Investigating the 'Atypical Rhythm Risk' hypothesis in children with developmental language disorder using an EEG rhythmic speech paradigm

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Abstract

Sensitivity to rhythmic and prosodic cues in speech has been described as a precursor of language acquisition. Consequently, atypical rhythmic processing during infancy and early childhood has been considered a risk factor for developmental language disorders. This is the 'atypical rhythm risk' (ARR) hypothesis. The neural processing of rhythmic speech has not yet been explored in children with developmental language disorder (DLD). Here we utilise EEG and a rhythmic speech paradigm previously utilised for dyslexia to investigate the ARR hypothesis in 9-year-old children with and without DLD. We investigate angular velocity, power, ERPs, phase-amplitude coupling (PAC) and phase-phase coupling (PPC), at three frequency bands, delta, theta and low gamma. Both groups demonstrated significant and equivalent phase entrainment in the delta and theta bands, but only the control children showed significant phase entrainment in the low gamma band. Further, while angular velocity in the delta and theta bands was equivalent by group, there was a significant gamma-band difference. The children with DLD also exhibited significantly more theta and gamma power compared to the control children. Regarding PAC and PPC, both groups showed significant and equivalent coupling strength. However, group resultant phase differences showed that low-frequency phase (delta and theta) affected gamma oscillations differently by group. The EEG data show important differences between children with and without DLD in the neural mechanisms underpinning the processing of rhythmic speech. The findings are discussed in terms of auditory theories of DLD.

Keywords

Developmental language disorder, rhythmic audio-visual speech, EEG, neural phase entrainment, atypical rhythm risk

1. Introduction

A growing number of cognitive behavioural studies have investigated rhythmic and/or musical processing in children with developmental language disorder (DLD), and have shown that atypical rhythm processing is associated with speech and language processing problems (e.g., Corriveau et al., 2007; Corriveau & Goswami, 2009; Przybylski et al., 2013; Cumming et al., 2015a,b; Richards & Goswami, 2015, 2019; Sallat & Jentschke, 2015; Bedoin et al., 2016). This apparent link between rhythm processing and language processing has been described by the Atypical Rhythm Risk (ARR) hypothesis (Ladányi et al., 2020). Following a review of the literature, Ladányi et al. (2020) proposed that individuals with atypical rhythm processing are at higher risk for developmental speech/language disorders, including developmental dyslexia, DLD and stuttering. Disordered development of oral language (DLD) affects between 3 – 7% of children in all cultures (Bishop et al., 2017). The clinical diagnosis for such children was previously Specific Language Impairment (SLI), however the multinational and multidisciplinary CATALISE study recommended a change in terminology to DLD (Bishop et al., 2017). Children given a diagnosis of DLD have a range of language difficulties that cause functional impairments in their daily lives, yet have no known biomedical aetiology. The core dimensions of difficulty are problems with syntax and grammar, problems with word semantics, impairments in verbal memory and problems with phonology (see Leonard, 2014; Bishop, 2013). The possibility that atypical *neural* processing of rhythm is associated with DLD has not yet been investigated, and is the focus of the current study.

At the sensory/neural level, the ARR hypothesis is captured by Temporal Sampling (TS) theory (Goswami, 2011; 2015, 2022). TS theory was originally proposed to explain the rhythmic impairments found in children with developmental dyslexia, a disorder of written language processing that presents with core phonological impairments (Stanovich, 1988; Swan & Goswami, 1997; Snowling, 2000). Prior TS-driven behavioural studies of rhythmic

processing by children with dyslexia and children with DLD have shown many similarities, such as impaired sensory discrimination of amplitude rise times (important acoustic cues to rhythm, Corriveau et al., 2007; Corriveau & Goswami, 2009; Fraser et al., 2010; Beattie & Manis, 2012; Cumming et al., 2015a; Richards & Goswami, 2015, 2019) and relative insensitivity to syllable stress patterns in words and phrases (Cumming et al., 2015a,b; Richards & Goswami, 2015, 2019). TS theory suggests that the sensory/neural processing of rhythm is impaired because acoustic processing of the amplitude envelope of both speech and musical signals is atypical in children with developmental disorders of language, particularly at amplitude modulation rates <10 Hz that are known to carry information about rhythm and (for speech) syllable stress patterns (see Greenberg, 2006). The amplitude envelope of speech is known to be tracked cortically, possibly via entrainment of endogenous low-frequency oscillations <10 Hz (corresponding to the electrophysiological delta and theta bands, 0.5-4 Hz and 4-8 Hz, respectively) to the speech envelope (Giraud & Poeppel, 2012). Regarding the neural side of TS theory, prior TS studies with dyslexic children have documented neural differences compared to typically-developing (TD) children during the processing of both speech and non-speech rhythmic stimuli (Power et al., 2013; Keshavarzi et al., 2022; Peter et al., 2022). For speech stimuli, children's neural processing of rhythm has been investigated using the syllable repetition task (Power et al., 2012). In this task, EEG is recorded while children with dyslexia listen to the speech syllable "ba" presented audio-visually at a repetition rate of 2 Hz (Power et al., 2013; Keshavarzi et al., 2022). Both 9-year-old (Keshavarzi et al., 2022) and 13-year-old (Power et al., 2013) children with dyslexia show atypical delta-band phase synchronisation in the syllable repetition task, exhibiting a different preferred phase to children without dyslexia for the delta band only. The theta and alpha bands are not affected. Both Power et al. (2013) and Keshavarzi et al. (2022) reported that neural responding in the delta band was aligned to a different temporal point for each group, despite the highly

predictable rhythmic input. These studies suggest that there is an optimal (or preferred) phase of entrainment regarding the accurate and efficient processing of rhythmic inputs, and that this preferred phase differs in the dyslexic brain. To date, the rhythmic syllable repetition task has not been administered to children with DLD. We provide such a study here.

Prior neural studies of children with DLD have not to date employed rhythmic processing tasks nor investigated the TS framework. The absence of neural rhythm studies is surprising, as adult neural studies show that temporal prediction of the location of stressed syllables facilitates syntactic processing (Kotz & Schmidt-Kassow, 2015). Linguistic analyses show that adults produce a stressed syllable on average every 500ms, a "beat rate" of 2Hz (Dauer, 1983), a finding that underpins the choice of repetition rate in the current task. Prior DLD studies have instead primarily utilised fMRI and have sought structural and functional differences that may throw light on the aetiology of this developmental disorder. For example, there has been interest in whether hemispheric asymmetries during linguistic processing are different when children with DLD are compared to TD children (Bishop, 2013). While laterality per se has not proved conclusive (Parker et al., 2022), one of the most consistent findings in the functional fMRI literature with DLD children is that left frontal and temporal cortical areas show reduced activation during language processing (Asaridou & Watkins, 2022, for review). Children with DLD also show microstructural differences in dorsal anatomical pathways that connect frontal and temporal areas, as well as structurally-atypical basal ganglia (Badcock et al., 2012; Lee et al., 2013; Watkins et al., 2002). Although both the temporal areas and the basal ganglia are important for speech processing, speech motor learning and temporal prediction (see Kotz & Schmidt-Kassow, 2015), fMRI studies have not revealed which neural mechanisms associated with these areas could be affected in DLD. There are also prior EEG and MEG studies with DLD children, but again these studies have not adopted a rhythmic nor a speech tracking focus, and have not thrown light on underlying mechanisms (e.g. Shafer et

al., 2001). Many of the prior child EEG studies concern the overlap between DLD diagnoses and epileptic seizures (e.g. Mehta et al., 2015; Systad et al., 2019). However, a series of EEG studies with German-learning infants has shown that those at family risk for DLD show impaired neural ERP markers of language learning, such as a delayed N400 (thought to index semantic integration, Friedrich & Friederici, 2005, 2006). ERP studies with English-learning infants at family risk for DLD using a non-speech tone pair task have shown that at-risk infants have ERPs with larger amplitudes than TD infants when the tones follow each other rapidly in time (gap of 70 ms, Benasich et al., 2006), and also show a longer latency (in the N250) which is right-lateralized. Meanwhile, prior MEG studies of DLD using speech processing tasks are consistent with the functional fMRI literature in showing atypical left hemisphere temporal function (Helenius et al., 2009, 2014).

