

*Original Research*

# Pure-Tone Frequency Discrimination and Auditory Functional Connectivity in Developmental Dyslexia

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## Abstract

**Background:** In previous studies, children with developmental dyslexia (DD) have been found to exhibit alterations in auditory sampling within the delta/theta and low-frequency gamma bands in auditory cortical areas during the initial processing stages, which affects the development of phonological skills. It has been suggested that auditory frequency discrimination measures sensory processing in language disorders such as DD. However, it is unclear how the pure-tone frequency discrimination task can detect abnormalities in functional connectivity in DD. **Methods:** We investigated local and global topological properties of functional networks in electroencephalographic (EEG) frequency bands from  $\delta$  to  $\gamma 2$  based on a small-world propensity (SWP) model. This was done in both groups during pure-tone frequency discrimination. **Results:** Auditory  $\alpha$ -,  $\beta$ -, and  $\gamma 1$ -networks in the DD group were more integrated and less segregated than those of the control group. They were also not as functionally specialized, as indicated by larger deviations in characteristic path lengths and smaller deviations in clustering. The balanced segregation and integration (moderate clustering and path length) observed in the control group's  $\gamma 2$ -network may explain the optimal structure underlying their better performance. In the low-tone auditory  $\theta$ - and  $\gamma 2$ -frequency networks, the DD group, when compared with controls, lacked hubs in the inferior frontal cortex (IFC) and parietal connectivity to sensory areas. In the control group, however, the superior parietal lobes (SPL) mediated sensory connections. In the high-tone auditory network, only the controls had strong hubs in the right sensorimotor/auditory cortex ( $\delta$ -frequency), bilateral IFC ( $\gamma 1$ ), and bilateral anterior temporal cortex (aITG,  $\gamma 2$ ), while the main hubs in the DD group were only in the left hemisphere. In the  $\gamma 1$  (high-tone) and  $\gamma 2$  (both tones) networks, controls showed strong right frontal-parietal-sensory hubs, which were lacking in the DD group during the task discrimination. **Conclusion:** The impairment in low-tone discrimination in the DD group is due to a lack of SPL-prefrontal connectivity within the auditory network. For high-tone discrimination, the DD group showed engagement of only the left-sided auditory network, with bilateral prefrontal recruitment ( $\delta$ -network). In contrast, the SPL in the control group integrates sensory input for tone prediction, establishing tone-specific sensory/auditory connections with left prefrontal involvement ( $\delta$ -network). Lower predictability for high tones in the DD group led to more localized processing with prefrontal influence. Overall, reduced frontotemporal connectivity in the DD group may indicate poorer auditory processing. This is likely due to impaired prefrontal-sensory communication and reduced interhemispheric auditory communication, which may underlie perceptual-cognitive biases in tone frequency discrimination.

**Keywords:** developmental dyslexia; auditory processing; functional connectivity

## 1. Introduction

Developmental dyslexia (DD), a reading and spelling disorder affecting children with normal intelligence, is primarily linked to difficulties in processing speech sounds, known as phonemic awareness [1–5]. Research also suggests that individuals with dyslexia may struggle with both speech and non-speech sound perception [6–8].

Auditory skills, which are crucial for language development, involve two distinct but related abilities: auditory recognition and auditory discrimination. Auditory recognition refers to the ability to identify and remember sounds, an early step in vocabulary building and language comprehension that focuses on overall sound processing rather than fine distinctions. In contrast, auditory discrimination involves a more refined level of listening. It is the ability to perceive and differentiate between subtle varia-

tions in sounds, such as pitch, tone, frequency, or duration. This skill is particularly critical for developing phonological awareness in young children—the capacity to hear and manipulate the sounds that make up words. Consequently, without strong auditory discrimination, children may struggle to distinguish between similar sounds, which can lead to difficulties in reading, spelling, and articulate speech.

Studies reveal links between auditory processing impairments and differences in the auditory cortex [5,9,10]. A prominent hypothesis suggests that dyslexia originates from a deficit in processing rapid auditory changes, which in turn leads to phonological and reading problems. Numerous studies indicate a fundamental auditory processing deficit in dyslexia [8,11–16], specifically concerning the processing of rapid temporal changes that differentiate consonants within a language [17–19]. The phonological prob-



lems associated with dyslexia may result from difficulties in processing rapid speech timing, a notion supported by the observed link to musical rhythm processing [20]. However, research has not definitively confirmed the precise nature of this auditory deficit—such as whether it is specific to speech or encompasses more general acoustic processing [11], or if it is limited to rapid temporal processing [8,12–17] versus broader spectrotemporal processing [18]. A core deficit in dyslexia is the ability to process and manipulate individual speech sounds (phonemes) [21,22].

