but given the heterogenous nature of SSD, that was not the case. Six different treated sound syllables were used. The acoustic variability in the treated sounds could have resulted in a range of P1/P2 peak latency measures, which when grouped together masked treatment-related latency changes. For example, some of the consonants had an abrupt onset (e.g., /ka/), while other consonants had a more gradual/extended onset (e.g., /ɪa/, /la/, /θa/, /sa/). Thus, children's encoding (i.e., P1/P2) of some treated consonants could have been delayed up to the point of clear identification of the sound, which likely varied across children. Alternatively, /ba/ had a discrete, abrupt onset that established a distinct point of sound identification. Moreover, all children responded to /ba/, which resulted in responses that were consistent enough to elicit

P1/P2 latency differences. Future studies examining children's responses to a single treated sound will provide converging evidence for the preliminary findings reported here.

Along with the variety of treated sounds in the present study was the variation in children's ages. Brain development occurs in stages, with the areas of cortex involved in auditory perception completing synaptic pruning between four and six years of age (Tierney & Nelson, 2009). Given that the children in the present study were primarily between the ages of four and six, it is a distinct possibility that the ERP responses elicited by the children varied to a greater extent than might otherwise have been expected due to the overlap in timing with the developmental event of synaptic pruning. This age-related variation in responses also likely masked some of the neural changes that occurred in conjunction with treatment.

Unfortunately, it is difficult to find a way around this developmentally-based problem. Approximately 15% of 3-year-olds have SSD (Campbell et al., 2003), with the prevalence of SSD decreasing to approximately 4% by the age of six (Shriberg, Tomblin, & McSweeney, 1999). Thus, preschool-aged children are the largest (and best) population to include in studies of SSD. Moreover, preschool children often make greater gains in treatment than do older children, quite likely due to the fact that their brains have greater flexibility to adapt and respond to their environment, which includes speech treatment. Future studies involving a greater number of participants will help reduce the variability in ERP responses and likely provide clearer evidence of how speech treatment alters neural responses.

Future directions. This study was not only the first to measure electrophysiological responses to phonemes in children with SSD, it was also the first to measure neural changes occurring in conjunction with traditional speech treatment. New insight into the underlying mechanisms of SSD was uncovered. Specifically, pre-treatment data suggested that children with SSD were

using atypical, or at least developmentally immature, neural networks to discriminate speech sounds they could not produce correctly. The pattern of children's speech discrimination responses changed after treatment, suggesting that extended practice producing (and hearing) a sound developed and trained children's neural response networks. Moreover, children's responses to sounds quickened after treatment, suggesting that their speech encoding systems became more efficient in processing information as treatment progressed.

While changes to children's mismatch response were observed, these responses should not be interpreted as indicators of fully remediated phonological representations. The children's post-treatment MMN responses were typically very small in amplitude. Given that children with good phoneme processing abilities have large MMN responses (Linnavalli et al., 2017), it is likely that the children in the present study were at the beginning stages of phonological development. In addition, while the latency of the mismatch response was not measured in the present study, the MMN analysis window was 250-350 ms based on visual analysis of grand averaged waveforms. Thus, the mismatch response had a latency that was later than the 150-250 ms time window that is typically reported in the MMN literature (Volkmer & Schulte-Körne, 2018). Longer latency mismatch responses are often associated with younger children (Alonso-Búa, Díaz, & Ferraces, 2006), and ERP latency reductions are often associated with cortical maturation (Shafer et al., 2015). Thus, the results of the present study suggest that the children with SSD were still developing their abilities to discriminate speech sounds. To determine whether the observed neural changes were due to the speech treatment process, or to general development, future studies should compare the responses of age-matched typically developing children to children with SSD.

That being said, the timing of children's pre-treatment treated sound neural responses correlated with their treatment outcomes. This result suggests that there might be certain time points in [brain] development when children may be more able to take advantage of treatment. The relationship between the overproduction, and subsequent pruning, of synapses in the brain is often related to the ease with which behavioral skills, such as speech perception (Kuhl, 2004), can be acquired (Tierney & Nelson, 2009). Moreover, the auditory system goes through a period of rapid development during the first four years of life (Choudhury & Benasich, 2011), though the rate of change likely varies across children (White-Schwoch & Kraus, 2013). Thus, it is not completely unfounded to think that there could also be a point in time where children's brains are most prepared to learn to produce speech sounds. Alternatively, it is possible that some brains might just be better able to process sounds (as indicated by the P1/P2 latency) due to inherent perceptual abilities.

