

## Article

# Functional Brain Activation Differences in School-Age Children With Speech Sound Errors: Speech and Print Processing

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**Purpose:** To examine neural response to spoken and printed language in children with speech sound errors (SSE).

**Method:** Functional magnetic resonance imaging was used to compare processing of auditorily and visually presented words and pseudowords in 17 children with SSE, ages 8;6[years;months] through 10;10, with 17 matched controls.

**Results:** When processing spoken words and pseudowords, the SSE group showed less activation than typically speaking controls in left middle temporal gyrus. They also showed greater activation than controls in several cortical and subcortical regions (e.g., left superior temporal gyrus, globus pallidus, insula, fusiform, and bilateral parietal regions). In response to printed words and pseudowords, children with SSE had greater activation than

controls in regions including bilateral fusiform and anterior cingulate. Some differences were found in both speech and print processing that may be associated with children with SSE failing to show common patterns of task-induced deactivation and/or attentional resource allocation.

**Conclusion:** Compared with controls, children with SSE appear to rely more on several dorsal speech perception regions and less on ventral speech perception regions. When processing print, numerous regions were observed to be activated more for the SSE group than for controls.

**Key Words:** functional magnetic resonance imaging (fMRI), speech sound errors, speech perception, print processing

Oververt misarticulations (e.g., sound substitutions, omissions, and distortions) that persist into elementary school are associated with negative academic, social, and speech-language outcomes (Lewis & Freebairn, 1992; Nathan, Stackhouse, Goulrandis, & Snowling, 2004; Silverman & Paulus, 1989). In English (as well as many other languages), children with residual

speech sound errors (SSE) typically misarticulate liquids (/r, l/), lingual fricatives (/s, z, ʃ, ʒ, θ, ð/), and/or affricates /tʃ, dʒ/ (e.g., Shriberg, 1994). Some SSE may be associated with clear etiologies (e.g., structural or sensory impairment), but the majority of children with misarticulations that persist into mid-elementary school age exhibit these errors in the absence of a known cause. The cognitive and psycholinguistic processes associated with misarticulations have been investigated for decades, but the neurobiological bases have not been well explored (Peterson, McGrath, Smith, & Pennington, 2007). Therefore, in this article, we provide an initial description of the neural bases of language processing in school-age children with SSE of unknown origin.

Understanding how children with SSE differ in their processing of spoken language at the neural level can have important implications for understanding differences at the behavioral level. For example, there is long-standing behavioral evidence that children with SSE differ from control children in their recognition and processing of the acoustic-phonetic components of spoken words and pseudowords and exhibit poorer discrimination and recognition

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of speech sounds (Bird & Bishop, 1992; Cohen & Diehl, 1963; Madison & Fucci, 1971; Rvachew & Grawburg, 2006; Shuster, 1998). Cohen and Diehl (1963) reported that first through third grade children with articulation impairments made almost twice as many errors as children with typical speech development on an auditory discrimination task of English phonemes. Moreover, Shiller, Rvachew, and Brosseau-Lapr e (2010) recently summarized several decades of research that has compared speech perception abilities of children with articulation difficulties to children without; of 13 studies they reviewed, very large group differences were observed (effect sizes ranged from  $d = 1.35$  to  $d = 8.75$ ). As a complement to these behavioral studies, in the present research we explored neurobiological differences in speech perception.

Shriberg and colleagues (Shriberg, 1994; Shriberg et al., 2005) have proposed a construct they call *phonological attunement* to describe differences in children with persisting speech errors. They describe phonological attunement as the process of “tuning in and tuning up” (Shriberg et al., 2005, p. 838) to phonetic information, presumably at the perceptual level. Although not fully specified in their writing, one can infer that attunement is driven by neurobiological differences in response to spoken language exhibited by children during early speech development.

In addition to speech perception differences, articulatory problems that persist into school age have been associated with difficulties in reading and spelling as well as with the phonological bases of these skills (Lewis & Freebairn, 1992; Peterson, Pennington, Shriberg, & Boada, 2009; Preston & Edwards, 2007). The nature of the specific mechanisms that underlie these behavioral associations remains unclear. From a cognitive–linguistic perspective, several studies have suggested that children with SSE have poorly specified phonological representations, which may be associated with poor speech perception, speech production, and phonological awareness (e.g., Preston & Edwards, 2007; Rvachew & Grawburg, 2006). In this study, we explored neurobiological differences in processing both printed language and spoken language in children with SSE.

## Neural Bases of Speech Perception

A number of neural regions have been implicated in speech perception. The classic Wernicke’s area in the posterior superior temporal gyrus (STG) is undoubtedly important in speech perception, but recent neuroimaging studies have identified several other regions that are engaged when humans process spoken information. Binder et al. (1997), for example, reported that processing of single spoken words in adults activated a widely distributed cortical network in the left hemisphere that included the superior, middle, and inferior temporal gyri; fusiform and parahippocampal gyri; inferior, middle, and superior

frontal gyri; anterior cingulate and angular gyrus; posterior cingulate/precuneus; and right posterior cerebellum. Similar networks have been observed in studies using phonologically based tasks; for example, in a rhyme judgment task, Booth, Wood, Lu, Houk, and Bitan (2007) observed correlated activations in left inferior/middle frontal gyrus, fusiform, superior/middle temporal gyrus, and putamen. It is clear that complex networks are involved in the perception and processing of phonological information.

Hickok, Poeppel, and their colleagues (Hickok & Poeppel, 2004, 2007; Poeppel, Idsardi, & van Wassenhove, 2008) outlined a network-based model of speech perception that provides a useful template for the present study. In their *dual-route model*, both a dorsal and a ventral perceptual system are implicated in processing of speech; specifically, acoustic–phonetic speech input is believed to be initially coded in the bilateral STG (e.g., Binder et al., 1997; Guenther, Nieto-Castanon, Ghosh, & Tourville, 2004; Whalen et al., 2006). After this initial activation, dorsal and ventral pathways are engaged. The dorsal pathways are composed of the auditory-to-motor mappings in the temporal–parietal juncture of the left Sylvian fissure and the left inferior frontal/premotor regions (Callan, Jones, Callan, & Akahane-Yamada, 2004; Hickok & Poeppel, 2004). Ventral pathways involve a more direct/more efficient sound-to-meaning interface in the left middle and inferior temporal/fusiform gyri (e.g., Majerus et al., 2005; Szaflarski et al., 2006). In general, dorsal paths are engaged to a greater extent when one is processing novel or phonologically complex speech, whereas ventral circuits are engaged more if the input is simple or more familiar (Hickok & Poeppel, 2004, 2007).