The TS framework (TSF) differs from these prior fMRI and MEG literatures in predicting impairments in right-lateralised neural mechanisms during language processing for children with DLD (Goswami, 2022, for review). However, the TSF is not based on laterality differences, rather the right hemisphere predictions arise from the nature of the neural mechanisms of speech encoding that are proposed to be impaired. These mechanisms involve the oscillatory encoding of slower amplitude modulation (AM) information in the speech amplitude envelope, also called cortical speech tracking. Slower AM information in speech is thought to be preferentially processed in the right hemisphere, while faster AM information is thought to be processed in the left hemisphere (Boemio & Poeppel, 2005; see also Rios-Lopez et al., 2020, 2022, for child data). For linguistic inputs, these hemispheric preferences appear to change during the acquisition of literacy when orthographic representations are acquired (Cutini et al., 2016; Lallier et al., 2017). TS theory proposes atypical sensory/neural processing of the slower rates of AM nested in the speech amplitude envelope (AE) for children with DLD (Goswami, 2011, 2015, 2022). The AE is the slow-varying energy contour of speech that aids

the perception of rhythm (Greenberg, 2006). The AE contains a range of AM patterns, created by hierarchical nesting of AMs at different temporal rates that are functionally connected by phase-phase and phase-power relations (Ghitza & Greenberg, 2009; Giraud & Poeppel, 2012; Goswami & Leong, 2013; Gross et al., 2013). For example, computational speech modelling studies show that the phase relations between these different AM rates (delta-theta, theta-beta/low gamma) provide systematic statistical cues to both phonological units such as stressed vs unstressed syllables, syllables, and onset-rimes (acoustic-emergent phonology, Leong & Goswami, 2015) and also to aspects of grammar such as plural marking (Flanagan & Goswami, 2018). By TS theory, therefore, impaired cortical tracking of one or more of the AM rates nested in the AE, or of the phase-amplitude or phase-phase relations between different AM bands, could cause atypical development of one or more aspects of language.

Recent infant data support the linguistic predictions made by TS theory. In the Cambridge UK BabyRhythm project, 120 infants were followed longitudinally from the age of 2 months, and EEG was recorded while the infants listened to either sung speech (nursery rhymes), to the rhythmic syllable repetition task, or to a matched drumbeat (nonspeech) rhythmic repetition task (Attaheri et al., 2022a,b; Ni Choisdealbha et al., 2022, 2023; Keshavarzi et al., 2023). In the syllable repetition task, infants showed evidence for a group preferred phase as early as 2 months of age, and by 6 months individual phase consistency was present over both left- and right-temporal regions as well as over parietal-occipital regions (Ni Choisdealbha et al., 2023). Individual phase angles predicted language acquisition at 18 months as measured by an infant-led vocabulary task (in this task, the infant selects one of two pictures that matches a spoken word, Ni Choisdealbha et al., 2023). In the rhythmic drumbeat (nonspeech) task, neural phase angle at 9 months predicted nonword repetition at 24 months, a phonological measure (Ni Choisdealbha et al., 2023). Accordingly, delta-band phase angles to both speech and nonspeech rhythmic stimuli predict later language outcomes for a TD sample.

For the nursery rhyme analyses, significant cortical tracking of the low-frequency envelope information was present in both delta and theta bands by 4 months of age, with no significant tracking present for faster bands (alpha band, see Attaheri et al., 2022a). Phase-amplitude coupling (PAC) between both delta and theta phase and beta and gamma amplitudes was present from 4 months, and showed no change over development for this sample. When language outcomes were assessed, only delta band cortical tracking and theta-gamma PAC predicted later vocabulary and phonology measured at 24 months (Attaheri et al., preprint). Grammar measures were included in the UK BabyRhythm study, but did not show sufficient individual differences for neural prediction analyses (Rocha et al., 2024). Nevertheless, these prior TSF-driven infant data suggest that both delta-band phase angle and theta-gamma PAC in the current study may be associated with DLD status.

There is also one prior study of rhythmic speech processing using a modulation band manipulation with children with DLD, which would predict atypical gamma band speech tracking. Goswami et al. (2016) used a nursery rhyme recognition task based on speech that was either low-pass filtered (< 4 Hz, providing delta band information only) or band-pass filtered (~33Hz, providing faster low gamma band modulations only) so that the speech input was degraded and difficult to recognise. The children's task was to recognise the matching nursery rhyme by making a selection in a picture-based multiple choice format. Goswami et al. reported that 9-year-old children with DLD were worse than TD age-matched children when selecting bandpass-filtered targets, but not when selecting low-pass filtered targets. Children with both DLD and dyslexia (DLD children with phonological impairments) were worse than TD children at recognising the nursery rhymes in both filtered speech conditions. All groups of children found it easier to recognise the low-pass filtered rhymes (e.g. children with DLD recognised 89% of the low-pass filtered rhymes compared to 42% of the bandpass filtered rhymes, while the TD children recognised 94% and 59% respectively). When relations with

196 language development were investigated in the full sample of 9-year-olds (N = 95), accuracy
197 in the bandpass-filtered speech condition was the stronger predictor of language outcomes,
198 accounting for 12% of unique variance in the Clinical Evaluation of Language Fundamentals
199 (CELF-III, Wiig, Semel & Secord, 2017) receptive language measures and 10% of unique
200 variance in CELF expressive language scores. By contrast, accuracy in the low-pass filtered
201 speech condition accounted for 9% of unique variance in the phonological measure (rhyme
202 awareness), but only 3% of unique variance in the CELF ($p < 0.05$). To interpret their data,
203 Goswami et al. (2016) suggested that although prior TSF studies of children with dyslexia and
204 children with DLD indicate a shared sensory difficulty in processing temporal modulation
205 patterns in speech, the neural temporal integration windows that are most impaired may differ
206 for each disorder. Children with DLD may have more difficulties with faster temporal
207 integration windows in speech-based tasks (gamma band difficulties).

208 Accordingly, we explored a number of *a priori* predictions in the current study. First, on
209 the basis of the shared behavioural rhythmic features of dyslexia and DLD and our prior TSF
210 dyslexia data, we expected atypical delta-band entrainment (reduced phase consistency) and a
211 different group preferred phase for the participants with DLD compared to the age-matched
212 TD control children. However, given the filtered speech findings discussed above (Goswami
213 et al., 2016), and in contrast to the earlier rhythmic syllable repetition studies of children with
214 dyslexia (Power et al., 2013; Keshavarzi et al., 2022), gamma band oscillations were measured
215 in the current study as well. Given Goswami et al.'s (2016) finding of rhythmic speech
216 processing difficulties at faster modulation rates, a second prediction was possible gamma band
217 oscillatory group differences. Third, on the basis of our TSF infant data, we tentatively
218 predicted atypical theta-gamma PAC for the children with DLD. The prior study of children
219 with dyslexia that explored angular velocity (Keshavarzi et al., 2022) had also reported that the
220 group angular velocity in the delta band for the children with dyslexia was almost twice the

rate of the TD control group, suggestive of different changes in phase across time for the dyslexic group. Accordingly, a final tentative prediction was that angular velocity in the delta band may differ between children with and without DLD. We note that the prior literature on sensory impairments in rapid auditory non-speech (tone) processing in infants at family risk for DLD would also predict gamma band impairments for the DLD children (Ortiz-Mantilla & Benasich, 2013).

2. Methods and materials

2.1. Participants

Sixteen typically developing children (mean age of 109 ± 13 months) and sixteen children with DLD (mean age of 108 ± 11 months) took part in the study. All participants were taking part in an ongoing study of auditory processing in DLD and had English as the main language spoken at home (see Parvez et al., 2024). All participants exhibited normal hearing when tested with an audiometer. In a short hearing test across the frequency range 0.25 – 8 kHz (0.25, 0.5, 1, 2, 4, 8 kHz), all children were sensitive to sounds within the 20 dB HL range. DLD status was assessed by administering two expressive and two receptive subtests of the CELF (Wiig, Semel & Secord, 2017). The subtests were recalling sentences, formulating sentences, and (depending on age) two of the word structure, sentence comprehension, word classes and semantic relationships subtests. Children who scored below the mean (7 or less when the mean score is 10) on at least 2 of these subtests were included as DLD. The control children also received either one or two of the CELF tasks, recalling sentences (N = 16) and formulating sentences (N = 11) and scored in the normal range. The Picture Completion subtest of the Wechsler Intelligence Scale for Children (WISC IV, Wechsler, 2016) was used to assess non-verbal intelligence (NVIQ) and did not differ between groups. Group performance for the tasks administered to both groups is shown in Table 1.