Since not all of the children had the same pre-treatment neural responses, some children were inherently better prepared for treatment. For the children who were not as prepared, the question is whether there are activities they could complete prior to starting speech treatment that might improve their response to intervention. That is, are there activities that could enhance neural development prior to the onset of treatment so that the brain is "primed" and prepared to learn during treatment? Certain activities have been found to improve children's neural responses to speech sounds. For example, children can develop stronger neurophysiological distinctions of stop consonants (e.g., /b/ and /g/) after participating in music training classes (Kraus et al., 2014). Moreover, children who can demonstrate beat synchronization while drumming can have more precise neural encoding of speech syllables, as well as better perceptual and cognitive language skills (Woodruff Carr, White-Schwoch, Tierney, Strait, & Kraus, 2014). These studies suggest

that the ability to detect the rhythm and timing of sounds has implications for speech production. Learning to detect rhythmic patterns prior to beginning treatment may allow children to better identify the speech patterns in treatment words.

Speech perception training has also been found to improve children's productive speech abilities. Children who receive traditional speech treatment in conjunction with perception training of their errored sound can make greater treatment gains than children who receive perception training of unrelated sounds or listen to books (Rvachew, 1994; Rvachew, Nowak, & Cloutier, 2004). Based on these findings, it is possible that a speech perception program might enhance children's auditory perceptual responses. Being able to distinguish the subtle differences between speech sounds is likely to subsequently improve speech production accuracy. It is to be determined whether speech treatment studies incorporating pre-treatment activities such as music, drumming, or speech perception may elicit greater gains in children's speech production abilities than traditional treatment alone.

It is not yet clear how speech-language pathologists (SLPs) could potentially identify a sensitive period for speech perception and/or production. While ERP methodology appears to have a promising future in the identification of the underlying neural mechanisms of SSD, it is not a functional clinical diagnostic tool. A promising neurophysiological alternative to ERPs is to record children's auditory brainstem responses (ABR), and specifically the Frequency Following Response (FFR) of the ABR. Given that FFRs have been correlated with children's reading ability and literacy skills (Carr et al., 2016; Hornickel & Kraus, 2013; White-Schwoch et al., 2015), it is likely that FFRs could also be correlated with speech production ability. Measuring cortical and subcortical responses to speech sounds together in the future will provide a wider

array of neural measures useful in identifying neurophysiological markers of speech ability and treatment effectiveness.

ACKNOWLEDGEMENTS

This research was supported by NIH grant numbers R15DC013359 (from the National Institute on Deafness and Other Communication Disorders) and C06RR022088 (from the National Center for Research Resources) awarded to the first author. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health. The authors do not have any financial or non-financial relationships relevant to the content of this manuscript.

REFERENCES

- Alonso-Búa, B., Díaz, F., & Ferraces, M. J. (2006). The contribution of AERPs (MMN and LDN) to studying temporal vs. linguistic processing deficits in children with reading difficulties. *International Journal of Psychophysiology*, 59(2), 159–167.
<https://doi.org/10.1016/j.ijpsycho.2005.03.020>
- American National Standards Institute. (1991). *American national standard specifications for audiometers (ANSI S3.6-1969)*. New York, NY: ANSI.
- Aslin, R. N., & Smith, L. B. (1988). Perceptual development. *Annual Review of Psychology*, 39, 435–473. <https://doi.org/10.1146/annurev.ps.39.020188.002251>
- Barlow, J. A. (2003). *The Assessment of English Phonology (AEP)*. San Diego, CA: San Diego State University.
- Barlow, J. A. (2012). *Little PEEP: Shorter Protocol for the Evaluation of English Phonotactics*. San Diego, CA: San Diego State University.
- Campbell, T. F., Dollaghan, C. A., Rockette, H. E., Paradise, J. L., Feldman, H. M., Shriberg, L. D., ... Kurs-Lasky, M. (2003). Risk factors for speech delay of unknown origin in 3-year-old children. *Child Development*, 74(2), 346–357.
- Carr, K. W., Tierney, A., White-Schwoch, T., & Kraus, N. (2016). Intertrial auditory neural stability supports beat synchronization in preschoolers. *Developmental Cognitive Neuroscience*, 17, 76–82. <https://doi.org/10.1016/j.dcn.2015.12.003>
- Čeponienė, R., Alku, P., Westerfield, M., Torki, M., & Townsend, J. (2005). ERPs differentiate syllable and nonphonetic sound processing in children and adults. *Psychophysiology*, 42(4), 391–406. <https://doi.org/10.1111/j.1469-8986.2005.00305.x>