It is notable that many of the functional regions identified in speech perception studies have also been implicated as regions that are important in speech production, including the left inferior frontal gyrus (IFG), precentral gyrus, STG, inferior parietal cortex, and cerebellum (e.g., Guenther, 2006). Functional magnetic resonance imaging (fMRI) data provide evidence of partial overlap between perception and production of speech, with the left posterior STG serving as an important region that is common in both processes (Buchsbaum, Hickok, & Humphries, 2001). These findings are particularly important for understanding typical and impaired speech because they suggest that perception-based activities engage at least some of the same brain regions as those activated during speech production. In the present study, we sought to examine regions that are engaged differently in children with SSE when listening to spoken words and pseudowords.

## Neural Differences in Individuals With Developmental SSE

Few extant studies have examined functional neural organization in persons who have SSE as their primary

communication deficit. In one early study, Lou, Henriksen, and Bruhn (1984) performed computerized axial tomography scans with 13 children ages 6 through 15 years with deficits that included SSE (described as having dyspraxia or phonological–syntactic dysphasia) and attention difficulties. Compared with nine age-matched controls with typical speech and attention scores, three children described as having “dyspraxia” had reduced blood flow in their left anterior perisylvian regions, and four children with “phonological–syntactic” disorder had reduced blood flow in both the anterior and posterior perisylvian regions during rest. During speaking, several of the children with SSE failed to show the typical pattern of increased blood flow to Broca’s area in the left IFG. Although provocative, this study is difficult to interpret because of the heterogeneity of participants and concomitant language deficits.

One of the most comprehensive genetic and neuroimaging studies of adults with congenital speech and language impairment comes from one highly unique family, known as the *KE family*. This family includes several members with a genetic mutation resulting in severe deficits in language production and oral dyspraxia (Alcock, Passingham, Watkins, & Vargha-Khadem, 2000; Belton, Salmond, Watkins, Vargha-Khadem, & Gadian, 2003; Liegeois et al., 2003; Watkins et al., 2002). In one fMRI study in this series, Liegeois et al. (2003) reported that, relative to five unaffected family members, affected adults from the KE family showed underactivation of Broca’s area during verb generation tasks. In a companion structural study, affected KE members had reduced gray matter density (bilaterally) in the caudate nucleus, cerebellum, and IFG (Belton et al., 2003).

Finally, in a recent fMRI study, Tkach et al. (2011) examined six adolescents (ages 12–18 years) with histories of speech disorders (including five whose speech errors had resolved) using a pseudoword repetition task. Compared with controls with typical speech and language, the participants with histories of speech difficulties showed less activation in right IFG and middle temporal gyrus. The clinical group also showed greater activation in the bilateral premotor cortex as well as in the left supplementary motor area, inferior frontal cortex, anterior cingulate, postcentral gyrus, angular gyrus, inferior parietal lobule, and supramarginal gyrus. Because this task involves both perception and production, further examination of regions associated with differences in perception is warranted.

## Speech and Literacy

Children with SSE tend to perform more poorly than typically speaking peers on tasks assessing phonological awareness, reading, and spelling; these differences may exist even in the absence of co-occurring language

impairments, and they can persist into adolescence and adulthood (Bird, Bishop, & Freeman, 1995; Larivee & Catts, 1999; Lewis & Freebairn, 1992; Lewis, Freebairn, & Taylor, 2002; Preston, 2010; Preston & Edwards, 2007). Thus, although co-occurring language impairment is often associated with poorer literacy skills than speech production deficits alone (e.g., Lewis et al., 2004; Nathan, Stackhouse, Goulandris, & Snowling, 2004), several studies have found weaknesses in phonological awareness and early reading and spelling in children with isolated articulation problems (Bird et al., 1995; Leitao, Hogben, & Fletcher, 1997; Lewis et al., 2002; Raitano, Pennington, Tunick, Boada, & Shriberg, 2004). It is interesting that the neural circuitry involved in processing spoken and printed language shows some overlap, including left-hemisphere temporal sites, the fusiform gyrus, and the IFG (Booth et al., 2001; Constable et al., 2004; Shankweiler et al., 2008). The inferior frontal subsystem, centered on Broca’s area in the posterior IFG, is believed to be sensitive to the phonological structure of printed language (Frost et al., 2009) and is important for the perception and production of phonetic information (Callan et al., 2004; Guenther, 2006). Moreover, although the sensory input modalities differ, behavioral data indicate that the processing of printed language engages the articulatory/phonological system (e.g., Lukatela, Eaton, Sabadini, & Turvey, 2004). Thus, a secondary goal of this study was to examine how children with SSE differ in their processing of printed language.

## Aims of This Study

In the present study, we examined functional brain responses to speech and to print in children with SSE of unknown origin whose speech errors had not yet resolved by at least age 8;6 [years;months]. Characterizing neural differences associated with the processing of both spoken and printed language may provide important and novel information about the biological mechanisms of school-age SSE, helping us to better specify how phonetic learning might go awry. We hypothesized that if speech errors arise primarily from dysfunction of speech perception circuits (which partially overlap with production circuits), then measures of functional activation will reveal differences between children with SSE and typically speaking children at cortical and subcortical speech perception circuits.

Compared with previous neuroimaging studies of children with SSE, the present study has several advantages. It includes a larger and more homogeneous sample than reported in any prior studies of SSE, and participants with co-occurring language problems were excluded. Participants were obtained from a community-based sample, which helps to alleviate ascertainment bias. Finally, when compared with previous studies, the control

and SSE groups in the present study were more closely matched on relevant variables.

Our specific research question was the following: Compared with typically speaking peers, will children with SSE demonstrate differences in neural activation in regions traditionally associated with phonological processing and speech perception when processing spoken and printed language?

## Method

### *Participants and Participant Classification Procedures*

The Haskins/Yale Study of Reading Development ([www.yalereadingcenter.com](http://www.yalereadingcenter.com)) is an ongoing prospective study examining the neural and genetic bases of spoken and printed language processing in children with a wide variety of reading abilities and cognitive–linguistic profiles. Potential participants are recruited on a rolling basis by means of public notices and contacts with local schools and clinics. Participants in the Haskins/Yale study are tested individually in a quiet sound-treated room, and sessions are recorded with a SONY ICD-P620 digital recorder or a JVC TD-W354 cassette recorder. Children complete a wide range of standardized cognitive, academic, and language tests, including the Peabody Picture Vocabulary Test—III (Dunn & Dunn, 1997), the Woodcock–Johnson Tests of Achievement—III (Woodcock, McGrew, & Mather, 2001), the Comprehensive Test of Phonological Processing (Wagner, Torgesen, & Rashotte, 1999), the Test of Word Reading Efficiency (Torgesen, Wagner, & Rashotte, 1999), and the Wechsler Abbreviated Scale of Intelligence (Wechsler, 1999). In addition, children participate in an fMRI study that asks them to make decisions about word and wordlike stimuli that are presented auditorily (auditory decision making) and visually (print decision making).