Table 1. Details of the participating children, showing Scaled Scores (ScS, population mean = 10) and Standard Scores (SS, population mean = 100).

	DLD	Age-Matched Control (TD)
N	16	16
Age (months) [A]	108.3 (11.3)	108.9 (13.3)
Recalling Sentences ScS [B]	5.3 (1.7)	10.9 (2.3)
Formulating Sentences ScS [B]	4.4 (1.9)	10.8 (1.7)
WISC Picture Completion ScS [A]	7.9 (3.0)	9.9 (2.8)

Note. **A**: TD and DLD groups are not significantly different; **B**: DLD group significantly worse than TD group, $p < 0.01$. WISC: Wechsler Intelligence Scale for Children.

2.2. Experimental set up and stimuli

The experimental set up and stimuli used in the EEG study were identical to those described by Keshavarzi et al. (2022, 2024). After putting the EEG cap, the children were seated in an electrically shielded soundproof room. They watched (on the screen) and listened (through earphones) to audio-visual stimuli and the EEG data were collected at a sampling rate of 1 kHz using a 128-channel EEG system (HydroCel Geodesic Sensor Net). The stimuli were a talking head presenting rhythmic sequences of the syllable “ba” at a rate of 2 Hz with both visual and additory information present. Visual cues began 68 ms before the onset of the stimulus “ba” as in natural speech. Each trial (sequence) consisted of sequence of 14 syllable “ba” with both visual and auditory information present (See Figure 1). One of the 14 syllables (either 9th, 10th or 11th syllable) in each trial could be out of time (rhythmic violation), in a random order. The temporal delay was calibrated individually and adaptively for each child, so that the oddball was detected around 79.4% of the time by a button press. Each child was presented with 90 trials, 15 of which did not have a violation and were presented randomly as catch trials. Each trial contained three periods (see Figure 1): entrainment period, violation period, and return-to-isochrony period. Children were instructed to concentrate on the lips of the female face on the screen and to listen to the auditory stimuli. They were also asked to press a target key on the keyboard when one of the syllables was out of time. The total time for the EEG recording

(excluding the set-up) was about 15 min. Via the adaptive procedure, the success rate in the behavioural task was equated for all participants.

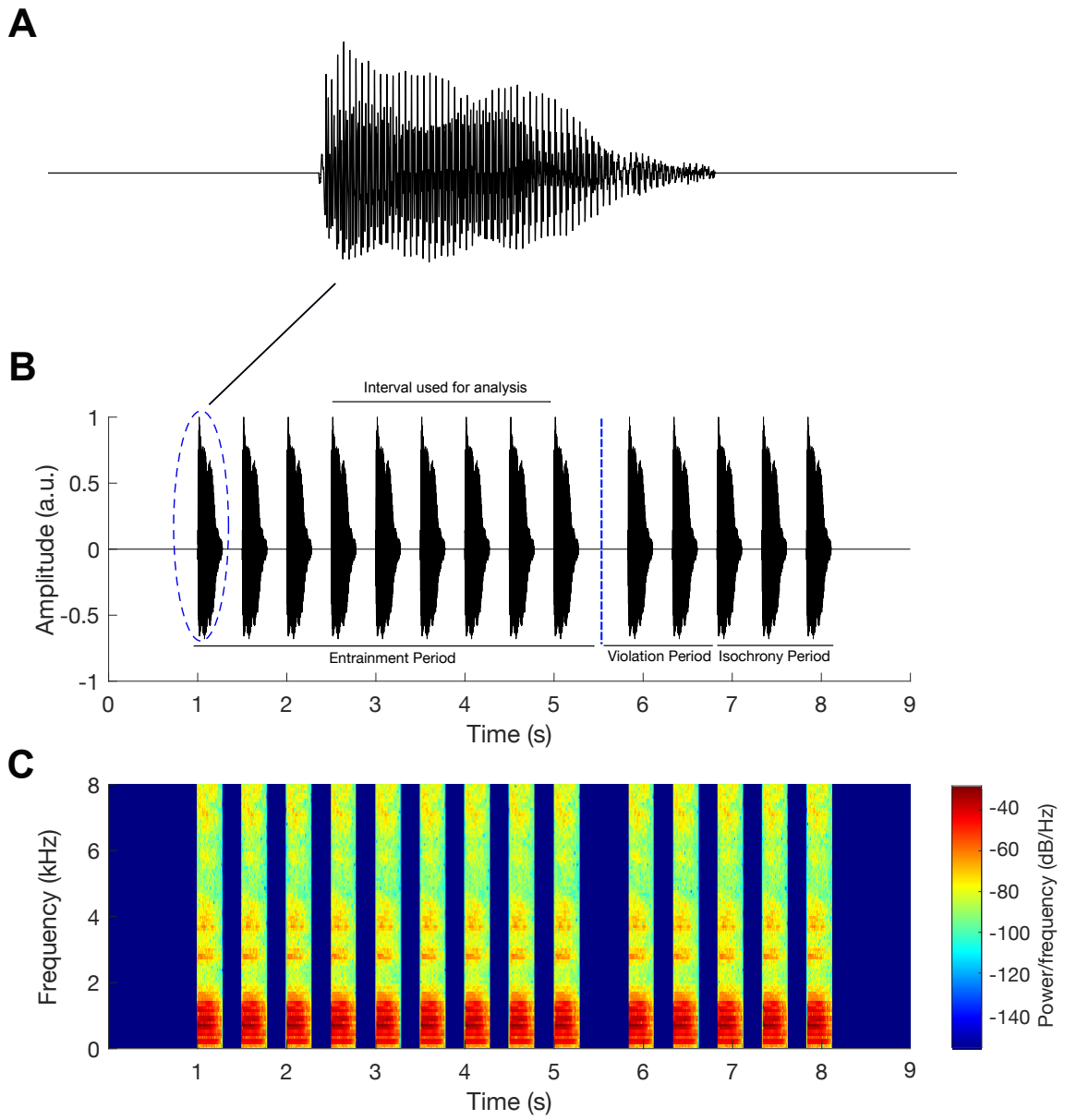


Figure 1. Experimental stimuli. Panel A shows the waveform of a single syllable "ba", panel B indicates an example trial (sequence of "ba" stimuli) with the violation at position of 10th stimulus, and panel C shows the spectrogram corresponding to sequence in panel B. This figure reproduced with permission from Keshavarzi et al., 2022.

2.3. EEG data pre-processing and analyses

The EEG data were first referenced to Cz and then band-passed filtered into 0.2 – 48 Hz using a zero phase FIR filter with low cutoff (−6 dB) of 0.1 Hz and high cutoff (−6 dB) of 48.1 Hz (EEGLab Toolbox; Delorme and Makeig, 2004). The bad channels were identified and interpolated using spherical interpolation (EEGLab Toolbox). The Independent Component Analysis (provided by EEGLab Toolbox) was run for each participant and the calculated independent components were then evaluated very carefully to remove artefactual components such as eye blinks and eye movements. The pre-proceed EEG data were downsampled to 100 Hz and band-pass filtered into delta (0.5 – 4 Hz), theta (4 – 8 Hz) and low gamma band (25 – 40 Hz) frequency bands (MNE Library-Python, Gramfort et al., 2013). The data were then epoched into individual trials, in the time interval of 0.5 s before the onset of the first "ba" stimulus and 4 s after that. In this study, the analyses only were focused on the EEG data corresponding to stimuli 4th-8th in the entrainment period of each trial to ensure that rhythmicity has occurred.