- Čeponienė, R., Rinne, T., & Näätänen, R. (2002). Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*, 113(6), 870–882.
- Čeponienė, R., Torki, M., Alku, P., Koyama, A., & Townsend, J. (2008). Event-related potentials reflect spectral differences in speech and non-speech stimuli in children and adults. *Clinical Neurophysiology*, 119(7), 1560–1577.
<https://doi.org/10.1016/j.clinph.2008.03.005>
- Chaney, C. (1988). Identification of Correct and Misarticulated Semivowels. *Journal of Speech and Hearing Disorders*, 53(3), 252–261.
- Cheour, M., Čeponienė, R., Leppänen, P., Alho, K., Kujala, T., Renlund, M., ... Näätänen, R. (2002). The auditory sensory memory trace decays rapidly in newborns. *Scandinavian Journal of Psychology*, 43(1), 33–39.
- Choudhury, N., & Benasich, A. A. (2011). Maturation of auditory evoked potentials from 6 to 48 months: prediction to 3 and 4 year language and cognitive abilities. *Clinical Neurophysiology*, 122(2), 320–338. <https://doi.org/10.1016/j.clinph.2010.05.035>
- Cornell, S. A., Lahiri, A., & Eulitz, C. (2013). Inequality across consonantal contrasts in speech perception: evidence from mismatch negativity. *Journal of Experimental Psychology. Human Perception and Performance*, 39(3), 757–772. <https://doi.org/10.1037/a0030862>
- Cummings, A., Hallgrimson, J., & Robinson, S. (2018). Speech intervention outcomes associated with word lexicality and intervention intensity. *Language, Speech, and Hearing Services in Schools*, 1–16. https://doi.org/10.1044/2018_LSHSS-18-0026
- Cummings, A., Madden, J., & Hefta, K. (2017). Converging evidence for [coronal] underspecification in English-speaking adults. *Journal of Neurolinguistics*, 44, 147–162.
<https://doi.org/10.1016/j.jneuroling.2017.05.003>

- Delorme, A., & Makeig, S. (2004). EEGLAB: An open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *Journal of Neuroscience Methods*, 134(1), 9–21. <https://doi.org/10.1016/j.jneumeth.2003.10.009>
- Dunn, L., & Dunn, D. (2007). *Peabody Picture Vocabulary Test - 4th ed.* Circle Pines, MN: American Guidance Service Publishing/Pearson Assessments.
- Eulitz, C., & Lahiri, A. (2004). Neurobiological evidence for abstract phonological representations in the mental lexicon during speech recognition. *Journal of Cognitive Neuroscience*, 16(4), 577–583.
- Fritz, J., Shamma, S., Elhilali, M., & Klein, D. (2003). Rapid task-related plasticity of spectrotemporal receptive fields in primary auditory cortex. *Nature Neuroscience*, 6(11), 1216–1223. <https://doi.org/10.1038/nm1141>
- Froud, K., & Khamis-Dakwar, R. (2012). Mismatch Negativity responses in children with a diagnosis of Childhood Apraxia of Speech (CAS). *American Journal of Speech-Language Pathology*, 21(4), 302–312. [https://doi.org/10.1044/1058-0360\(2012/11-0003\)](https://doi.org/10.1044/1058-0360(2012/11-0003))
- Gierut, J. A. (2007). Phonological complexity and language learnability. *American Journal of Speech-Language Pathology*, 16(1), 6–17. [https://doi.org/10.1044/1058-0360\(2007/003\)](https://doi.org/10.1044/1058-0360(2007/003))
- Gierut, J. A., Simmerman, C. L., & Neumann, H. J. (1994). Phonemic structures of delayed phonological systems. *Journal of Child Language*, 21(2), 291–316.
- Goldman, R., & Fristoe, M. (2000). *Goldman-Fristoe Test of Articulation - 2nd ed.* Minneapolis, MN: Pearson.
- Hayes, E. A., Warrier, C. M., Nicol, T. G., Zecker, S. G., & Kraus, N. (2003). Neural plasticity following auditory training in children with learning problems. *Clinical Neurophysiology*, 114(4), 673–684.