Identification of children with SSE and matched controls from this larger sample involved a multistep process. Three initial selection criteria were imposed: (a) All participants had to be monolingual English speakers with no frank cognitive, physical, or sensory disabilities that might affect speech production; (b) fMRI data had to be available and of sufficient quality for review (i.e., data had to be equivalent to at least six functional runs); and (c) children had to be between 8;6 and 11;0 at the time of fMRI. Applying these criteria resulted in a pool of 76 potential participants, who were subsequently divided into those with and without SSE through an analysis of approximately 5 min of audiorecorded connected speech (the Vocabulary subtest of the Wechsler Abbreviated Scale of Intelligence, although other speech samples were used if this recording was of poor quality).

The audio recordings of all 76 potential participants were analyzed by a trained listener to identify the presence of perceptually identifiable articulation errors. The listeners first determined whether the recording was of adequate quality to evaluate the phonetic accuracy of the child's speech, resulting in exclusion of data from four participants. The remaining 72 tapes were subsequently classified as follows: no misarticulations (potential control group,  $n = 51$ ), questionable speech (infrequent errors, excluded from the study,  $n = 4$ ), or persistent misarticulations (SSE group,  $n = 17$ ). These initial classifications were confirmed by a second listener (either the first or second author) who was blind to the initial classification decision. Agreement between the first and second listeners for classification of SSE was 100%. As a final step, the potential control group was reexamined to exclude children for whom there was a positive parent report that the child had received speech-language therapy for any reason ( $n = 14$ ) and to balance gender ( $n = 19$  females). This adjustment resulted in a final study sample of 17 control participants (14 males; three females) and 17 SSE participants (14 males; three females) between the ages of 8;6 and 10;10.

We used the Percentage of Consonants Correct—Late 8 metric (Shriberg, Austin, Lewis, McSweeney, & Wilson, 1997) to quantify accuracy of the late-developing phonemes (/r, l, s, z, ð, θ, ʃ, ʒ/) from the speech samples. All samples included more than 300 consonant attempts and at least 120 Late 8 consonant attempts. The SSE group achieved Percentage of Consonants Correct—Late 8 scores that ranged from 42% to 88%, whereas all control children achieved scores that ranged from 95% to 100%. All children in the SSE group displayed errors on the /s/ and/or the /r/ phonemes and, on occasion, other sounds. Sounds that were in error on greater than 30% of a child's attempts included /r/ (10 children), /l/ (two children), /s/ (nine children), /z/ (eight children), /θ/ (three children), /ʃ/ (four children), /ʒ/ (three children), and /dʒ/ (one child). Error patterns reflected those typically seen in children with residual articulation impairments (e.g., gliding or derhoticization of /r/, dentalization or lateralization of /s, z/, depalatalization or lateralization of palatals).

Of the 17 children with SSE, 16 parents provided information about the child's history of speech-language therapy. Eight of these children were reported to have received articulation therapy at some point in the past or present, and two of these had targeted language goals (vocabulary, syntax) at some point. One was reported to have been diagnosed with verbal dyspraxia as a toddler. When asked to qualitatively describe early communication, five parents of children with SSE reported that the child had spoken his or her first words "late" (compared with four controls), and six were reported as having begun to form sentences "late" (compared with five

controls). Ten parents of children with SSE reported that reading and spelling were difficult for their child (compared with seven controls). Two children in each group were reported to have been diagnosed with attention-deficit disorder. None had a reported history of head trauma.

Group comparison data are presented in Table 1. Effect sizes of  $d < 0.35$  were observed for age, receptive vocabulary, oral language, and verbal IQ. Larger effect sizes were observed on some phonological awareness and reading measures. This finding of somewhat lower reading and phonological awareness performance in children with SSE is consistent with previous findings (e.g., Bird et al., 1995; Preston, 2010).

## fMRI Procedures

We acquired fMRI data using a 1.5T Siemens scanner on a separate day from the behavioral testing. Participants were provided with a neck support, foam padding, and a restraining band within the headcoil to stabilize the head. Prior to scanning, participants engaged in a practice session to become familiar with the paradigm and procedures.

The fMRI task assessed functional activation in response to words and pseudowords presented visually or auditorily (see Frost et al., 2009, and Preston et al., 2010, for additional examples of this paradigm). Trials were

presented in a randomized fast event-related design with trial durations of 4–7 s (to provide sufficient jitter to deconvolve overlapping hemodynamic responses) with occasional longer null trials (to provide better baseline estimates). All conditions were represented in each run, and up to 10 runs were presented per participant. Functional runs began with a picture cue presented in the upper central portion of the display; this picture remained on the screen during presentation of spoken and printed monosyllabic word and pseudoword targets. Each picture remained on the screen during presentation of seven or eight targets (45–55 s), after which it was replaced by another picture. For example, while a picture of a tent was on the screen, participants either heard or saw the correct word (*tent*, a match), or they heard or saw a closely related monosyllabic real word (e.g., *test*, a mismatch) or pseudoword that began with the same phoneme (e.g., *tert*, a mismatch). Participants indicated, via button press, whether targets matched or mismatched the picture cue. Print stimuli were displayed in a box beneath the picture cue for 1,000 ms, and speech stimuli were presented through magnetic resonance-compatible headphones ( $M$  duration of audio stimuli = 543 ms,  $SD = 115.3$ ).

All stimuli comprised two to five phonemes. Printed stimuli were three to five letters. There was no significant difference between auditory words and visual words,

**Table 1.** Descriptive and inferential statistics on behavioral and functional magnetic resonance imaging (fMRI) tasks for 17 typically speaking controls and 17 children with speech sound errors (SSE).