2.4. Computation of angular velocity

The angular velocity, $w(t)$, for signal $s(t)$ over time interval $\Delta t = t_1 - t_2$ is calculated as:

$$w(t) = \frac{\Delta\varphi_t}{\Delta t} \quad (1)$$

where $\Delta\varphi_t$ refers to the changes in the instantaneous phase ($\varphi(t)$) of the signal over the time interval Δt. The instantaneous phase is calculated as:

$$\varphi(t) = \arctan\left(\frac{s_H(t)}{s(t)}\right) \quad (2)$$

where $s_H(t)$ is the Hilbert transform of signal $s(t)$, and is calculated as:

$$s_H(t) = \frac{1}{\pi}\int_{-\infty}^{\infty}\frac{s(\tau)}{t-\tau}d\tau \quad (3)$$

2.5. Computation of phase entrainment

To check the phase entrainment for each group in each frequency band, we performed the following steps (Keshavarzi et al., 2022):

(1) Calculating instantaneous phases of all 128 EEG channels at the times corresponding to onsets of the 5 "ba" stimuli (4th-8th) in each trial.

(2) Computing mean phase for each trial by averaging across the phase values obtained for all EEG channels and for the 5 "ba" stimuli in step 1. This resulted in a single-phase value for each trial.

(3) Considering a single unit vector whose angle is determined by the phase value obtained in step 2 for each trial.

(4) Calculating the mean vector for each child by averaging across the unit vectors in step 3. This resulted in a single vector for each child, called *child resultant vector*.

(5) Considering a single unit vector whose angle is determined by the phase of *child preferred vector* in the vector space for each child.

(6) Calculating the mean vector for each group by averaging across the unit vectors obtained in step 5. This results in a single vector, called *group resultant vector*, for each group.

2.6. Computation of event related potential

To obtain the time-domain ERP for each group and each frequency band, the following steps were conducted: (1) Calculating the mean voltage amplitude for each trial by averaging across the voltage amplitudes of all 128 pre-processed for that each trial; (2) Calculating the mean voltage amplitude for each participant by averaging across the mean voltage obtained (in step 1) for all trials of that participant; (3) Calculating the ERP for each group by averaging across the mean voltage obtained (in step 2) for all participants in that group.

2.7. Computation of the band-power

To investigate the power of neural response over the entrainment period used for analysis, we calculated the power of EEG responses for each child and each group using in four steps: (1) Calculating the power of each EEG channel separately for each trial; (2) Calculating the power for each trial by averaging across power obtained (in step 1) for channels; (3) Calculating the

power for each participant by averaging across power obtained (in step 2) for all trials of that participant; (4) Calculating the power for each group by averaging across power values obtained (in step 2) for all children in that group.

2.8. Computation of cross frequency phase-amplitude coupling

Cross frequency phase-amplitude coupling refers to the modulation of amplitude of signal at a high frequency band by the phase of low frequency oscillation. Here we quantified the strength of this type of modulation using modulation index (*MI*; Tort et al. 2008; Hülsemann, 2019):

$$MI = \frac{KL\,(U,X)}{logB} \qquad (4)$$

where B (=18) is the number of bins, U refers to the uniform distribution, X is the distribution of the data, and $KL\,(U,X)$ is Kullback–Leibler distance which is calculated as:

$$KL\,(U,X) = logB - H(P) \qquad (5)$$

where $H(.)$ is the Shannon entropy and P is the vector of normalized averaged amplitudes per phase bin which is calculated as:

$$P(j) = \frac{\bar{a}}{\sum_{k=1}^{N} \bar{a}_k} \quad , \quad j = 1, \ldots, N \qquad (6)$$

where $\bar{a}$ is the average amplitude of each bin, k refers to running index for the bins. Note that P is a vector with N elements.

2.9. Computation of cross frequency phase-phase coupling

Cross-frequency phase-phase coupling (PPC) refers to the phase synchrony between oscillations in two frequency bands. PPC between two oscillations is a value between 0 and 1, calculated as:

$$PPC = \frac{1}{T} \left| \sum_{t=1}^{T} e^{-j\Delta\varphi(t)} \right| \qquad (7)$$

where $|\,.\,|$ refers to the absolute value operator, T is the number of time samples, and $\Delta\varphi(t)$ is the phase difference, and is calculated as:

$$\Delta\varphi(t) = \varphi_1(t) - \varphi_2(t) \qquad (8)$$

where $\varphi_1(t)$ is the instantaneous phase of first signal and $\varphi_2(t)$ is the instantaneous phase of the second signal.

3. Results

3.1. Phase entrainment consistency in each group

Given our prior TSF dyslexia data, we expected consistent phase entrainment in each group in the delta and theta bands. To assess the phase entrainment consistency in each group and in each frequency band, we applied the Rayleigh test of uniformity to the child preferred phases separately for each group and for each frequency band (see Figure 2). For the control children, we found consistency in all bands: delta band ($z = 5.14$, $p = 0.004$, see Figure 2A), theta band ($z = 3.70$, $p = 0.022$, see Figure 2B), and low gamma band ($z = 3.33$, $p = 0.033$, see Figure 2C). For the children with DLD, we also found significantly consistent phase entrainment in the delta band ($z = 8.31$, $p = 8.35 \times 10^{-5}$, see Figure 2D) and in the theta band ($z = 6.00$, $p = 0.0016$, see Figure 2E). However, phase consistency in the low gamma band was absent ($z = 0.01$, $p = 0.99$, see Figure 2F). This result indicates that the DLD brain does not show significant entrainment to rhythmic input in the gamma band.

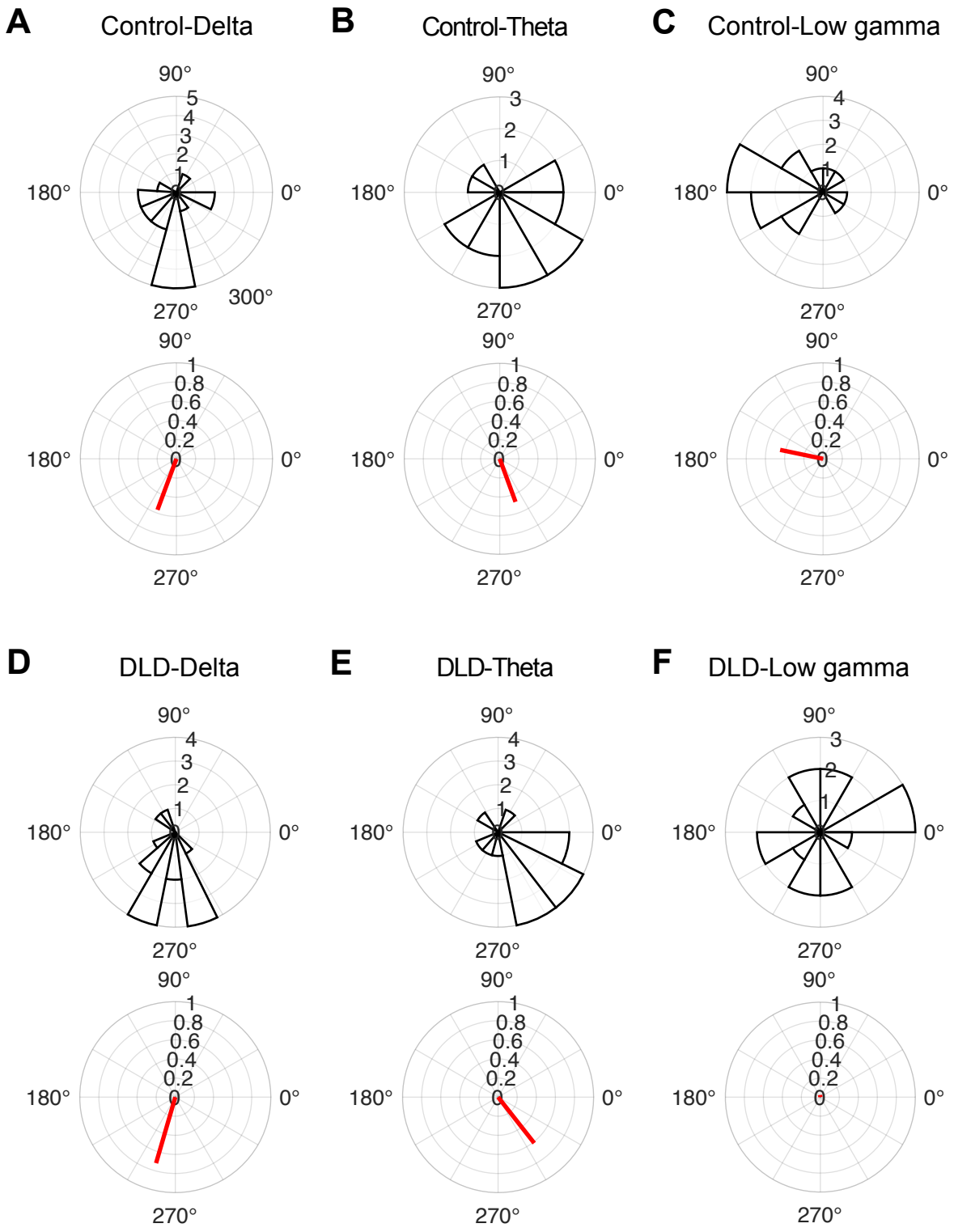


Figure 2. Entrainment phase distribution across children in each group (rose plots) and group resultant vectors (red lines). Note the absence of phase consistency in the low gamma band for the children with DLD.

