

# Subcortical differentiation of stop consonants relates to reading and speech-in-noise perception

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Children with reading impairments have deficits in phonological awareness, phonemic categorization, speech-in-noise perception, and psychophysical tasks such as frequency and temporal discrimination. Many of these children also exhibit abnormal encoding of speech stimuli in the auditory brainstem, even though responses to click stimuli are normal. In typically developing children the auditory brainstem response reflects acoustic differences between contrastive stop consonants. The current study investigated whether this subcortical differentiation of stop consonants was related to reading ability and speech-in-noise performance. Across a group of children with a wide range of reading ability, the subcortical differentiation of 3 speech stimuli ([ba], [da], [ga]) was found to be correlated with phonological awareness, reading, and speech-in-noise perception, with better performers exhibiting greater differences among responses to the 3 syllables. When subjects were categorized into terciles based on phonological awareness and speech-in-noise performance, the top-performing third in each grouping had greater subcortical differentiation than the bottom third. These results are consistent with the view that the neural processes underlying phonological awareness and speech-in-noise perception depend on reciprocal interactions between cognitive and perceptual processes.

brainstem | dyslexia | electrophysiology | experience-dependent plasticity | learning impairment

Learning impairments, primarily reading disorders, are among the most prevalently diagnosed exceptionalities in school-aged children, affecting  $\approx 5\%$  to  $7\%$  of the population (1). These impairments coincide with a number of perceptual deficits including inordinate difficulty perceiving speech in noise as well as neural encoding deficits in the auditory system. In typically developing children, differences in contrastive speech stimuli are encoded subcortically (2), but the possible relationship between subcortical encoding of stimulus differences and reading ability has not been previously explored.

**Behavioral Impairments.** Children with reading impairments often show deficits in phonological processing, which may be caused by degraded phonological representations or an inability to access these representations effectively (3–5). This population also exhibits impairments in speech sound discrimination (i.e., contrastive syllables) relative to controls matched for age and reading level (6, 7), suggesting that impairments are not simply caused by a maturational delay. These effects are especially prevalent for place of articulation and voice onset time contrasts, which reflect dynamic spectral and temporal contrasts, respectively. Perceptual discrimination deficits seem to be limited to the rapid spectral transitions between consonants and vowels and are not found for steady-state vowels or when formant transitions are lengthened (8–10). Moreover, when presented with between- and within-phonemic category judgments, typically developing children successfully discriminate between categories (e.g., [da] vs. [ba]), whereas children with reading impairments do not (11). Reading-impaired children also show deficits on a number of nonphonetic psychophysical measures, including frequency discrimination (12, 13), sequencing sounds

(14), and temporal judgments (15, 16), and are affected more than normal-learning children by backward masking of tones (17, 18).

Additionally, children with reading impairments and those at risk for developing impairments have poorer speech-in-noise perception than their typically developing peers and show a greater deterioration in performance with decreasing signal-to-noise ratio (19, 20). Poor speech-in-noise perception and temporal discrimination are predictive of reading deficits and language delays, respectively, in young children (20, 21). Moreover, dyslexic adults have difficulty grouping streams of tones into distinct auditory events (22). These results, along with evidence showing higher thresholds for visual discrimination in noise (23), suggest that a subset of children with reading disorders may have compromised or reduced ability to overcome the adverse effects of noise on sensory stimuli. Ahissar and colleagues suggest that, unlike typically developing children, children with reading disorders are unable to profit from the repetition of stimuli, suggesting a deficit in forming a perceptual anchor (4) that may preclude separating target stimuli from background noise. This theory suggests that adults and children with reading disorders may have a perceptual attention deficit and trouble attending to target stimuli in the presence of background noise. These multiple lines of evidence suggest that reading and learning impairments arise from the interplay of multiple factors and not from a single mechanism.