Variable	Control group $M$ ( $SD$ )	SSE group $M$ ( $SD$ )	$t$	$p$	Effect size ( $d$ )
Age (years;months)	9;10 (0;7)	9;7 (0;9)	1.05 <sup>a</sup>	.303	0.35
PPVT-III	114 (11)	113 (14)	0.41 <sup>a</sup>	.684	0.14
WJ Oral Expression	117 (14)	115 (15)	0.27 <sup>b</sup>	.616	0.18
WJ Listening Comprehension	116 (13)	113 (11)	0.75 <sup>b</sup>	.56	0.21
WASI Verbal IQ	113 (12)	113 (14)	0.10 <sup>a</sup>	.911	0.04
WASI Performance IQ	103 (17)	111 (17)	1.07 <sup>a</sup>	.134	0.52
TOWRE Phonemic Decoding	104 (15)	96 (11)	1.86 <sup>a</sup>	.073	0.64
TOWRE Sight Words	104 (14)	95 (12)	1.8 <sup>a</sup>	.082	0.62
WJ Word Attack	105 (11)	98 (9)	2.0 (31)	.054	0.70
WJ Letter-Word ID	105 (14)	99 (14)	1.4 (31)	.176	0.48
WJ Spelling	104 (17)	95 (18)	1.4 (31)	.158	0.50
CTOPP Elision	11.4 (2.3)	8.4 (2.6)	3.5 <sup>a</sup>	.001	1.20
CTOPP Blending Words	11.4 (2.8)	9.6 (3.0)	1.85 <sup>a</sup>	.074	0.63

*Note.* All measures except age are age-normed standard scores ( $M = 100$ ,  $SD = 15$ ) except for the CTOPP, for which we report scaled scores that have a  $M$  of 10 and an  $SD$  of 3. One participant from the control group did not complete the Woodcock-Johnson Tests of Achievement (WJ) because the parent reported that very similar tests had just been administered through the school (hence,  $df = 31$ ); although the school had not administered the WJ, similar standardized tests revealed language and reading scores within the average range (commensurate with group means presented here). PPVT-III = Peabody Picture Vocabulary Test—III; WASI = Wechsler Abbreviated Scales of Intelligence; TOWRE = Test of Word Reading Efficiency; CTOPP = Comprehensive Test of Phonological Processing.

<sup>a</sup> $df = 32$ . <sup>b</sup> $df = 31$ .

or between auditory pseudowords and visual pseudowords, in phonotactic probabilities (based on phonotactic probabilities calculated from procedures described by Vitevitch & Luce, 2004, all  $ps > .22$  when comparing the phoneme and biphoneme frequencies across conditions).

In 80% of the trials, the stimulus did not match the picture cue—that is, they were auditory or visual mismatches, and these were the trials of interest. The rare match trials were coded as separate conditions and were excluded from main analyses. The task was designed to elicit a high degree of accuracy, and, as can be seen in Table 2, both groups made few errors in either condition (accuracy rates were 89% or better for both groups in both auditory and visual conditions). Moreover, the groups did not differ significantly in reaction time to either speech or print. Thus, differences in activation should not be driven by differences in task performance.

Twenty axial-oblique anatomical images were acquired, parallel to the intercommissural line based on sagittal localizer images. At these same 20 slice locations, activation images were acquired through the use of single-shot, gradient echo, echo planar acquisitions (flip angle 80°; echo time [TE] 50 ms; repetition time [TR] 2000 ms; field of view [FOV] 20 × 20 cm; 6-mm slice thickness, no gap; 64 × 64 × 1 number of excitations [NEX]; voxel resolution 3.125 × 3.125 × 6 mm). High-resolution anatomical images were collected for 3D reconstruction. Images were sinc-interpolated to correct for slice acquisition time, motion corrected with SPM-99 (Friston et al., 1995), and spatially smoothed with a 4.69-mm full width at half maximum (FWHM) Gaussian filter. Images were excluded if they exceeded a tolerance of 2 mm displacement or 2° rotation from the first image in the functional series, or if they exceeded an image-to-image change of 1 mm displacement or 1° rotation. We used regression-based estimation for the hemodynamic response at each voxel and for each condition, without prior specification of a reference function (Miezin, Maccotta, Ollinger, Petersen, & Buckner, 2000). These parameters estimated the mean response for each condition from -3 s to +15 s relative to stimulus onset, and individual activation maps were created to estimate the

mean difference between a baseline (0–3 s before onset) and an activation period (3–8 s postonset). Before conducting the between-subjects analysis, we transformed their data into 2-mm isotropic standardized reference space as defined by the Montreal Neurological Institute by mapping the high-resolution anatomic to the “Colin” brain using linear and nonlinear registration parameters obtained with the BioImage Suite (see www.bioimagesuite.org; Papademetris, Jackowski, Schultz, Staib, & Duncan, 2003).

We compared the two groups in four conditions in a repeated measures analysis of variance: auditory words and pseudowords, and printed words and pseudowords. We used planned contrasts from the analysis of variance to compare the groups for the main effect of auditory and print conditions (collapsed across words and pseudowords). We tested the Group × Lexicality (real words vs. pseudowords) interactions in both auditory and printed conditions using planned contrasts. A statistical threshold of  $p < .01$  (false discovery rate corrected; Genovese, Lazar, & Nichols, 2002), with a minimum cluster criteria of 10 contiguous voxels, was used to identify regions of significant differences in the perception of auditory and printed stimuli.

## Results

### Differences in Auditory Processing of Speech

In several distributed (but potentially networked) regions, the mean activations for the SSE and control groups were found to differ significantly when participants were engaged in a task that required them to identify auditory tokens that did not match a picture cue. These differences involved both significantly greater activation by the SSE group relative to controls in some regions (SSE > control) and significantly less activation by the SSE group relative to controls in other regions (SSE < control), described in more detail below. In Table 3, we present these regions of significant difference and associated  $p$  values.

**Table 2.** Functional magnetic resonance imaging (fMRI) task performance for 17 typically speaking controls and 17 children with SSE.

Variable	Control group M (SD)	SSE group M (SD)	$t(32)$	$p$
A-prime	0.94 (0.04)	0.93 (0.05)	0.31	.759
Speech accuracy (% correct)	0.90 (0.06)	0.89 (0.06)	0.59	.561
Print accuracy (% correct)	0.92 (0.06)	0.90 (0.06)	0.73	.470
Reaction time: Speech (ms)	1,485 (249)	1,503 (198)	0.24	.815
Reaction time: Print (ms)	1,436 (283)	1,457 (209)	0.24	.812

**Table 3.** Regions showing differences between the typically speaking control group and the SSE group in response to spoken words and pseudowords.