3.2. Comparing the strength of phase entrainment between groups

A priori, we had expected the strength of the consistency of phase entrainment to differ by group in the delta band. The length of a *child resultant vector* can be considered as a criterion of the strength of phase entrainment across different trials for that child. Indeed, longer *child resultant vectors* indicate higher consistency of phase entrainment. To compare the strength of phase consistency in control children with children with DLD, we applied Wilcoxon rank sum tests based on the length of the *child resultant vectors* for the two frequency bands showing significant entrainment, delta and theta. Contrary to prediction, there was no significant difference between the two groups for either band (delta: $z = -0.70$, $p = 0.49$, Figure 3A; theta: $z = -0.55$, $p = 0.58$, Figure 3B). We also checked these comparisons after removing outliers. A data point was considered as an outlier if the corresponding power value was greater than a 1.5 interquartile range above the upper quartile or below the lower quartile of the population data in that group. There were also no significant group differences after outlier removal.

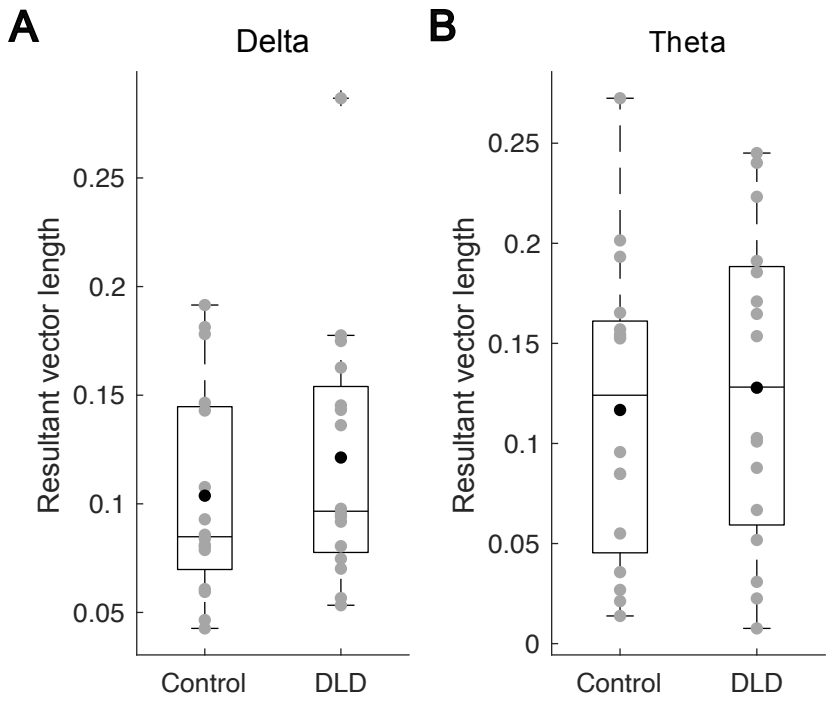


Figure 3. The length of child resultant vectors in the delta (A) and theta (B) bands in the two groups. The grey circles denote the length of the child resultant vectors for individual participants, the black disks denote the mean values for each group, and the horizontal line on each box shows the median.

3.3. Comparing preferred phase and angular velocity between groups

A priori we expected group differences in both preferred phase in the delta band and in angular velocity in the delta band. To compare preferred phase between groups, the Watson-Williams test was applied to the delta and theta band data. The low gamma band was excluded, as it did not show consistent phase entrainment for the children with DLD. The Watson-Williams test assumes von Mises distributions, although this test is fairly robust against deviations from this assumption (Zar, 1999; Berens, 2009). In our data, the phase distribution for both delta and theta bands, as well as for both groups (TD and DLD), followed von Mises distributions (Watson's U2 test, $p > 0.05$). Contrary to prediction, there was no significant difference in terms of preferred phase between the groups, neither in the delta band (Watson-Williams test, $p = 0.84$) nor in the theta band (Watson-Williams test, $p = 0.46$).

We also explored the behaviour of preferred phase across time separately for each group and each frequency band by computing angular velocities. Here the gamma band data could be included, as angular velocity (see Section 2.4) analyses do not require phase consistency. To this end, we calculated the group preferred phase for each frequency band prior to and after the occurrence of a stimulus (stimuli 4th–8th) in the entrainment period over the time interval of –500 ms to 500 ms. This time interval corresponds to two repetitions of the syllable "ba". Figure 4 shows the group preferred phase for the two groups and for delta, theta, and low gamma bands. For both groups, delta-band and theta-band group preferred phases appear to follow a quasiperiodic pattern, with a frequency of around 2 Hz for the delta band (angular velocity of 4 rad/s, see Figure 4A) and a frequency of around 4 Hz for the theta band (angular velocity of 8 rad/s, see Figure 4B). For the low gamma band, however, the pre-stimulus group preferred phase for children with DLD appears to be different from that of the control children. We therefore employed a two-sample t-test to find the time bins with a statistical difference in terms of angular velocity. The result suggested a significant difference between the two groups

over the time interval of –440 ms to –240 ms (two-sample t-test, $p = 0.017$, see Figure 4C). This suggests that phase changes differently across time for the children with DLD, in the gamma band only.

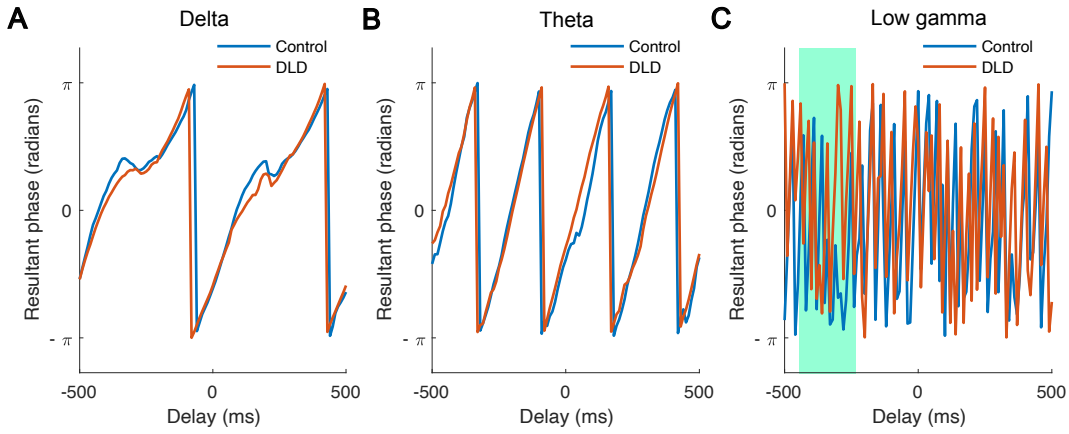


Figure 4. The group preferred phase (radians) as a function of the delay (ms) relative to the onset of "ba" stimuli. The blue and red curves are related to control (N = 16) and DLD group (N = 16), respectively. Both curves appear to follow a quasiperiodic pattern with a frequency of around 2 Hz in the delta band (A) and a quasiperiodic pattern with a frequency rate of around 4 Hz in the theta band (B). The curves related to the low gamma band (C) reveal a pre-stimulus difference between the two groups. In particular, the pre-stimulus angular velocity in this band over the time interval of –440 ms to –240 ms (marked with green colour) differs significantly by group.