- Hornickel, J., & Kraus, N. (2013). Unstable representation of sound: A biological marker of dyslexia. *The Journal of Neuroscience*, 33(8), 3500–3504.
<https://doi.org/10.1523/JNEUROSCI.4205-12.2013>
- Kral, A., & Eggermont, J. J. (2007). What's to lose and what's to learn: Development under auditory deprivation, cochlear implants and limits of cortical plasticity. *Brain Research Reviews*, 56(1), 259–269. <https://doi.org/10.1016/j.brainresrev.2007.07.021>
- Kraus, N. (2001). Auditory pathway encoding and neural plasticity in children with learning problems. *Audiology & Neuro-Otology*, 6(4), 221–227. <https://doi.org/46837>
- Kraus, N., & Chandrasekaran, B. (2010). Music training for the development of auditory skills. *Nature Reviews. Neuroscience*, 11(8), 599–605. <https://doi.org/10.1038/nrn2882>
- Kraus, N., McGee, T. J., Carrell, T. D., Zecker, S. G., Nicol, T. G., & Koch, D. B. (1996). Auditory neurophysiologic responses and discrimination deficits in children with learning problems. *Science (New York, N.Y.)*, 273(5277), 971–973.
- Kraus, N., McGee, T. J., & Koch, D. B. (1998). Speech sound representation, perception, and plasticity: A neurophysiologic perspective. *Audiology & Neuro-Otology*, 3(2–3), 168–182.
- Kraus, N., Slater, J., Thompson, E. C., Hornickel, J., Strait, D. L., Nicol, T., & White-Schwoch, T. (2014). Music enrichment programs improve the neural encoding of speech in at-risk children. *The Journal of Neuroscience*, 34(36), 11913–11918.
<https://doi.org/10.1523/JNEUROSCI.1881-14.2014>
- Kuhl, P. K. (2004). Early language acquisition: Cracking the speech code. *Nature Reviews. Neuroscience*, 5(11), 831–843. <https://doi.org/10.1038/nrn1533>
- Lahiri, A., & Reetz, H. (2010). Distinctive features: Phonological underspecification in representation and processing. *Journal of Phonetics*, 38, 44–59.

- Linnavalli, T., Putkinen, V., Huotilainen, M., & Tervaniemi, M. (2017). Phoneme processing skills are reflected in children's MMN responses. *Neuropsychologia*, 101, 76–84. <https://doi.org/10.1016/j.neuropsychologia.2017.05.013>
- Luck, S., & Lopez-Calderon, J. (2012). *ERPLAB Toolbox*. University of California, Davis.
- Macken, M. A. (1980). The child's lexical representation: The 'puzzle-puddle-pickle' evidence. *Journal of Linguistics*, 16(01), 1–17. <https://doi.org/10.1017/S0022226700006307>
- Maurer, U., Bucher, K., Brem, S., & Brandeis, D. (2003). Development of the automatic mismatch response: From frontal positivity in kindergarten children to the mismatch negativity. *Clinical Neurophysiology*, 114(5), 808–817.
- McGregor, K. K., & Schwartz, R. G. (1992). Converging evidence for underlying phonological representation in a child who misarticulates. *Journal of Speech and Hearing Research*, 35(3), 596–603.
- McReynolds, L. V., & Kearns, K. P. (1983). *Single-subject experimental designs in communicative disorders*. Austin, Tex.: PRO-ED.
- McReynolds, L. V., & Thompson, C. K. (1986). Flexibility of single-subject experimental designs. Part I: Review of the basics of single-subject designs. *Journal of Speech and Hearing Disorders*, 51(3), 194–203.
- Miccio, A. W., Elbert, M., & Forrest, K. (1999). The relationship between stimulability and phonological acquisition in children with normally developing and disordered phonologies. *American Journal of Speech-Language Pathology*, 8, 347–363.
- Mueller, J. L., Friederici, A. D., & Männel, C. (2012). Auditory perception at the root of language learning. *Proceedings of the National Academy of Sciences of the United States of America*, 109(39), 15953–15958. <https://doi.org/10.1073/pnas.1204319109>