Difference	Region	Volume (mm <sup>3</sup> )	p	MNI coordinates		
				x	y	z
SSE > Control	Bilateral inferior/superior parietal lobule	18,264	< .0001	-14	-64	66
	Precuneus	11,936	< .0001	-18	-50	50
	R supramarginal and postcentral gyrus	4,416	.0003	54	-34	40
	L fusiform gyrus	3,936	.0001	-51	-60	-17
	L superior temporal gyrus	3,536	< .0001	-66	-28	12
	Posterior cingulate	3,432	.0009	0	-68	9
	R middle frontal gyrus	2,824	.0001	32	54	28
	Cuneus	2,784	< .0001	-2	-90	16
	L globus pallidus	2,616	.0002	-10	6	-8
	R postcentral gyrus	2,248	.0001	30	-26	38
	R lingual gyrus	2,200	.0005	30	-78	-16
	Anterior cingulate	1,200	.0005	18	10	36
	R superior temporal gyrus	832	.0004	51	-36	10
	L cerebellum	736	.0004	-38	-52	-44
	R temporal pole	656	.001	42	14	-40
	L middle frontal gyrus	568	.0021	-44	38	28
	R superior frontal gyrus	472	.0004	10	4	72
	L middle occipital gyrus	448	.0001	-46	-80	16
	L insula	424	.002	-34	10	6
	R precentral gyrus	392	.0031	54	-2	24
SSE < Control	R orbital gyrii	1,944	.0001	14	40	-22
	L temporal pole	1,160	< .0001	-42	10	-18
	L orbital gyrii	608	.0005	-16	38	-22
	R inferior temporal gyrus	408	.0002	62	-36	-24
	L middle temporal gyrus	232	.0025	-56	-30	-4

Note. Only regions greater than 200 mm<sup>3</sup> are reported. MNI = Montreal Neurological Institute.

Children with SSE exhibited greater activation than controls during the processing of auditory input in several regions, including left STG, left insula, and fusiform as well as right supramarginal and postcentral gyrus. Several of these regions are associated with speech-motor processing and production (e.g., STG, insula). Figure 1 shows axial slices of selected regions in which the groups differed (where blue/purple represents greater activation for the SSE group). Figure 2 illustrates the group mean activation patterns for selected regions (left STG, insula); positive values represent a group mean increase in the blood oxygen level dependent (BOLD) signal following the stimulus, whereas negative values represent a group mean decrease in BOLD. The specific pattern was for children with SSE to activate some of these dorsal regions and for the control group to show little or no activation when performing the same task.

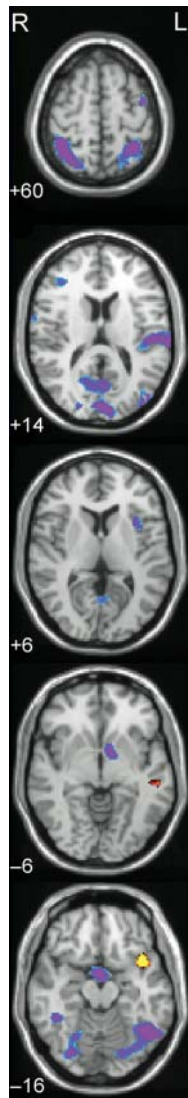
The SSE group was also observed to have significantly more activation than controls in several other regions in which the control group displayed a tendency to deactivate (reduce BOLD) in response to speech. These regions included the bilateral superior and inferior parietal lobule,

the left globus pallidus (see Figure 1), and the anterior cingulate and cuneus. However, the SSE group tended to activate these areas during the same task (see Figure 2). Stated differently, these results suggest a pattern of task-induced deactivation in these regions within the control group when processing speech, which failed to occur in children with SSE.

Table 3 also reveals a small number of regions that were underactivated in the SSE group relative to the control group. These underactivated regions in the SSE group (shown as red/yellow in Figure 1) included the left middle temporal gyrus (see the Figure 2 bar plot), left temporal pole, and right inferior temporal gyrus. As previously mentioned, some of these regions play an important part of the ventral (lexical) speech processing system.

Finally, we explored a Group  $\times$  Lexicality interaction to determine whether the SSE and control groups differed in their responses to spoken material as a function of stimulus type (words vs. pseudowords). At  $p < .01$ , a significant interaction was found for only two regions: (a) the left IFG/middle frontal gyrus and (b) the left anterior fusiform

**Figure 1.** Axial slices of selected regions of interest in which the groups differed in the processing of auditory words and pseudowords. Activations are significant at  $< .01$ , false discovery rate (FDR) corrected. Images are presented in radiological format, with the right hemisphere displayed on the left side of the image. Numerals represent z-axis mm values in Montreal Neurological Institute (MNI) space. Blue/purple represents greater activation for the SSE group; yellow/red represents greater activation for the control group. From top to bottom: bilateral superior/inferior parietal lobule and left precentral gyrus (+60); left superior temporal gyrus and cuneus (+14); left insula (+6); left globus pallidus and left middle temporal gyrus (-6); bilateral fusiform, right lingual gyrus, left temporal pole (-16).



gyrus/occipito-temporal sulcus. As can be seen in the right-most bar plots in Figure 2, the control group tended to engage these regions only during the auditory presentation of pseudowords (but not real words), whereas the SSE group displayed the opposite pattern, with activation in response to real words but not to pseudowords.

## Differences in Print Processing

In the print condition, no Group  $\times$  Lexicality interactions were significant; consequently, group comparisons for print processing were collapsed across words and pseudowords. These group differences are reported in Table 4, and selected regions are displayed in Figure 3 (axial slices of selected regions) and Figure 4 (patterns of response activation). Children with SSE displayed significantly greater activation than controls ( $p < .01$ , shown as blue/purple in Figure 3) in response to printed stimuli mismatches in several regions, including left IFG, left globus pallidus, left superior temporal sulcus/middle temporal gyrus, right STG/superior temporal sulcus, and bilateral fusiform gyrus. In regions such as anterior cingulate and precuneus, deactivation was found in the control group, whereas no such deactivation occurred in the SSE group.