**Deficits in Neural Encoding of Speech.** Deficits in phonological awareness, reading, and temporal resolution are linked to abnormal neural representation of sound in the auditory system (18, 24–32). Relative to normal-learning children, children with reading impairments show reduced cortical asymmetry of language processing (31, 33, 78) and reduced amplitude or delayed latencies of the mismatch negativity, a cortical response reflecting preconscious neural representation of stimulus differences (27, 32). Children with the weakest mismatch negativity responses also are the most likely to have the lowest literacy scores (27) and the poorest performance on categorical discrimination tasks using the same stimuli (32). Additionally, for poor readers, cortical encoding deficits are found for rapidly presented stimuli and stimuli with rapid frequency changes (similar to formant transitions) (28, 34), mimicking their impaired behavioral performance in temporal order judgments and supporting the hypothesis of a temporal processing deficit (16).

Abnormalities in neural encoding also are found subcortically. The auditory brainstem response to speech closely mimics the spectrotemporal features of the stimulus (35–37) and demonstrates experience-related plasticity (38–41). Phase-locking to speech for-

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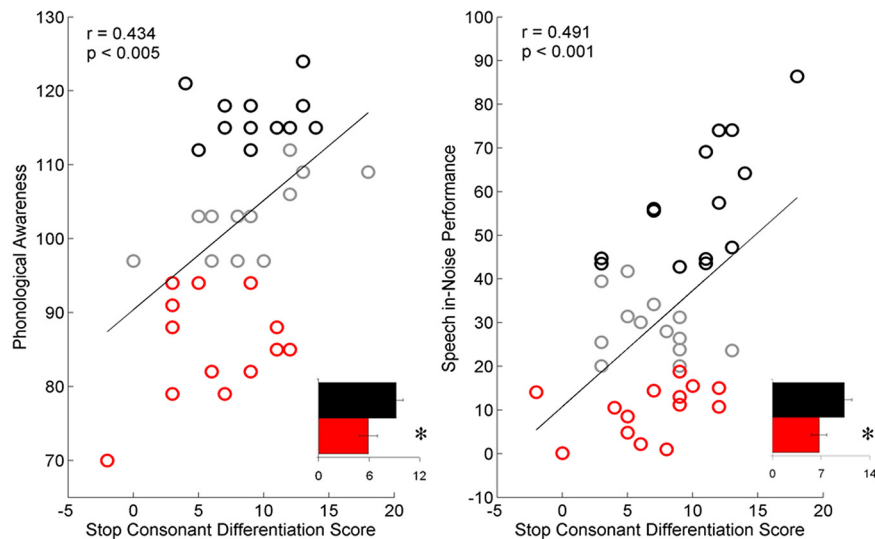
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**Fig. 2.** Subcortical differentiation is related to phonological awareness and speech-in-noise performance. The relationships between the stop consonant differentiation score (minor peaks) and phonological awareness (*Left*) and speech-in-noise performance (*Right*) are plotted. Performers in the top and bottom tertiles for each measure are marked in black and red, respectively. The stop consonant differentiation scores of performers in the top (*black*) and bottom (*red*) tertiles on each measure are plotted in the insets (means  $\pm$  1 standard error). \*,  $P < 0.05$ .