Dyslexia is not solely a language-based disorder; it also has fundamental auditory processing underpinnings, with both temporal and tone processing affecting phonemic awareness [23–26]. These auditory deficits extend beyond speech, impacting the processing of various non-speech sounds, and their connection with linguistic abilities suggests shared neural pathways and processing mechanisms. Research highlights the significant role of tone processing in understanding dyslexia. Beyond temporal processing, studies also link phonemic awareness to pitch processing. Since speech sounds have distinct frequencies and pitch perception reflects frequency, it is hypothesized that both phoneme and pitch perception may rely on a common mechanism of frequency discrimination. Consequently, pitch sensitivity is relevant to reading because phonemic awareness has been shown to correlate with performance on pitch-related tasks [27]. Furthermore, the fact that specific language impairment (SLI) is associated with deficits in pitch discrimination strengthens the connection between musical pitch ability and phonemic awareness [28–31].

The hypothesis of a common neural substrate for linguistic sound and musical processing, which could explain both dyslexia and tone-deafness, is supported by evidence that the auditory deficit affects both speech and non-speech perception, as well as by the established link between phonemic awareness and pitch perception [19,21,22]. This framework suggests that understanding the relationship between developmental language disorders and tone-deafness is crucial for future research.

Frequency discrimination, a fundamental auditory skill [32], is significantly affected in children with auditory processing challenges, particularly auditory processing disorder (APD). Both APD and SLI impact frequency discrimination and the detection of frequency differences above a certain threshold level ( $\Delta f$ ). APD involves difficulties processing auditory information despite normal hearing, while SLI involves language acquisition difficulties without other underlying issues. When tested at various levels of difficulty, with frequency differences ( $\Delta f$ ) ranging from small (20 Hz) to large (150 Hz), children with APD, SLI, or both performed better at larger frequency differences [32,33]. For children with SLI, the impairment primarily affected the discrimination of larger, easier-to-detect frequency differences, which suggests a higher-level cognitive compo-

nent. In contrast, APD primarily impacted the discrimination of smaller, more-difficult-to-detect frequency differences, which suggests a deficit in a perceptual component. This highlights the importance of examining frequency discrimination above the difference threshold ( $\Delta f$ ), as this is more relevant to everyday listening [32,33].

Research on pure-tone frequency discrimination in typically developing school-aged children [34] reveals significant variability among them. Some children could detect very small frequency differences (around 1% of a 1000 Hz base frequency [34]), while others required much larger differences (around 10%) in a bimanual presentation [33]. Children with dyslexia have demonstrated impaired tone discrimination, with a difference threshold of up to 15% (e.g., distinguishing 0.9 kHz from 1.05 kHz [33]). This impairment is even greater in dichotic presentations, where a 20% impairment has been reported [35].

### *1.1 Neuroimaging Studies of the Auditory Functional System*

Crucial for cortical sound processing [36], the auditory network encompasses several key brain regions: the primary and auditory association cortices, both residing in the superior temporal cortex. This network reaches beyond the temporal lobe to include portions of the parietal lobe, the posterior superior and middle temporal cortices, and the temporal pole. Additionally, subregions of the inferior parietal cortex (IPL) and the temporal-parieto-occipital junction contribute. These auditory regions functionally connect with frontal areas, alongside the primary somatosensory and middle occipital cortices [37]. The parietal and frontal lobes handle the final stages of sound processing. The primary auditory cortex (A1) is the main site for simplified auditory processing, handling simple sounds like unchanging single-frequency tones. However, complex sounds, such as noise bursts or speech, require more extensive processing that spans A1 and other auditory areas. Within this network, neural frequency tuning seems to mirror perceptual qualities, such as tone, rather than just the sound stimulus's physical traits [38,39]. Significantly, the right auditory cortex is more sensitive to sound's spectral details (tonality), while the left auditory cortex shows greater attunement to rapid temporal dynamics, evident in speech [40]. The auditory cortex's belt region, particularly its caudal fields, forms significant connections with the secondary visual [41] and secondary somatosensory cortices [42,43]. Moreover, tonality is represented not only within the auditory cortex but also in the rostromedial prefrontal cortex [44].