3.4. Comparing ERPs between groups

Figure 5 depicts the ERP curves for each group. We calculated the ERP in the time interval of –500 ms to 500 ms separately for each group and each frequency band, as described in Section 2.6. Although differences between group could be expected in terms of pre- and post-stimulus peaks in ERPs from visual inspection of Figure 5, no significant group difference was found in any frequency band (delta, theta, low gamma).

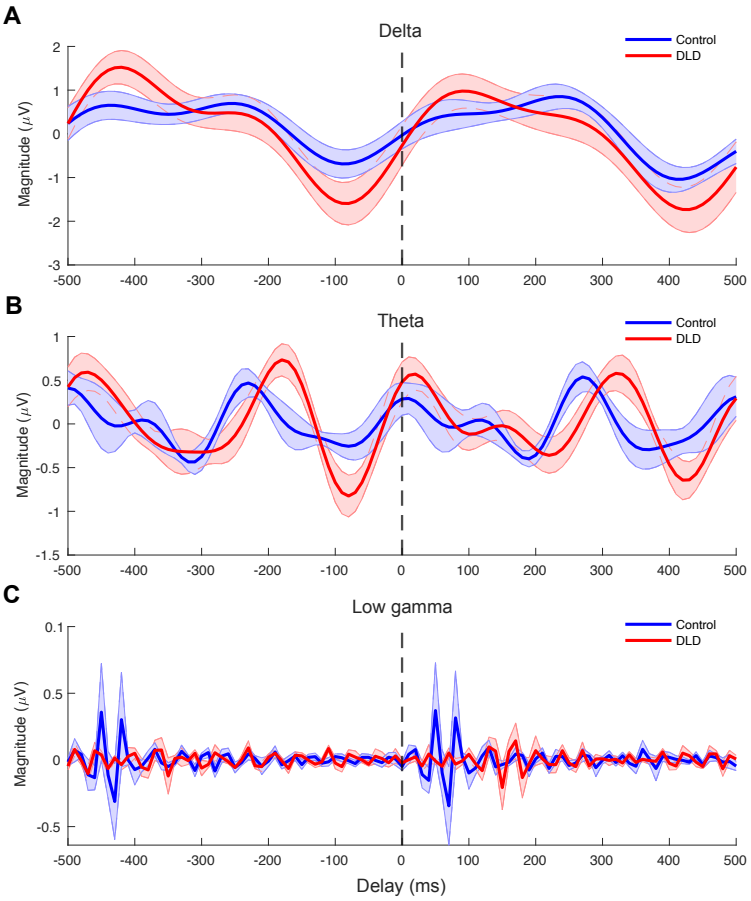
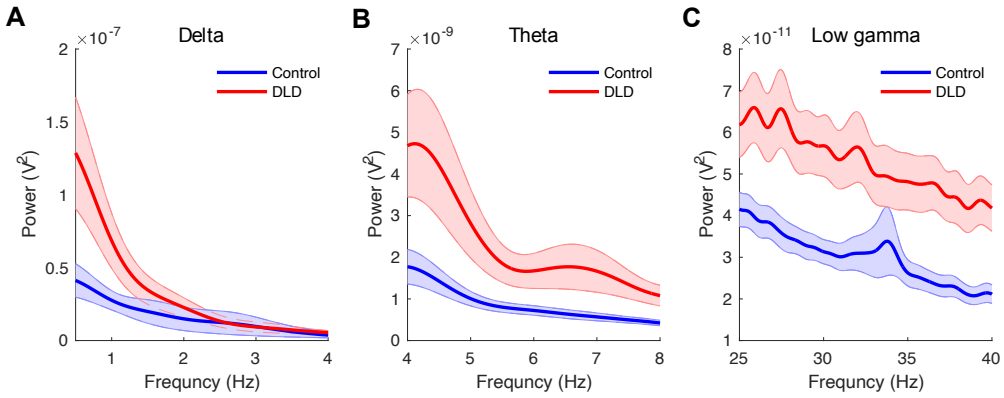


Figure 5. Event related potentials for delta (A), theta (B) and low gamma (C) bands. The blue and red curves denote the ERPs for control and DLD groups, respectively. The shaded areas denote the standard error of mean for each group.

3.5. Comparison of band-power between groups

To compare the band-power between groups, Wilcoxon rank sum tests were applied for each frequency band after removing outliers. We calculated the average power over the entrainment period in each frequency band separately for each group, using steps described in Section 2.7. Figure 6 shows the average power versus frequency separately for each group and for delta (see Figure 6A), theta (see Figure 6B), and low gamma (see Figure 6C) bands. A significant difference between control children and children with DLD was found in theta ($z = –2.01$, $p = 0.04$; Figure 6B; outliers [control] N = 3, outliers [DLD] N = 2;) and in low gamma ($z = –2.64$, $p = 0.008$; Figure 6C; outliers [control] N = 2, outliers [DLD] N = 3) bands. However, there

451 was no difference in band-power for the delta band (z = –1.45, p = 0.15; Figure 6A; outliers
452 [control] N = 2, outliers [DLD] N = 2).
453



454
455 **Figure 6. Delta-band (A), theta-band (B), and low gamma-band (C) power in the**
456 **entrainment period.** The shaded areas denote the standard error of the mean for each group.
457 Please note the numerical power differences in the y axis by band.
458

459 **3.6. Comparing cross-frequency PAC between groups**

460 *A priori*, we had tentatively predicted a group difference in theta-gamma PAC between groups.
461 Delta-theta PAC, delta-low gamma PAC and theta-low gamma PAC were computed over the
462 entrainment period separately for each group by computing the respective modulation indices.
463 We then applied Wilcoxon rank sum tests to compare these modulation indices between the
464 two groups. No significant differences were found for delta-theta PAC (z = –1.34, p = 0.18,
465 Figure 7A) or delta-low gamma PAC (z = –1.07; p = 0.28, Figure 7B), nor for theta-low gamma
466 PAC (Wilcoxon rank sum test, z = –0.85; p = 0.40, Figure 7C). Similar non-significant results
467 were found following outlier removal. Contrary to prediction, therefore, theta-gamma PAC was
468 not different in the children with DLD for the rhythmic syllable repetition task.
469

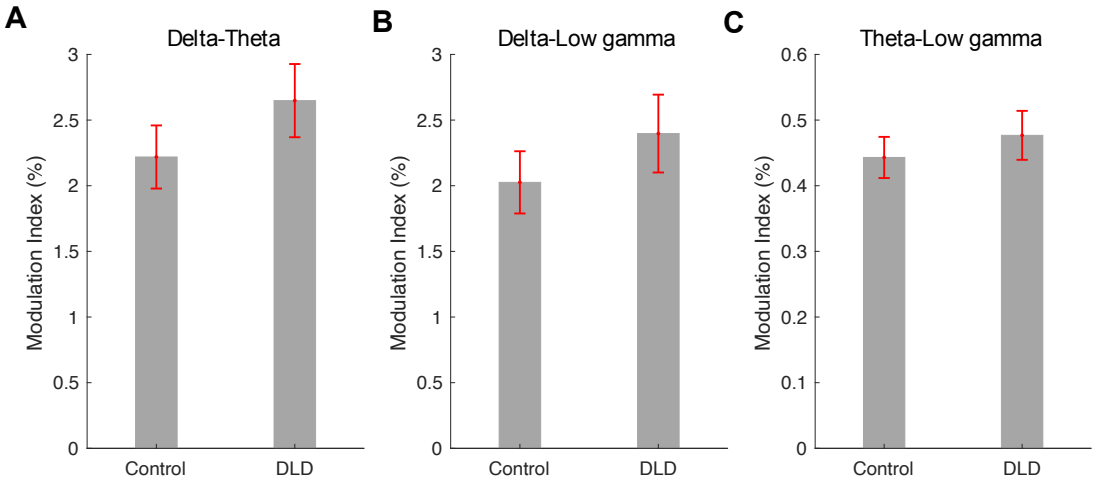


Figure 7. Modulation index was used as a measure for delta-theta (A), delta-low gamma (B), and theta-low gamma (C) phase-amplitude coupling. The error bars denote the standard error of mean.