- Näätänen, R. (2001). The perception of speech sounds by the human brain as reflected by the mismatch negativity (MMN) and its magnetic equivalent (MMNm). *Psychophysiology*, 38(1), 1–21.
- Näätänen, R., Lehtokoski, A., Lennes, M., Cheour, M., Huotilainen, M., Iivonen, A., ... Alho, K. (1997). Language-specific phoneme representations revealed by electric and magnetic brain responses. *Nature*, 385(6615), 432–434. <https://doi.org/10.1038/385432a0>
- Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: A review. *Clinical Neurophysiology*, 118(12), 2544–2590. <https://doi.org/10.1016/j.clinph.2007.04.026>
- Näätänen, R., Teder, W., Alho, K., & Lavikainen, J. (1992). Auditory attention and selective input modulation: a topographical ERP study. *Neuroreport*, 3(6), 493–496.
- Palmer, J. A., Makeig, S., Kreutz-Delgado, K., & Rao, B. D. (2008). Newton method for the ICA mixture model. In *Acoustics, Speech, and Signal Processing, 2008. ICASSP 2008*. (Vol. 33rd, pp. 1805–1808). Las Vegas, NV.
- Patel, A. D. (2011). Why would musical training benefit the neural encoding of speech? The OPERA Hypothesis. *Frontiers in Psychology*, 2, 142. <https://doi.org/10.3389/fpsyg.2011.00142>
- Pennington, B. F., & Bishop, D. V. M. (2009). Relations among speech, language, and reading disorders. *Annual Review of Psychology*, 60, 283–306. <https://doi.org/10.1146/annurev.psych.60.110707.163548>
- Phillips, C., Pellathy, T., Marantz, A., Yellin, E., Wexler, K., Poeppel, D., ... Roberts, T. (2000). Auditory cortex accesses phonological categories: An MEG mismatch study. *Journal of Cognitive Neuroscience*, 12(6), 1038–1055.

- Ponton, C. W., Eggermont, J. J., Khosla, D., Kwong, B., & Don, M. (2002). Maturation of human central auditory system activity: Separating auditory evoked potentials by dipole source modeling. *Clinical Neurophysiology*, 113(3), 407–420.
- Ponton, C. W., Moore, J. K., & Eggermont, J. J. (1999). Prolonged deafness limits auditory system developmental plasticity: Evidence from an evoked potentials study in children with cochlear implants. *Scandinavian Audiology*, 51, 13–22.
- Robbins, J., & Klee, T. (1987). Clinical assessment of oropharyngeal motor development in young children. *Journal of Speech and Hearing Disorders*, 52(3), 271–277.
- Roid, G., & Miller, L. (1997). *Leiter International Performance Scale - Revised (Leiter-R)*. Wood Dale, IL: Stoelting.
- Rose, Y., & MacWhinney, B. (2014). The PhonBank Project: Data and software-assisted methods for the study of phonology and phonological development. In J. Durand, U. Gut, & G. Kristoffersen (eds.), *The Oxford Handbook of Corpus Phonology* (pp. 308–401). Oxford: Oxford University Press.
- Rvachew, S. (1994). Speech perception training can facilitate sound production learning. *Journal of Speech and Hearing Research*, 37(2), 347–357.
- Rvachew, S., & Jamieson, D. (1995). Learning new speech contrasts: Evidence from adults learning a second language and children with speech disorders. In W. Strange (ed.), *Speech perception and linguistic experience: Issues in cross-language research* (pp. 411–432). Timonium, MD: York Press.
- Rvachew, S., Nowak, M., & Cloutier, G. (2004). Effect of phonemic perception training on the speech production and phonological awareness skills of children with expressive

- phonological delay. *American Journal of Speech-Language Pathology*, 13(3), 250–263.
[https://doi.org/10.1044/1058-0360\(2004/026\)](https://doi.org/10.1044/1058-0360(2004/026))
- Sams, M., Paavilainen, P., Alho, K., & Näätänen, R. (1985). Auditory frequency discrimination and event-related potentials. *Electroencephalography and Clinical Neurophysiology*, 62(6), 437–448.
- Sander, E. K. (1972). When are speech sounds learned? *The Journal of Speech and Hearing Disorders*, 37(1), 55–63.
- Scott, S. K. (2012). The neurobiology of speech perception and production--can functional imaging tell us anything we did not already know? *Journal of Communication Disorders*, 45(6), 419–425. <https://doi.org/10.1016/j.jcomdis.2012.06.007>
- Scott, S. K., & Wise, R. J. S. (2004). The functional neuroanatomy of prelexical processing in speech perception. *Cognition*, 92(1–2), 13–45.
<https://doi.org/10.1016/j.cognition.2002.12.002>
- Shafer, V. L., Yu, Y. H., & Wagner, M. (2015). Maturation of cortical auditory evoked potentials (CAEPs) to speech recorded from frontocentral and temporal sites: Three months to eight years of age. *International Journal of Psychophysiology*, 95(2), 77–93.
<https://doi.org/10.1016/j.ijpsycho.2014.08.1390>
- Sharma, M., Purdy, S. C., Newall, P., Wheldall, K., Beaman, R., & Dillon, H. (2006). Electrophysiological and behavioral evidence of auditory processing deficits in children with reading disorder. *Clinical Neurophysiology*, 117(5), 1130–1144.
<https://doi.org/10.1016/j.clinph.2006.02.001>