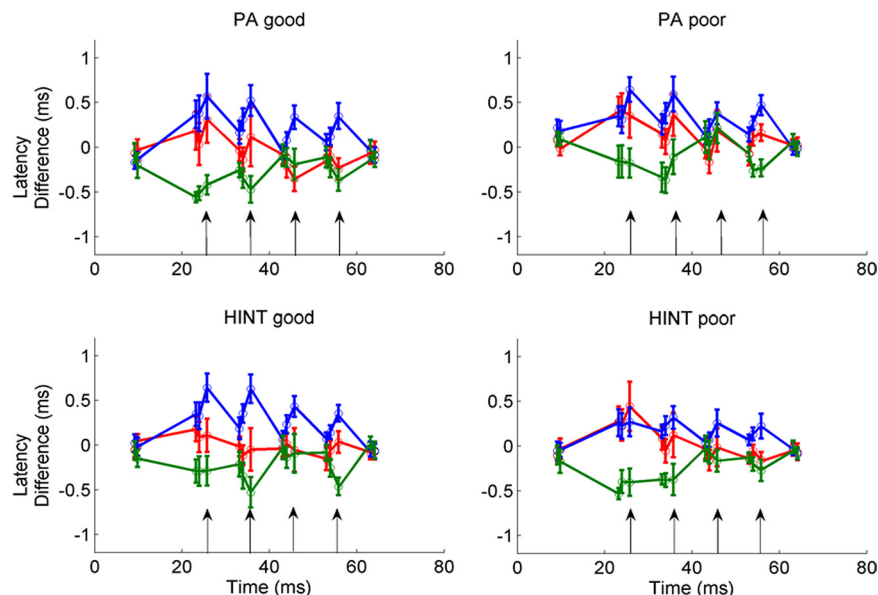
only for the response peaks reflecting the spectrotemporally changing formant structure (minor) and not for those reflecting the fundamental frequency (major), onset of the stimulus (onset), or end of the formant transition period (endpoint). Specifically, the stop consonant differentiation score for the minor peaks was positively correlated with phonological awareness (PA) [Comprehensive Test of Phonological Processing (CTOPP) Phonological Awareness  $r = 0.434$ ,  $P = 0.004$ ], reading fluency [Test of Silent Word Reading Fluency (TOSWRF)  $r = 0.399$ ,  $P = 0.008$ ], and speech perception in noise [Hearing in Noise Test-Right (HINT-R) percentile  $r = 0.492$ ,  $P = 0.001$ ] (Fig. 2). To rule out the effect of intellectual ability, attention deficits, and age on the relationships with minor peak stop consonant differentiation score, the correlational analyses were repeated with age, non-verbal IQ, and measures of hyperactive and inattentive behavior as covariates. The strength of the relationships decreased slightly but remained significant, except for reading fluency (PA:  $r = 0.306$ ,  $P = 0.059$ ; TOSWRF:  $r = 0.248$ ,  $P = 0.13$ ; HINT-R:  $r = 0.510$ ,  $P = 0.001$ ).

Between-group analyses of stop consonant differentiation scores for the minor peaks also were conducted on the highest and lowest

performing tertiles ( $n = 14$  each) for PA and HINT-R, the 2 behavioral measures that remained significant after covarying age, attentive behavior, and IQ. Similar analyses were not conducted for the major, onset, or endpoint differentiation scores because they did not correlate with any behavioral measures. The good PA group (top third: PA range 112–124, mean stop consonant differentiation score = 9.21) had higher stop consonant differentiation scores than the poor PA group (bottom third: PA range 70–94, mean stop consonant differentiation score = 5.93;  $t(26) = 2.414$ ,  $P = 0.023$ ). Similarly, the good HINT-R group (top third: range 42.8–86.4, mean stop consonant differentiation score = 10.28) had greater stop consonant differentiation scores than the poor HINT-R group (bottom third: range 0.1–18.8, mean stop consonant differentiation score = 6.71;  $t(26) = 2.287$ ,  $P = 0.031$ ), although the effect was only marginally significant after correcting for multiple comparisons (see Figs. 2 and 3).