3.7. Comparing cross-frequency PPC between groups

Phase-phase coupling was also compared between groups. Delta-theta PPC, delta-low gamma PPC and theta-low gamma PPC were computed over the entrainment period separately for each group, as described in Section 2.9. We then applied two-sample t-tests to compare the two groups. As for PAC, no significant group differences were found, delta-theta PPC (p = 0.89, Figure 8A), delta-low gamma PPC (p = 0.26, Figure 8B), theta-low gamma PPC (p = 0.11, Figure 8C). Group PPC remained statistically equivalent after outlier removal.

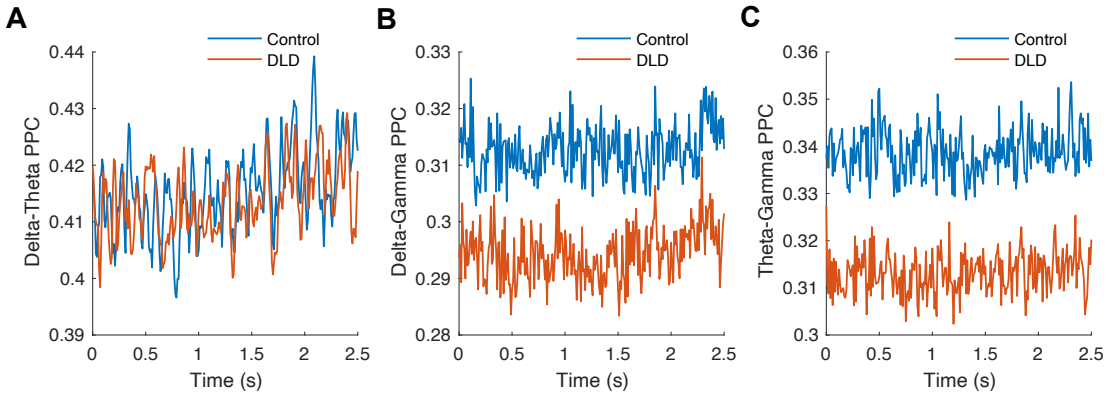


Figure 8. Delta-band (A), theta-band (B), and low gamma-band (C) phase-phase coupling. The blue and red curves denote the PPCs for control and DLD groups, respectively.

Finally, we calculated the phase difference (see Equation 8) for delta-theta, delta-low gamma, and theta-low gamma oscillations for each child at the onsets of stimuli (4th–8th) during the entrainment period. The resultant phase difference for each group was then computed by averaging across the phase differences obtained for children in that group, and separately for delta-theta, delta-low gamma, and theta-low gamma oscillations (see Figure 9). We applied Watson-Williams tests to compare the group resultant phases between the groups. All measures for both groups (TD and DLD) followed von Mises distributions (Watson's U2 test, $p > 0.05$). In this case, low-frequency resultant phase (delta and theta) did show significant differences between the two groups when coupling with the higher-frequency gamma band, delta-low gamma ($p = 0.04$), theta-low gamma ($p = 3.28\times10^{-4}$). Accordingly, the synchronisation of low-frequency phases (delta, theta) with high-frequency phase (low gamma) appears to operate differently for children with DLD. These differences are clearly visible in Figure 9. However, regarding delta-theta resultant phase differences, the two groups were not significantly different ($p = 0.11$).

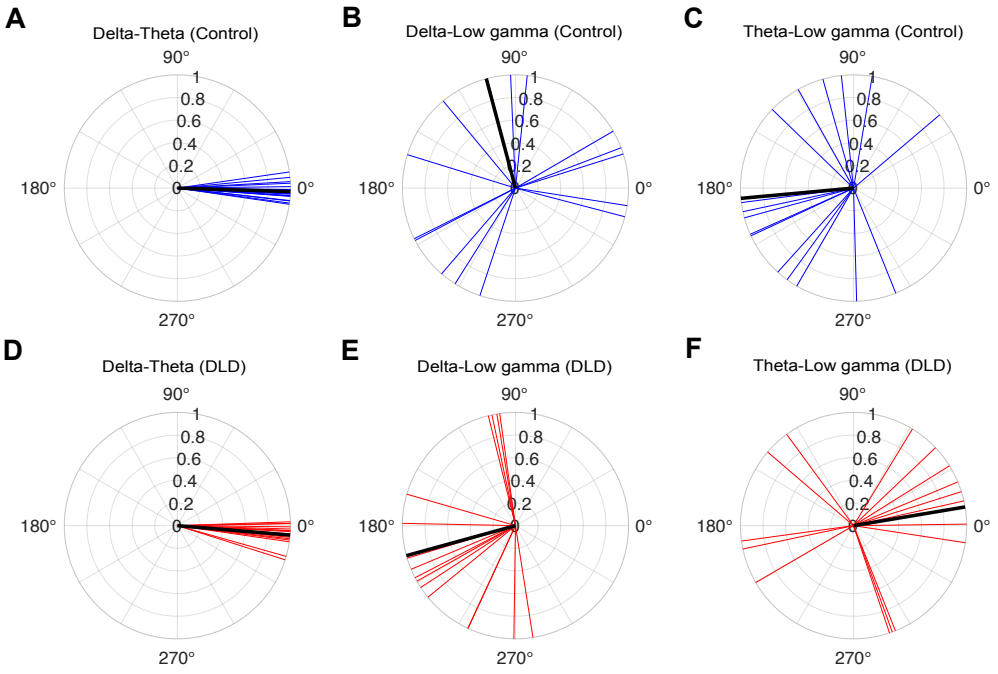


Figure 9. Delta-theta, delta-low gamma, and theta-low gamma resultant phase differences at the onsets of stimuli (4th–8th) during the entrainment period for control (A, B, C) and DLD (D, E, F) children. The blue and red vectors denote the resultant phase difference for

individual children in control and DLD groups, respectively. The black vectors show the group resultant phase difference.

4. Discussion

The current study investigated the ARR hypothesis of developmental disorders of language at the neural level, by examining different mechanistic aspects of the oscillatory encoding of a highly constrained rhythmic speech input, the repetition of the syllable "ba" at a 2 Hz rate. Despite the similarities in behavioural and sensory rhythm processing tasks that have been documented for children with DLD and children with dyslexia (Richardson et al., 2004; Corriveau et al., 2007; Thomson & Goswami, 2008; Corriveau & Goswami, 2009; Fraser et al., 2010; Goswami et al., 2011; Beattie & Manis, 2012; Goswami et al., 2013; Cumming et al., 2015a,b; Richards & Goswami, 2015, 2019), the neural mechanisms that were atypical for the children with DLD in this paradigm were quite different from those previously documented for children with dyslexia (Power et al., 2013; Keshavarzi et al., 2022). In particular, the expectation of impaired delta band responses for children with DLD was not supported. The children with DLD showed equivalent phase entrainment strength to TD children in the delta band, they showed equivalent preferred delta phase, and they showed equivalent delta band angular velocity. ERPs in the delta band did not differ by group, and nor did delta band power in the entrainment period. Similar results were found for the theta band regarding these parameters, with the exception that theta band power during the entrainment period was significantly elevated in DLD children (Figure 6). These data suggest that the delta and theta band mechanisms that are thought to underpin rhythmic speech encoding are operating in an essentially similar way in the DLD brain when compared to the brains of age-matched TD children, at least in the rhythmic syllable repetition task paradigm.