- Shriberg, L. D., Tomblin, J. B., & McSweeney, J. L. (1999). Prevalence of speech delay in 6-year-old children and comorbidity with language impairment. *Journal of Speech, Language, and Hearing Research*, 42(6), 1461–1481.
- Tierney, A. L., & Nelson, C. A. (2009). Brain development and the role of experience in the early years. *Zero to Three*, 30(2), 9–13.
- Tremblay, K., & Kraus, N. (2002). Auditory training induces asymmetrical changes in cortical neural activity. *Journal of Speech, Language, and Hearing Research*, 45(3), 564–572.
- Tremblay, K., Kraus, N., McGee, T., Ponton, C., & Otis, B. (2001). Central auditory plasticity: Changes in the N1-P2 complex after speech-sound training. *Ear and Hearing*, 22(2), 79–90.
- Uwer, R., Albrecht, R., & von Suchodoletz, W. (2002). Automatic processing of tones and speech stimuli in children with specific language impairment. *Developmental Medicine and Child Neurology*, 44(8), 527–532.
- Volkmer, S., & Schulte-Körne, G. (2018). Cortical responses to tone and phoneme mismatch as a predictor of dyslexia? A systematic review. *Schizophrenia Research*, 191, 148–160. <https://doi.org/10.1016/j.schres.2017.07.010>
- White-Schwoch, T., & Kraus, N. (2013). Physiologic discrimination of stop consonants relates to phonological skills in pre-readers: A biomarker for subsequent reading ability? *Frontiers in Human Neuroscience*, 7, 899. <https://doi.org/10.3389/fnhum.2013.00899>
- White-Schwoch, T., Woodruff Carr, K., Thompson, E. C., Anderson, S., Nicol, T., Bradlow, A. R., ... Kraus, N. (2015). Auditory processing in noise: A preschool biomarker for literacy. *PLoS Biology*, 13(7), e1002196. <https://doi.org/10.1371/journal.pbio.1002196>

Winkler, I., Lehtokoski, A., Alku, P., Vainio, M., Czigler, I., Csépe, V., ... Näätänen, R. (1999).

Pre-attentive detection of vowel contrasts utilizes both phonetic and auditory memory representations. *Brain Research. Cognitive Brain Research*, 7(3), 357–369.

Woodruff Carr, K., White-Schwoch, T., Tierney, A. T., Strait, D. L., & Kraus, N. (2014). Beat synchronization predicts neural speech encoding and reading readiness in preschoolers.

Proceedings of the National Academy of Sciences of the United States of America, 111(40), 14559–14564. <https://doi.org/10.1073/pnas.1406219111>

PREPRINT

FIGURE CAPTIONS

Figure 1. ERP waveforms elicited by the treated sound (top) and /ba/ (bottom) syllables in the ERP study. The deviant waveforms represent the neural responses when the deviant syllable was presented within a stream of the opposite syllable standards. Subtracting the standard syllable response from the deviant syllable response resulted in the identity difference waves. Negative is plotted *up* in this figure.

Figure 2. Children who had earlier treated sound deviant P1/P2 peak latency measures pre-treatment had higher post-treatment production accuracy of their treated sound.

Table 1. Individual children's characteristics.

Participant Number	Treated Sound	Age (months) Pre-Treatment	Age (months) Post-Treatment	Pre-Treatment GFTA2 Standard Score	Pre-Treatment GFTA2 Percentile Score
1	ɹ	65	68	86	8
2	ʃ	44	47	55	2
3	l	47	51	50	1
4	ɹ	54	58	53	1
5	θ	51	54	50	1
6	θ	84	86	69	7
7	l	75	77	81	11
8	ʃ	48	50	81	16
9	ʃ	44	48	57	3
10	k	55	58	59	3
11	ɹ	58	61	62	4
12	ɹ	61	63	71	3
13	ɹ	65	67	56	2
14	ɹ	70	74	73	4
15	ʃ	69	71	<40	<1
16	l	48	50	77	9
17	ɹ	75	77	90	18
18	s	48	50	94	32
19	ʃ	48	53	42	<1
20	ɹ	66	70	64	4

- GFTA2 refers to the standard score on the Goldman-Fristoe Test of Articulation 2nd Edition (Goldman & Fristoe, 2000). Standard scores between 85 and 115 are considered to be in the normal range.
- GFTA% refers to the percentile score on the Goldman-Fristoe Test of Articulation 2nd Edition (Goldman & Fristoe, 2000). Percentile scores between 16 and 84 are considered to be in the normal range.