## Discussion

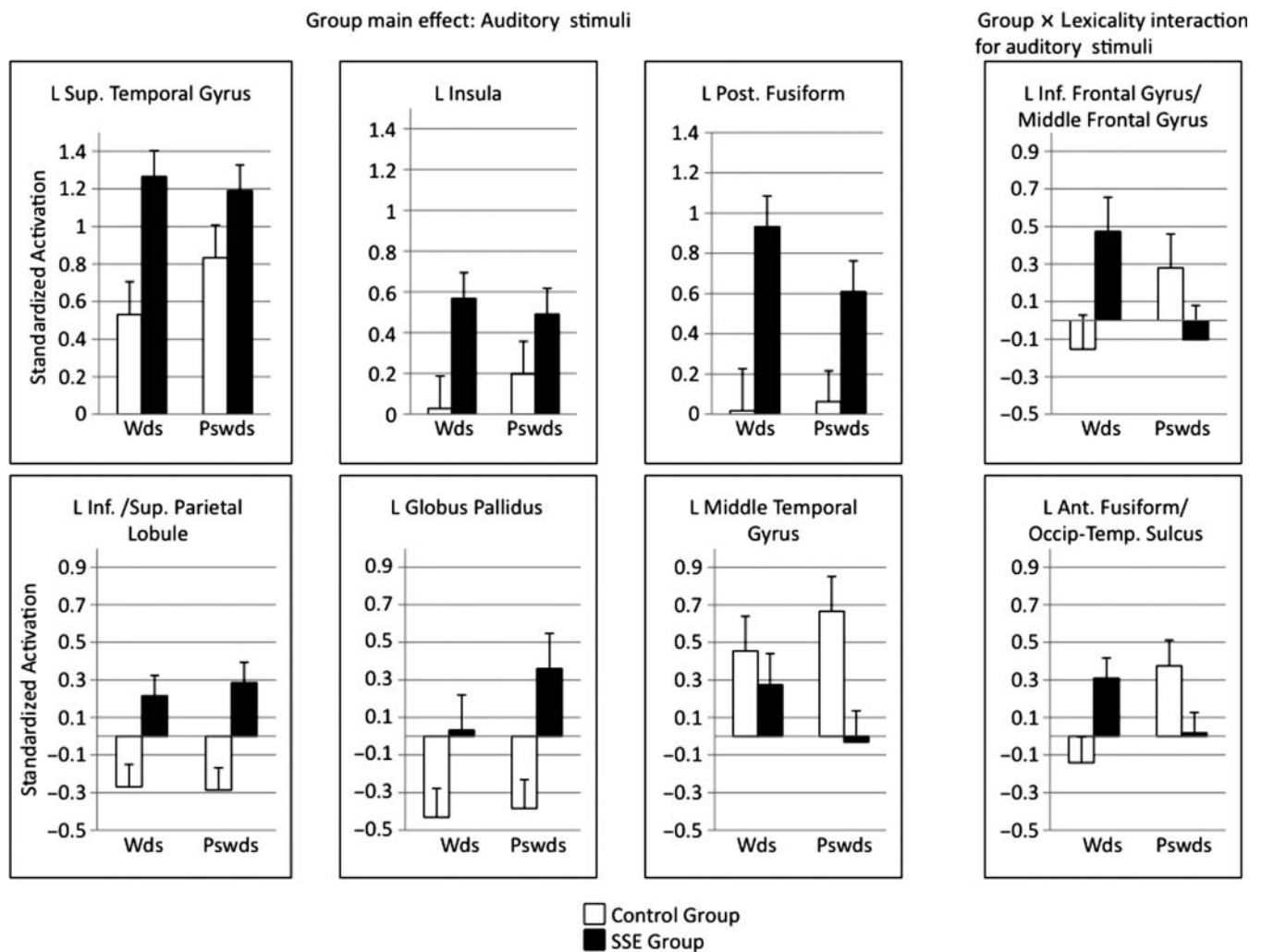
The present study provides an initial account of how children with SSE and children with normal-range language skills differ from well-matched children with typical speech articulation in their neural processing of spoken and printed language in tasks not involving overt pronunciation. The fMRI task required children to identify (by a buttonpress) whether a spoken or printed word or nonword stimulus matched a picture target. A number of regions in both cortices and in subcortical areas were identified that met our statistical threshold for distinguishing the two groups during this task. Taken together, these results provide a neurobiological complement to prior behavioral studies that have revealed differences in speech perception and reading in school-age children with SSE (Bird et al., 1995; Cohen & Diehl, 1963; Larivee & Catts, 1999).

## Differences in Speech Processing

To provide a framework for the presentation and interpretation of our complex findings, we refer in part to Hickok and Poeppel's (2004, 2007) dual-route model of single-word processing (previously described). Although it does not explain all of the findings, we use it as an organizational heuristic. For example, greater engagement for the SSE group was observed in several potentially networked dorsal regions that Hickok, Poeppel, and colleagues have described as important for the acquisition of new auditory-motor representations and/or the processing of novel, complex, or low-frequency speech input (Hickok & Poeppel, 2004, 2007; Poeppel et al., 2008). In addition, less activation was observed for the SSE group relative to controls in some ventral regions that may participate in auditory-lexical mapping, such as the left



**Figure 2.** Bar plots of blood oxygen level dependent response to auditorily presented words (Wds) and pseudowords (Pswds) in selected regions of interest and in regions in which groups differed by lexicality. Error bars represent 1 SE. Sup. = superior; Post. = posterior; Inf. = inferior; Ant. = anterior; Occip.-Temp. = occipital-temporal.



middle temporal gyrus (although the opposite pattern was observed in the fusiform gyrus). Beyond this model, our data also suggest that children with SSE show increased attention and/or failure to demonstrate the typical patterns of task-induced deactivation when presented with linguistic input. Note that these components need not be viewed as functioning independently.

*Dorsal articulatory-phonetic perception regions.* Relative to control participants, children with SSE displayed a pattern of greater engagement of the left STG networks when processing spoken speech. These networks may be recruited when speech-processing demands are high (e.g., when articulatory rehearsal is needed; see Hickok & Poeppel, 2004). The region in left STG is close (although slightly posterior) to the sensorimotor interface in the posterior temporal-parietal junction described by Hickok

and colleagues (Hickok, Okada, & Serences, 2009; Hickok & Poeppel, 2007). We also observed greater engagement by children with SSE of right supramarginal and postcentral gyrus, which has been observed to be related to perception of acoustic-phonetic information (Caplan, Gow, & Makris, 1995; Guenther et al., 2004). The left insula was also engaged to a greater extent in children with SSE than in controls. The left insula is known to be important for speech production as well as for the processing of phonological information (Hillis et al., 2004; Wise, Greene, Buchel, & Scott, 1999).

One possible explanation for these findings may be that the children with SSE tended to process most spoken forms as unfamiliar or challenging phonological forms, thus requiring more recruitment of dorsal articulatory-motor regions. In these children, mapping from auditory

**Table 4.** Regions showing differences between typically speaking control group and SSE group in response to printed words and pseudowords.