By contrast, low gamma band oscillatory responses in the rhythmic syllable repetition task showed some differences in the DLD brain. Low gamma band phase entrainment in the

children with DLD was absent in the current paradigm, suggesting that phase consistency in this band was not attained despite the highly predictable input. There was also a faster angular velocity in the gamma band for the DLD children, suggesting that gamma phase changed differently over time when compared to TD children. Furthermore, when low-frequency phases had to synchronise with high-frequency gamma responses in our task, there were group differences in resultant phase compared to the TD controls, for both delta-low gamma and theta-low gamma coupling (Figure 9). Although the strength of phase-amplitude and phase-phase coupling in the two groups of children was not significantly different (Figure 8), the resultant phase differences suggest that this similar synchronisation strength was generated by a different underlying relationship between low-frequency phase and high-frequency phase in the DLD brain. One way of interpreting the consequences for speech processing is that the interactions or connectivity between neural networks at these different rates is impaired, namely the theoretical notion of *communication through coherence* (Fries, 2015). Fries (2009, 2015) proposed that neuronal inputs that arrive at random phases of the excitability cycle of related rhythmic neuronal responses will have lower effective connectivity, with consequently negative effects for cognition. Fries used examples from gamma band activity during visual cognition (primarily in animals) to illustrate communication through coherence, but the fundamental principle that he was proposing is relevant here, which is that the synchronization of brain oscillations is a key mechanism for information encoding and perceptual binding. In our data, the gamma rhythmic response to the highly predictable speech input does not attain phase consistency, making it more difficult for low-frequency responses (delta, theta) to synchronise their phases to gamma phase. This could suggest reduced coherence during speech processing, negatively affecting the precision of linguistic encoding. In communication through coherence theory, gamma-band coherence enables communication to be not only effective but also precise. Although some aspects of our data suggest that the low-frequency

responses (delta and theta) and their phase relations are operating in the same way as for TD children during the rhythmic speech task (with equivalent PAC and PPC strength), an optimal phase relation with gamma band responses in the temporal domain cannot be established, thereby impairing speech processing. This is mechanistically quite different from our previous findings with children with developmental dyslexia in this paradigm, where delta phase was atypical (Power et al., 2013; Keshavarzi et al., 2022). Nevertheless, for dyslexic children also, PAC mechanisms were explored and appear to be intact (delta-beta PAC, see Power et al., 2016; Keshavarzi et al., 2024).

A related view of the importance of gamma band processing for speech encoding concerns the parsing of phonemic information in the speech signal (Giraud & Poeppel, 2012; Poeppel, 2014). For example, it is known that speech contains crucial information required for successful linguistic decoding of the signal that is carried at multiple temporal scales, such as intonation-level or prosodic information at the scale of 500–2000 ms (argued to be encoded by delta band responses), syllabic information that is correlated to the acoustic envelope of speech at the scale of 150–300 ms (encoded by theta band responses), and rapidly-changing featural information at the scale of 20–80 ms (encoded by gamma band responses). One mechanism for encoding is suggested to be the sliding and resetting of neural temporal windows that match information in the signal via oscillatory responses, implemented as phase locking to key features of the input via phase-resetting of the intrinsic oscillations. While more recent work has shown that acoustic feature information is also encoded by the slower delta and theta band responses (for adults, children and infants, see Di Liberto et al., 2015, 2018, 2023), the idea that gamma band responses encode phoneme-relevant information has been supported by transcranial alternating current stimulation (tACS) studies using gamma band stimulation. For example, Marchesotti et al. (2020) reported that 20 minutes of tACS stimulation at ~30Hz significantly improved phonemic awareness in a behavioural task for French adults with

dyslexia. The data presented here may suggest that the ~30 Hz response to acoustic feature information in speech is atypical in the DLD brain, which could impair some aspects of phoneme awareness. However, given that most phonetic features are present in natural speech over much longer time windows (see Menn et al., 2023), and given that the cortex maintains representations of phonetic information even after it is lost from sensory input (Gwilliams et al., 2022), the idea that the gamma band differences reported here prevent children with DLD from representing phonemic information seems unlikely. Accordingly, we prefer to interpret the data using communication through coherence theory. Our findings appear to indicate reduced co-ordination of low-frequency oscillatory phase with gamma band responses in the DLD brain, reflecting altered neuronal network interactions during linguistic processing that may affect encoding precision and thereby impair speech perception at all phonological levels, not only the phoneme level. Converging studies with natural speech rather than the rhythmic speech paradigm utilised here are required to assess these different possibilities.

The gamma band differences reported here are also interesting with respect to an alternative auditory theory of DLD, the rapid auditory processing (RAP) theory (Tallal, 1980, 2004). RAP theory proposed that basic auditory processing of brief, rapidly successive acoustic changes is impaired in children with DLD, drawing on data from a nonspeech task using brief (75 ms) pairs of tones that differed in pitch and succeeded each other rapidly in time (e.g., gaps of 8, 15, 30, 60 ms, see Tallal & Piercy, 1973). The children's task was to decide whether a high tone preceded a low tone in a pair or vice versa. Children with DLD were worse than TD controls in the tone task when the gap between stimuli was short, but were not different to TD controls when the gaps were longer (greater than 150 ms). Regarding speech processing, RAP difficulties were expected to affect phonetic discrimination (thought to require processing at rapid timescales around 40 ms, for example discriminating /b/ from /d/) via impaired discrimination of formant transitions (rapid acoustic changes in frequency and intensity).

606 Further, for children with DLD, intervention studies based on RAP can lead to improved
607 language skills (e.g., Stevens et al., 2008, although see also Gillam et al., 2008).

608 Regarding neural data with DLD children, a nonspeech MEG study by Heim and
609 colleagues indicated altered temporal organization of gamma-band oscillatory activity when
610 children with DLD processed rapid tone sequences (Heim et al., 2011). Children listened to
611 pairs of tones separated by 70 ms, and judged the pitch of the second tone. Heim et al. reported
612 significantly reduced amplitude and phase-locking of early (45–75 ms) oscillations in the
613 gamma-band range (29–52 Hz) for the second stimulus. ERP studies of infants at family risk
614 for DLD using brief rapidly-presented tones have reported differences in auditory evoked
615 potentials indexing change detection compared to TD infants, and in resting state gamma
616 power, which in some studies predicted later language skills (English, language measured at 3,
617 4 and 5 years, Benasich et al., 2008; Choudhury & Benasich, 2011; Gou et al., 2011; Italian,
618 language measured at 20 months, Cantiani et al., 2016, 2019). Of particular interest regarding
619 the current dataset, an investigation of the same English-exposed infants studied by Choudhury
620 and Benasich (2011) at 6 and 12 months showed that the infant change detection response for
621 rapidly-presented tone pairs was mainly determined by low frequency power and phase-locking
622 responses (2-3 Hz, Hamalainen et al., 2019). Regarding the ARR hypothesis, it is logically
623 possible that while children with both DLD and dyslexia share a difficulty in processing AE
624 rise times and show difficulties in behavioural speech rhythm tasks, the rates of temporal
625 integration that are impaired neurally may differ for each developmental disorder. These prior
626 infant studies and the current gamma band data are consistent with Tallal's (2004) claim that
627 auditory processing in temporal integration windows of around 40ms (25 Hz) is adversely
628 affected in children with DLD. However, low frequency phase-phase mechanisms may
629 contribute to this impaired processing. Further RAP studies based on processing natural speech

rather than pairs of tones are required to investigate this further, and to explore potential links to phoneme representation.

The current study has a number of limitations. Firstly, the rhythmic speech task is not a natural language task. Further investigation of the ARR hypothesis using natural speech listening tasks is required in order to establish whether the current gamma band and resultant phase differences are present during natural language processing. Second, the sample size is small, with 16 DLD children. However, it is representative of the existing neural DLD literature (e.g., Heim et al. 2011 studied 17 children with DLD, Choudhury & Benasich 2011 studied 17 infants at risk for DLD). This reflects the difficulty of engaging families regarding neuroimaging.

In conclusion, here we find that the 9-year-old DLD brain does not show the delta-band deficits predicted by TS theory to underpin impaired rhythmic speech processing. Instead, the children with DLD exhibited an absence of phase consistency in the gamma band when processing highly predictable rhythmic speech, and showed different angular velocity in the gamma band compared to TD children. The children with DLD also showed significantly greater gamma and theta power during the task, and a difference in resultant phase when low-frequency phases (delta, theta) coupled with low gamma. These latter data are consistent with some prior neural nonspeech investigations of children with DLD. Overall, the data suggest reduced coherence between slower and faster networks during speech processing, which would negatively affect the precision of linguistic encoding. Further studies employing natural continuous speech are required to investigate this interpretation in more depth.

Data and Code Availability

Data and code will be made available on request.

Author contributions

M.K. conceptualisation, methodology, data collection, data analyses, visualisation, writing – original draft; S.R. data collection, writing – review & editing; G.F. data collection, writing – review & editing; L.P. data collection, writing – review & editing; U.G. funding acquisition, project administration, supervision, conceptualisation, methodology, writing – original draft.

Conflict of interest

The authors declare no conflicts of interest.

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