## Discussion

Preconscious cortical differentiation of place of articulation contrasts has been related previously to reading ability (27, 32). In the present study, subcortical differentiation of contrastive



**Fig. 3.** Children who are poor performers on measures of phonological awareness (PA) and speech-in-noise perception (HINT) have reduced subcortical differentiation of 3 stop consonant stimuli relative to good performers. Normalized latency differences are plotted over time for the good performers (*Left*) and poor performers (*Right*) for phonological awareness (*Top*) and speech-in-noise perception (*Bottom*). There is better separation among [ga] (green), [ba] (blue), and [da] (red) responses for the good performers. Response latencies were normalized by subtracting individual peak latencies from the grand average of responses to all 3 stimuli (see *Methods* and ref. 2). Minor peaks are marked with arrows.

stop consonant syllables was linked to performance on measures of phonological awareness and speech-in-noise perception for peaks corresponding to the minor voltage fluctuations between primary response peaks. These peaks reflect the formant transitions, which differ across stimuli, and are suggestive of the manner by which the brainstem represents frequencies above phase-locking limits (2), making them likely predictors of reading measures. The current study is the first to demonstrate an unambiguous relationship between reading ability and subcortical temporal encoding of contrastive speech sounds that present phonological challenges for poor readers. When children were categorized into terciles based on phonological awareness or speech-in-noise perception, the children in the top third in both groupings exhibited greater subcortical differentiation across the minor peaks than did the children in the bottom third.

The current study also discovered a relationship between subcortical differentiation of stop consonants and the perception of speech-in-noise in conditions that mimic real-word listening situations (e.g., noisy classrooms). This study builds upon previous work identifying broad relationships between perception of signals in noise and brainstem encoding (24). The current study indicates that the subcortical differentiation of stop consonants, known to pose particular perceptual challenges in noise (54), is related to the ability to perceive speech in noise.

Relationships identified in the current study were limited to the minor peaks, thought to be most representative of the differing stimulus formant trajectories (i.e., continuous formant frequency changes over time) (2, 36). By showing that children with poorer phonological awareness and speech-in-noise perception have smaller or absent latency differences among responses, the current study reinforces the notion that formant information is encoded less precisely in the reading-impaired population (18, 24–27, 43, 45). On the other hand, the major peaks are largely driven by the fundamental frequency of the stimulus (36), and previous work has found that children with reading impairments do not differ from normal-reading children in the encoding of fundamental frequency in stop consonants (24, 26, 44).

The phonological deficits experienced by children with reading impairments have been ascribed to both the perception of and the use of phonology (3–5, 16, 17, 21, 55, 56), and the relationships between phonological skills and neural transcription of sound observed in the current study are compatible with “bottom-up” and “top-down” mechanisms. Poor temporal discrimination is reflected in poor neural synchrony and abnormal transient elements of the brainstem response to speech (18), which in the present study reflect the formant trajectories of the stimuli. The current results also support the seminal work by Tallal and colleagues demonstrating that perceptual deficits seen in children with learning impairments are limited to short, spectrally dynamic elements (i.e., formant transitions) and do not affect steady-state vowels (8–10). An inability to represent formant differences in the brainstem could lead to deficient phonological representations (16, 17, 21, 55, 56) and contribute to poor reading. Brainstem processes also are shaped by corticofugal “top-down” influences, leading to the subcortical malleability observed with lifelong experience and short-term training (39–41, 57–60; see refs. 38 and 61 for reviews of language and music work, respectively). In animal models, behaviorally relevant stimuli can elicit selective cortical, subcortical, and even cochlear tuning of receptive fields via corticofugal mechanisms (62–67). Similar plasticity is found in the human auditory brainstem where attentional context can affect online brainstem encoding of speech (68). Children with reading disorders often have impaired phonological retrieval and short-term memory store (3–5), which, over time, may result in a failure to sharpen the subcortical auditory system, resulting in the deficient brainstem responses to speech stimuli and reduced differentiation of stop consonants observed here. We speculate that reading and learning

impairments are caused by mutually maladaptive interactions between corticofugal tuning and basic perceptual deficits.