Table 2. Children’s pre- and post-treatment (Tx) speech production percentage accuracy scores, and the percentage of change from pre- to post-treatment, of their selected treated sound and all consonants (percent consonants correct, PCC).

Participant Number	Treated Sound	Pre-Tx Treated Sound Accuracy	Post-Tx Treated Sound Accuracy	Treated Sound Change	Pre-Tx PCC	Post-Tx PCC	PCC Change
1	ɹ	0.24	8.14	7.90	72.64	84.41	11.76
2	ʃ	0.00	2.94	2.94	24.06	30.20	6.14
3	l	3.00	5.59	2.59	30.20	35.81	5.61
4	ɹ	0.00	11.39	11.39	39.32	77.92	38.61
5	θ	0.00	8.46	8.46	35.81	39.32	3.51
6	θ	1.47	10.52	9.05	73.76	73.30	-0.46
7	l	12.10	65.00	52.90	84.77	89.77	5.00
8	ʃ	0.00	29.41	29.41	60.24	73.48	13.24
9	ʃ	0.00	5.41	5.41	35.80	41.63	5.83
10	k	19.51	31.67	12.16	59.03	67.50	8.47
11	ɹ	0.00	1.60	1.60	52.00	59.03	7.02
12	ɹ	13.68	21.61	7.93	79.97	84.28	4.31
13	ɹ	0.52	0.00	-0.52	53.23	65.61	12.38
14	ɹ	5.90	33.86	27.96	72.54	85.01	12.48
15	ʃ	1.96	1.96	0.00	42.20	41.23	-0.96
16	l	1.88	4.90	3.02	37.87	44.00	6.14
17	ɹ	1.45	11.33	9.88	75.25	78.12	2.87
18	s	0.00	5.39	5.39	65.50	71.56	6.06
19	ʃ	0.00	24.85	24.85	20.51	22.01	1.50
20	ɹ	0.92	12.91	12.00	62.98	79.63	16.65

Table 3. Consonant-vowel (CV) syllable formant frequencies (in hertz). Two frequency values are given for the formants of each syllable. The first value specifies the formant onset (On) frequency at the beginning of the syllable and the second represents the steady-state (vowel, V) frequency attained after the formant transition toward the center of the syllable. NA denotes formants that were not identifiable in the spectrogram.

	/ba/		/ɪa/		/la/		/tʃa/		/θa/		/sa/		/ka/	
	On	V	On	V	On	V	On	V	On	V	On	V	On	V
F1 (Hz)	486	818	591	993	362	824	NA	821	NA	794	NA	840	1069	802
F2 (Hz)	998	1158	1172	1041	883	1166	NA	1191	1792	1217	1539	1189	1656	1176
F3 (Hz)	2575	2731	1492	2658	2882	2782	2539	2655	2817	2764	2621	2748	2927	2641

Table 4. Mean amplitude MMN measurements in microvolts taken from three electrodes during the 250-350 ms post-syllable onset time window in the identity difference waves. All values are in μv . Values of the standard error of the mean are in parentheses.

MMN Mean Amplitudes					
	Fz	FCz	Cz	Overall	Syllable Overall
Pre: Treated	0.838 (.405)	1.188 (.624)	0.625 (.381)	0.884 (.324)	Treated
Post: Treated	0.811 (.479)	-0.280 (.426)	-0.693 (.359)	-0.054 (.273)	0.415 (.219)
Electrode Overall	0.825 (.337)	0.454 (.427)	-0.034 (.264)	--	--
Pre: /ba/	0.064 (.802)	-0.094 (.637)	-1.679 (.894)	-0.570 (.473)	/ba/
Post: /ba/	-0.859 (.866)	0.841 (.738)	1.976 (.962)	0.653 (.623)	0.041 (.374)
Electrode Overall	-0.397 (.578)	0.373 (.442)	0.148 (.688)	--	--

Table 5. A) Mean amplitude P1/P2 measurements in microvolts taken from three regions of interest (ROI) during the 100-200 ms post-syllable onset time window for the treated sound standard and deviant stimuli. B) Mean amplitude P1/P2 measurements in microvolts taken from three ROI during the 100-200 ms post-syllable onset time window for the /ba/ standard and deviant stimuli. All values are in μV . Values of the standard error of the mean are in parentheses.