Difference	Region	Volume (mm <sup>3</sup> )	p	MNI coordinates		
				x	y	z
SSE > Control	Precuneus	25736	< .0001	10	-58	54
	R superior temporal gyrus/superior temporal sulcus	14,872	< .0001	62	-30	4
	Anterior cingulate	11,144	< .0001	6	26	26
	L fusiform gyrus	10,064	< .0001	-38	-52	-13
	Posterior cingulate	9,808	.0001	4	-62	20
	L middle/superior frontal gyrii	7,624	.0001	20	60	19
	L superior temporal sulcus/middle temporal gyrus	4,344	.0002	-42	-66	22
	L middle/superior frontal gyrii	2,816	.0002	-28	40	46
	L superior parietal lobule	2,624	.0001	-24	-77	46
	R fusiform gyrus	2,560	< .0001	46	-40	-14
	R middle temporal gyrus	2,064	< .0001	54	-22	-10
	L globus pallidus	1,576	.0009	-10	-4	-1
	L superior frontal gyrus	1,384	.0001	4	2	58
	R anterior inferior parietal sulcus	1,320	.0005	44	-24	28
	R lingual gyrus	944	.0006	14	-76	-8
	L postcentral gyrus	848	.0007	-46	-14	24
	L inferior frontal gyrus	808	.0004	-36	30	-5
	R precentral gyrus	744	.001	60	4	10
	R middle frontal gyrus	744	.0005	46	12	38
	L middle frontal gyrus	688	.0002	-42	46	6
R parahippocampal gyrus	408	.0002	22	-20	-24	
R superior temporal gyrus	320	.0003	68	0	0	
SSE < Control	L superior occipital gyrus	848	< .0001	-14	104	4
	L cerebellum	312	.0002	-52	-62	-48

Note. Only regions greater than 200 mm<sup>3</sup> are reported.

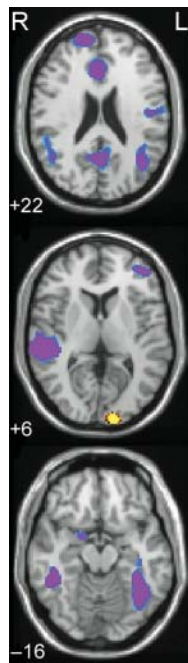
input to a phonological representation may be weak. Alternatively, it is possible that the children with SSE were relying on these regions to a greater extent than control children to compensate for ventral systems that are developing differently. Both explanations, which are not mutually exclusive, suggest that the SSE group may have auditory perceptual systems that rely on articulatory/motor coding (with or without actual subvocalization) when confronted with tasks that require phonological processing. This hypothesis is supported by studies of individuals learning the phonetic contrasts of new languages, a task that is likely to tax the dorsal system. In one study, for example, Callan et al. (2004) found that several of the regions identified in the present study (i.e., insula, STG) were also highly activated in second-language learners who were attempting to master new phonetic productions.

A Group  $\times$  Lexicality interaction was observed in the left inferior frontal/middle frontal gyrus; children with SSE engaged these regions when processing real words but not pseudowords, whereas the opposite pattern was observed in the control group (see Figure 2). The responses from the control group are consistent with the notion that IFG activation is observed primarily when

the phonological demands of the task are high and/or novel. Children with SSE, on the other hand, demonstrated heightened activation of this region to process real words, suggesting that, for them, even real words require more articulatory-phonetic encoding. Thus, when presented with real words, the response pattern in the IFG may reflect evidence that the phonological systems of children with SSE do not deal efficiently with the phonological properties of spoken lexical information (as supported by differences in the ventral system). Moreover, in response to pseudowords, children with SSE did not engage IFG, but control children did (see Figure 2). These perceptual findings may support data from Tkach et al. (2011), who reported greater engagement of left IFG by typically speaking adolescents relative to children with SSE when repeating pseudowords. Therefore, the IFG may not be properly recruited by individuals with SSE when processing novel phonetic forms.

*Ventral perception circuit.* The left middle temporal and inferior temporal gyri were regions where children with SSE showed significantly less activation than controls. These temporal regions have been described as being involved in directly and efficiently connecting

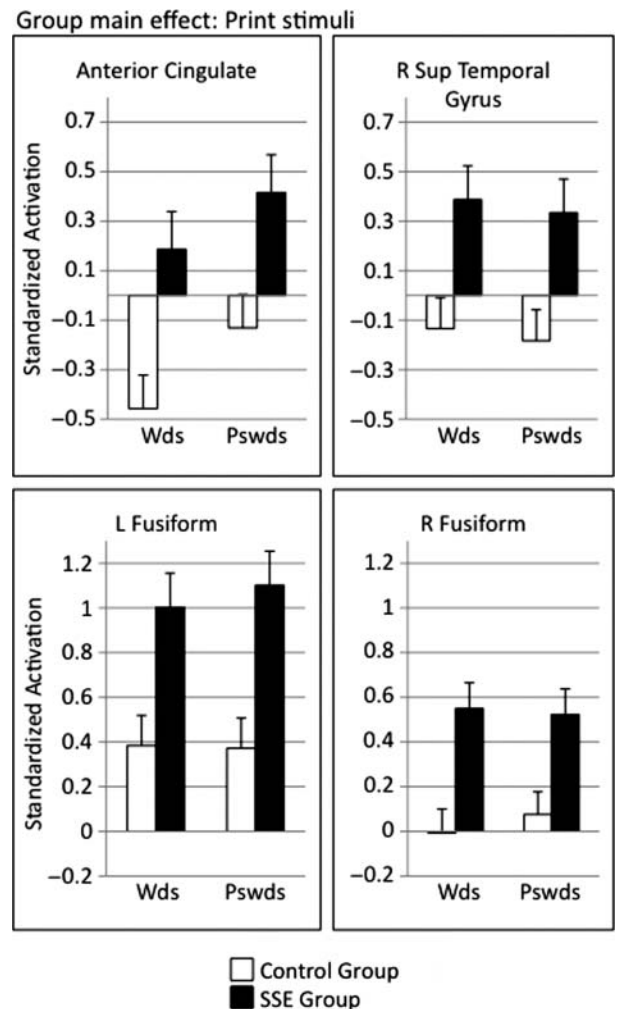
**Figure 3.** Axial slices of selected regions of interest in which the groups differed in the processing of printed words and pseudowords. Activations are significant at  $< .01$ , FDR corrected. Images are presented in radiological format, with the right hemisphere displayed on the left side of the image. Numerals represent z-axis mm values in MNI space. Blue/purple represents greater activation for the SSE group; yellow/red represents greater activation for the control group. From top to bottom: anterior cingulate (+22), right superior temporal gyrus/superior temporal sulcus (+6), bilateral fusiform (-16).



acoustic and lexical information (Hickok & Poeppel, 2004; Szaflarski et al., 2006; Poeppel et al., 2008). Reduced engagement of these ventral regions could indicate less efficient acoustic-to-lexical processing for children with SSE within this circuit—that is, the SSE group appears to overengage the less efficient dorsal articulatory motor mapping regions and underengage some of the (potentially more efficient) auditory-lexical regions when listening to spoken words and pseudowords.