The relationship between speech-in-noise performance and subcortical differentiation of stop consonants can be viewed within a similar framework. Children with reading impairments demonstrate decreased lower-brainstem efferent modulation relative to normal-learning children, similar to deficits observed in speech-in-noise perception (69). Moreover, improved performance on speech-in-noise perception tasks after training coincided with increases in efferent modulation of peripheral hearing mechanisms both in normal-reading adults and in children with learning impairments (60). As proposed by Ahissar and colleagues, cortical influences on brainstem function may result from a backwards search for more concrete representations during adverse listening conditions such as speech in noise (70). Impoverished phonological representations or an inability to access and manipulate them effectively would result in a mismatch between the proposed higher level (phonological) and lower level (auditory brainstem) representations of the stimulus (70). Because lifelong experience, short-term training, and online changes in directed attention and context can affect the auditory brainstem response to speech (38–41, 57–60, 68), it is possible that repeated failures to increase the signal-to-noise ratio in adverse listening conditions, caused by mismatches between poor phonological retrieval and deficient brainstem encoding, negatively affects brainstem encoding of speech. As with phonological deficits, deficits in speech-in-noise perception in reading-impaired children probably are the consequence of interactions among perceptual and cognitive factors.

## Conclusion

The current study identified relationships between subcortical differentiation of 3 stop consonants and phonological awareness, reading performance, and speech-in-noise performance in a group of children on a continuum of reading ability. These relationships are consistent with the view that improper utilization of phonology, probably through a combination of deficits in phonological perception and working memory, is manifested in deficient encoding of sound elements important for phoneme identification in the auditory brainstem. Behavioral training, which could enhance task-directed auditory attention, could be an effective vehicle for improving brainstem differentiation of contrastive stimuli in children with poor phonological awareness and speech-in-noise perception.

## Materials and Methods

**Participants.** Participants were 43 children, ages 8–13 years (mean age = 10.4 years, 20 girls). Of the children, 23 carried an external diagnosis of learning or reading disorders, and of those all but 1 attended a private school for children with severe reading impairments. Additionally, 11 of these children were diagnosed with comorbid attention disorders. All other children were recruited from area schools. All participants had normal hearing defined as air conduction thresholds < 20 dB normal hearing level (nHL) for octaves from 250–8000 Hz, no evident air–bone conduction gap, and click-evoked brainstem response latencies within normal limits (the 100- $\mu$ s click stimulus was presented at 80 dB sound pressure level (SPL) at a rate of 31/s). We required all children to have clinically normal click-evoked auditory brainstem responses to ensure that observed differences between groups could not be attributable to neurological or audiological impairments.

All children also had normal or corrected-to-normal vision and normal IQ scores (> 85, the boundary of the “below average” range) on the Wechsler Abbreviated Scale of Intelligence (71). The Attention Deficit Hyperactivity Disorder Rating Scale IV (72) was included as a measure of attentive behavior. Age- and sex-normed hyperactive and inattentive subtype scores were used as covariates in the correlational analyses. Children were compensated monetarily for their time. All procedures were approved by the Internal Review Board of Northwestern University.

**Reading-Related Measures.** Phonological awareness and processing were assessed with subtests of the CTOPP (73). The 6 subtests (Elision, Blending Words, Rapid Number Naming, Rapid Letter Naming, Nonword Repetition, Number

Repetition) yield 3 cluster scores, PA, Phonological Memory, and Rapid Naming, which were used for the current analyses. Children also completed the Test of Word Reading Efficiency (TOWRE), which consists of 2 subtests, Sight and Phoneme (74), that assess the ability to read real words and non-words, respectively, under a time constraint. The TOSWRF also was administered and required children to parse continuous strings of letters into words under a time constraint (75).

**Speech-in-Noise Perception Measures.** Speech-in-noise perception was evaluated using the HINT (Bio-logic Systems Corp.), which yields both a threshold signal-to-noise ratio and age-normed percentile score. Children repeated sentences presented in speech-shaped background noise as the program adaptively adjusted the signal-to-noise ratio until the child repeated  $\approx 50\%$  of the sentences correctly. The speech stimuli were always presented from the front, and the noise was presented from the front, from  $90^\circ$  to the left, or from  $90^\circ$  from the right in 3 separate conditions. Percentile scores for each of the 3 noise conditions and a composite score for all 3 conditions were used in the present analyses.