A. Treated Sound P1/P2 Standard and Deviant Stimuli Mean Amplitudes

	Left	Midline	Right	Overall	Syllable Type Overall	Assessment Session Overall
Pre: Standard	1.316 (.228)	1.749 (.193)	1.394 (.384)	1.486 (.214)	Standard	Pre-treatment
Post: Standard	1.131 (.186)	1.516 (.238)	1.203 (.298)	1.283 (.177)	1.385 (.174)	1.566 (.225)
Pre: Deviant	1.174 (.462)	2.321 (.296)	1.444 (.356)	1.646 (.278)	Deviant	Post-treatment
Post: Deviant	1.896 (.491)	1.753 (.441)	1.417 (.346)	1.689 (.336)	1.668 (.246)	1.486 (.246)
ROI Overall	1.379 (.243)	1.835 (.226)	1.364 (.269)	--	--	--

B. /ba/ P1/P2 Standard and Deviant Stimuli Mean Amplitudes

	Left	Midline	Right	Overall	Syllable Type Overall	Assessment Session Overall
Pre: Standard	0.776 (.188)	1.130 (.236)	0.967 (.248)	0.958 (.172)	Standard	Pre-treatment
Post: Standard	1.110 (.164)	1.365 (.168)	1.101 (.252)	1.192 (.152)	1.075 (.137)	0.930 (.165)
Pre: Deviant	0.832 (.398)	1.028 (.281)	0.848 (.474)	0.903 (.233)	Deviant	Post-treatment
Post: Deviant	1.101 (.254)	1.178 (.381)	0.645 (.455)	0.974 (.216)	0.938 (.179)	1.083 (.154)
ROI Overall	0.955 (.185)	1.175 (.179)	0.890 (.276)	--	--	--

Table 6. A) Peak latency P1/P2 measurements in microvolts taken from three regions of interest (ROI) during the 100-200 ms post-syllable onset time window for the treated sound standard and deviant stimuli. B) Peak latency P1/P2 measurements in microvolts taken from three ROI during the 100-200 ms post-syllable onset time window for the /ba/ standard and deviant stimuli. All values are in milliseconds (ms). Values of the standard error of the mean are in parentheses.

A. Treated Sound P1/P2 Standard and Deviant Stimuli Peak latency

	Left	Midline	Right	Overall	Syllable Type Overall	Assessment Session Overall
Pre: Standard	144.7 (4.6)	151.1 (4.4)	151.1 (4.6)	149.0 (2.9)	Standard	Pre-treatment
Post: Standard	151.9 (4.6)	151.6 (5.1)	153.3 (4.0)	152.3 (3.7)	150.6 (2.9)	150.7 (2.3)
Pre: Deviant	150.4 (3.9)	152.1 (4.5)	154.6 (4.4)	152.4 (3.0)	Deviant	Post-treatment
Post: Deviant	154.9 (4.2)	158.1 (4.8)	151.7 (4.1)	154.9 (3.3)	153.6 (2.4)	153.6 (3.2)
ROI Overall	150.5 (3.1)	153.2 (2.8)	152.7 (2.5)	--	--	--

B. /ba/ P1/P2 Standard and Deviant Stimuli Peak latency

	Left	Midline	Right	Overall	Syllable Type Overall	Assessment Session Overall
Pre: Standard	126.0 (3.6)	130.2 (4.1)	130.3 (2.5)	128.8 (2.8)	Standard	Pre-treatment
Post: Standard	125.1 (1.6)	124.1 (2.7)	124.2 (2.1)	124.5 (1.7)	126.6 (2.0)	132.3 (2.4)
Pre: Deviant	130.2 (2.9)	137.2 (4.9)	139.8 (3.7)	135.7 (2.7)	Deviant	Post-treatment
Post: Deviant	133.3 (2.5)	130.7 (4.2)	124.5 (2.0)	129.5 (2.1)	132.6 (1.8)	126.9 (1.6)
ROI Overall	128.7 (1.8)	130.6 (2.7)	129.7 (1.6)	--	--	--

Treatment Sound Syllable

Ba

- Pre-Treatment Standard
- - - Pre-Treatment Deviant
- Post-Treatment Standard
- - - Post-Treatment Deviant

- - - Pre-Treatment Difference Wave
- Post-Treatment Difference Wave