The left fusiform gyrus, which has been described as part of the semantic processing network (e.g., Poeppel et al., 2004; Vigneau et al., 2006) was found to be engaged to a greater extent in the SSE group relative to the control group. Thus, not all regions of the ventral circuit were observed to be underengaged. However, in typically developing individuals, reduced fusiform activation during auditory tasks is observed with increasing age (Church, Coalson, Lugar, Petersen, & Schlaggar, 2008), suggesting maturational influences. On the basis of this developmental perspective, it is possible that the SSE group's greater fusiform activation is due to a less "mature" perceptual system.

**Figure 4.** Barplots of blood oxygen level dependent response to printed words and pseudowords in selected regions of interest. Error bars represent 1 SE.



*Attention / task-induced deactivation.* Task-induced deactivation is a phenomenon observed in many neuroimaging studies that is presumed to reflect a reallocation of resources from a resting state to task engagement (here, attending to spoken or printed stimuli; Fox & Raichle, 2007; McKiernan, Kaufman, Kucera-Thompson, & Binder, 2003; Raichle & Snyder, 2007). The circuitry involved in task-induced deactivation is broadly distributed and overlaps with regions related to cognitive processes of selective attention and orientation to stimuli (Behrmann, Geng, & Shomstein, 2004; Fan, McCandliss, Fossella, Flombaum, & Posner, 2005). In the present study, children in the SSE group displayed a marked pattern in which there was greater activation than controls in multiple regions within this broad orienting network when processing speech and print. The general pattern was for the control group to show reduced

blood flow when processing speech (but for the SSE group to show no such reduction) in several regions, including inferior and superior parietal lobules, anterior cingulate, precuneus, cuneus, and middle frontal gyrus. Although alternative explanations for this observation are possible, one plausible “cognitively based” interpretation is that children with SSE are “working harder” than control participants to pick up sensitive phonological cues in both modalities. Returning to Shriberg’s concept of phonological attunement (cf. Shriberg, 1994; Shriberg et al., 2005), the present results may indicate that school-age children with SSE (but normal language) must expend more neural/cognitive effort to achieve the same result as typically speaking children on perception-based tasks that require them to pay close attention to phonetic details in their environment.

## **Differences in Print Processing**

As has been observed in prior studies, the children with SSE were, on average, somewhat below the controls on behavioral measures of reading, spelling, and phonological awareness (see Table 1). Therefore, we examined the patterns of functional activation in response to printed words and pseudowords to understand the neural characteristics of the differences in the phonological bases of literacy. As was found with spoken language, children with SSE displayed several differences in the processing of printed mismatches (both real words and pseudowords) when compared with their typically speaking peers. In response to printed words and pseudowords, children with SSE showed greater activation than controls in several regions, including the left IFG (a region associated with articulatory encoding during reading), left globus pallidus (a region that also differed between groups in response to speech), and right STG. Right-hemisphere compensation has been observed in fMRI studies of children with reading disabilities (although more commonly in IFG and temporal–parietal regions; for a review, see Sandak, Mencl, Frost, & Pugh, 2004); this pattern may reflect failure to consolidate a left hemisphere–dominant circuit.

In addition, the SSE group had significantly greater activation in both left and right fusiform gyrus. The control group, on average, did not activate the right fusiform gyrus when processing the printed stimuli, suggesting a pattern of left lateralization and perhaps a more mature neural response pattern to print in this region (Turkeltaub, Gareau, Flowers, Zeffiro, & Eden, 2003). In contrast, the children with SSE engaged the fusiform bilaterally, reflecting perhaps a reading system that is not left hemisphere dominant. Studies of children with reading disabilities often implicate the fusiform gyrus, although the canonical pattern is for poor readers to show less engagement of this region than typical readers (Hoeft et al.,

2007; Pugh et al., 2008). Thus, the fusiform gyrus may play a critical role in the speech- and print-processing differences in children with SSE, although these children do not show the same neural signatures in the fusiform as do children with reading disabilities.

Children with SSE also showed significantly greater activation than their typically speaking peers in anterior cingulate; the control group was observed to show some deactivation of this region when processing printed words, and to show neither activation nor deactivation when processing pseudowords. Similar differences were observed in response to speech in this region. Carter et al. (1998) found that this region activates during conditions when errors are likely to occur; therefore, we speculate that greater activation may indicate that children with SSE (implicitly) recognize the reading and processing of spoken language as a task in which errors may be more likely to happen.

## **Caveats and Future Directions**

Because this is one of the first studies to examine neural processing differences between children with and without persistent SSE, the results must be considered preliminary. It is clear that prospective replication is needed to determine whether the regions identified here will continue to be the most robust indicators of perceptual differences in children with SSE when a different sample is assessed. Indeed, different neural profiles may emerge depending on how subtypes (and control groups) are defined and the imaging tasks that are used. The fMRI task that was completed in this study—a perceptual judgment task—could be complemented by production tasks (cf. Tkach et al., 2011). Longitudinal investigation of changes in neural response under different treatment conditions might help us to better understand differences in the neurobiology of children with SSE; for example, prior research has demonstrated that perceptual training may improve speech perception and production (e.g., Rvachew, Nowak, & Cloutier, 2004), and reasonable predictions can be made about regions that might respond to perceptual training (Guenther et al., 2004). Finally, differences in brain structure and connectivity among regions/networks may improve our understanding of the neural profiles of children with SSE.

The group differences in print processing did not directly mirror the differences identified in speech processing. The extent to which reading and listening rely on the same brain regions depends on reading skill, such that better readers show greater neural integration across listening and speaking modalities (Shankweiler et al., 2008). In the present study, we used controls matched on IQ, age, and oral language skills, but this procedure also resulted in moderate but significant group differences in some phonological and reading skills. Future studies might be designed to compare children with

SSE with reading-matched controls. This would enable a more direct assessment of potential similarities and differences in the neural circuitry of children who are similar in reading performance but differ only in speech production abilities.

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## Summary and Implications

School-age children with SSE were found to differ from typically speaking controls in their neural responses to spoken and printed words and nonwords. Compared with control participants, activation differences in speech perception found among children with SSE were hypothesized to reflect overengagement of a dorsal (motor) speech perception system, underengagement of a ventral (lexical) speech perception system, and a failure to show the typical task-induced deactivation patterns. The results of this study may be viewed in some ways as confirmatory—that is, in children with SSE, several regions canonically associated with speech processing (based on many years of study of normal processes and acquired pathologies in adults) were found to differ from those in controls. Further specifying the neural differences in childhood SSE (with respect to brain function, structure, and connectivity) should have important implications for informing neurobehavioral models of speech development and articulatory impairments. Moreover, continued work in the neurobiological bases of SSE may help to provide foci for neurogenetic studies of speech development and disorders (Peterson et al., 2007) and may help to further define developmental trajectories of speech perception and production in children with SSE.

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