**Electrophysiological Stimuli and Recording Parameters.** Speech stimuli were synthesized in KLATT (76). Stimuli were 170 ms long with a 50-ms formant transition and a 120-ms steady-state vowel. Voicing onset was at 5 ms. Stimuli ([ba], [da], [ga]) differed from each other only on the second formant ( $F_2$ ) trajectories during the formant transition period (see [supporting information \(SI\) Fig. S1](#)). The fundamental frequency and the fourth, fifth, and sixth formants were constant across time at 100, 3300, 3750, and 4900 Hz, respectively, and within the formant transition period the first and third formants were dynamic, rising from 400 to 720 Hz and falling from 2580 to 2500 Hz, respectively. In the [ga] stimulus,  $F_2$  fell sharply from 2480 to 1240 Hz, in the [da] stimulus  $F_2$  fell from 1700 to 1240 Hz, and in the [ba] stimulus  $F_2$  rose from 900 to 1240 Hz (Fig. 1, *Inset*). The  $F_2$  transitions resolved to 1240 Hz during the steady-state vowel portion. Stimuli of alternating polarity were presented at 80 dB SPL with an interstimulus interval of 60 ms through an insert earphone (ER-3, Etymotic Research) to the right ear with Neuroscan Stim 2 (Compumedics) and were interleaved with 5 other speech stimuli differing in both spectral and temporal characteristics. Responses were collected with a vertical electrode montage (forehead ground, Cz active, and ipsilateral earlobe reference) with Neuroscan Acquire 4.3 (Neuroscan Scan, Compumedics) and digitized at 20,000 Hz.

**Data Reduction and Analysis.** Data reduction and preliminary analysis procedures followed those of Johnson et al. (2). Responses were offline bandpass filtered from 70–2,000 Hz at 12 dB roll-off per octave and averaged over a 230-ms window with 40-ms prestimulus and 190-ms poststimulus onset. Separate averages were created for each stimulus polarity, and then the 2 averages were added together (77). The final baseline-corrected average consisted of 6,000 artifact-free sweeps (3,000 sweeps per polarity; artifact defined as activity  $\pm 35 \mu\text{V}$ ). To aid in peak identification, final averages were additionally high-pass filtered at 300 Hz (12 dB/octave slope). In total, 15 peaks were identified: the onset peak and trough (onset peaks), the 8 major response peaks and troughs (major peaks), the 4 minor troughs following the major troughs (minor peaks), and the first peak and trough during the response to the steady-state vowel (endpoint peaks). For stimuli that were phonetically similar to those used in the present study, we previously found that the major and minor response peaks to [ga] occurred earlier than those to [da], which also occurred earlier than those to [ba] over the formant transition region, reflecting the second and third formant frequency differences in those stimuli (2). These differences were not found in the onset response. The latency differences gradually diminished over the course of the first 60 ms of the response, reflecting the diminishing differences among frequencies in the stimuli over time. Importantly, as is the case in the current study, there were no timing differences in the previously used stimuli (2) that might have accounted for these response latency differences.

Given the expected latency pattern of [ga] < [da] < [ba], the responses in the present study were analyzed with a 3-step metric that takes into account both the presence of the expected pattern and the magnitude of the latency differences. Stop consonant differentiation scores were calculated for each of the 15 onset, major, minor, and endpoint peaks.

**Step 1.** For each of the 3 pairwise latency comparisons ([ba] vs. [da], [ba] vs. [ga], [da] vs. [ga]) for a given peak, the peak was given a score of 1 if the expected

pattern was present and a score of 0 if it was not, resulting in an interim score ranging from 0 to 3.

**Step 2. (i)** For each peak, the difference in average latency ( $\Lambda$ ) and standard deviation were calculated for each stimulus pair (e.g., [ba] minus [da]) using the children who did not carry an external diagnosis of a learning or reading disorder ( $n = 20$ ).

(ii)  $+\Lambda'$  and  $-\Lambda'$ , defined as  $\Lambda + 0.67$  and  $\Lambda - 0.67$  standard deviations, respectively, were computed. The range between  $-\Lambda'$  and  $+\Lambda'$  accounts for 50% of the cases in a normally distributed population.

(iii) For each peak and each pairwise comparison, if the pattern was present (Step 1) and the latency difference was greater than  $+\Lambda'$ , then an additional point was added to the interim score. A point was subtracted if the pattern was absent and the latency difference was less than  $-\Lambda'$  (a latency difference in the wrong direction outside normal variance). Additionally, a point was subtracted if the pattern was present but the latency difference was less than  $-\Lambda'$ . In this case it was assumed that, although the pattern was shown, the latency difference between the 2 responses was so small that it was equivalent to there being no difference between the responses.

(iv) The scores derived from Steps 1 and 2iii were summed across all 3 pairwise comparisons ([ba] vs. [da], [ba] vs. [ga], [da] vs. [ga]), resulting in a minimum score of  $-3$  (earned by not showing the pattern in any comparison and having clear latency differences in the wrong direction) and a maximum score of 6 (earned by showing the pattern for all 3 comparisons and having clear latency differences in the expected direction).

**Step 3.** The individual differentiation scores then were summed separately for the major peaks ( $n = 8$ ) and minor peaks ( $n = 4$ ) to give maximum scores of 48 and 24 and minimum scores of  $-24$  and  $-12$ , respectively.

For the onset and endpoint differentiation scores, because there was no expected latency pattern (2), and because this finding was replicated for typically developing children in the current study, latency deviation in either direction was penalized. Any deviation greater than  $+\Lambda'$  or less than  $-\Lambda'$  (with  $\Lambda \approx 0$ ) earned  $-1$  point, resulting in a maximum score of 0 (within normal latency limits for all comparisons) and a minimum score of  $-3$  (showing deviation outside latency norms on all 3 comparisons). Scores then were summed across onset ( $n = 2$ ) and endpoint ( $n = 2$ ) peaks, yielding a maximum score of 0 and a minimum score of  $-6$  in both cases.

For graphing purposes in Fig. 3, normalized latency values were calculated as described in ref. 2. The response latencies for the typically developing children ( $n = 20$ ) were averaged across all 3 stimuli for each peak. This grand average latency was subtracted from the latency of each individual child for each peak. Thus, peak latencies that were earlier than the grand average (i.e., responses to [ga]) would have negative normalized values, and peak latencies later than the grand average (i.e., responses to [ba]) would have positive normalized values. Responses to [da] occurred at roughly the same latency as the grand average and thus have normalized latencies near zero.

Pearson's correlations between stop consonant differentiation scores and behavioral measures of reading, phonological awareness, and speech-in-noise perception were conducted in SPSS (SPSS Inc.). Outliers on psychoeducational measures (3 scores) were corrected to be at  $-2$  standard deviations from the standard normed mean of the test (a score of 100). No outliers were present for the speech-in-noise measures. IQ, attentional behavior, and age were included as covariates in correlational analyses. Additionally, children were divided into terciles based on CTOPP PA score and HINT noise right percentile because those measures were the most strongly related to subcortical differentiation, even after controlling for covariates. [Table S1](#) gives performance on behavioral measures for each group. Stop consonant differentiation scores were compared between top and bottom terciles ( $n = 14$ ) of each measure using independent  $t$ -tests. Because of multiple comparisons in the correlations, alpha was set at 0.01. Because the  $t$ -test comparison was conducted only on the stop consonant differentiation score for the minor peaks, alpha was adjusted to 0.025.

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