Individuals with dyslexia often show auditory processing differences, marked by altered neural activity in the auditory cortex. These manifest as prolonged auditory evoked potentials (P1, N1, P2) and atypical mismatch negativity (MMN) responses. The MMN responses are elicited solely by the brain's ability to distinguish deviant from standard stimuli, regardless of focused attention. The

brain's MMN, a response to changes in repeated sounds, serves as an indicator of central auditory processing, relying on both novel acoustic input and the memory of preceding sounds [45,46]. The finding of reduced electroencephalographic (EEG) mismatch responses in dyslexia has led to their widespread application in auditory and cognitive research [47–50]. Both adults and children demonstrate MMN responses even to subtle frequency differences [51]. An insensitivity to repetition could explain the abnormal mismatch response in individuals with dyslexia [25,52], especially for speech, by disrupting repetition-induced neural habituation [53]. Within a predictive coding framework [54,55], this phenomenon highlights the brain's continuous model updating for stimulus consistency, a process fundamental to perception and learning [45]. In children, the P1-N1-P2 acoustic complex shows age dependency and reflects sensitivity to frequency changes, capable of detecting variations as minor as 1% of the base frequency. This complex, recorded at temporal electrodes, holds significant importance for studying auditory cortex activity related to frequency discrimination. Magnetoencephalography (MEG) studies, specifically, have documented delayed brain responses (P1, N1, P2) in individuals with dyslexia, suggesting an underlying auditory and sensory processing deficit [51]. Further, recent research points to structural and functional changes within the auditory cortex in dyslexia [37,56–58].

Expanding on connections with other disorders and insights from dyslexia research, the auditory processing hypothesis for dyslexia proposes that the auditory difficulties experienced by individuals with dyslexia aren't confined to complex sounds [56–61]. Instead, it suggests they may also encounter challenges discriminating simple tones that typically demand minimal auditory processing. These anticipated difficulties with both pure tones and complex sounds would inherently require broad engagement from brain networks, including both primary and associative auditory cortices [62]. Brain abnormalities are evident in Neuroimaging studies of disorders such as dyslexia (a reading and language impairment), tone-deafness (congenital amusia), and developmental prosopagnosia (face recognition impairment). These issues notably affect the inferior frontal cortex (IFC) and the crucial pathway linking the frontal, parietal, and temporal lobes [63,64]. A shared underlying pathology, like early neuronal migration disorders or problems with axon outgrowth or guidance during development, could be at play. This common root might result in subtle malformations across multiple brain networks, manifesting as various functional impairments: a more pronounced effect on the left hemisphere for dyslexia, the right for tone-deafness, and more widespread regions for prosopagnosia. Functional magnetic resonance imaging (fMRI) and electrophysiology investigations have identified brain abnormalities within the auditory cortex of children presenting with SLI and APD. However, MMN studies have yielded

inconsistent results: some indicate reduced MMN amplitude in SLI children who performed poorly on a gap detection task [65,66], while others report no significant differences. Notably, children with both SLI and APD displayed the weakest MMN responses [60,67]. Anomalies in the P1, N1, and P2 components have also been observed, indicative of immature or delayed auditory pathway development.

While control subjects typically show a right hemisphere preference for tone processing, this is frequently absent in individuals with dyslexia [35]. Compensated dyslexia, even with typical speech lateralization, might involve less specialized general auditory lateralization [64]. Furthermore, the medial temporal lobe plays a supportive role, enabling individuals with dyslexia and other procedural memory deficits to sustain performance on serial reaction time tasks [68].

The reliability of neural findings, particularly MMN evidence concerning reduced automatic speech discrimination in dyslexia, remains a subject of ongoing debate [51]. To reconcile these conflicting outcomes, future research should differentiate between speech perception and general regularity detection and, critically, establish a stronger link between neural measures, behavioral performance, and broader language difficulties [67]. Ultimately, dyslexia is best characterized by a reduced capacity to learn and effectively utilize short-term statistical regularities, which are essential for enhancing perception and minimizing processing costs.

It's been proposed that graph theory [69] be applied to brain networks, offering another perspective for understanding auditory processing in DD [70]. In functional brain networks, unusual graph metrics often point to changes in aspects like path length, clustering coefficients, or how hubs are distributed. Crucially, researchers have observed that DD involves both altered graph metrics and changes in the overall network structure. This includes a disrupted balance between local specialization and global integration within the brain's networks. By using graph theory to analyze functional connectivity data, we can uncover precisely how different brain regions interact and organize themselves to support cognitive functions. This powerful approach could illuminate the specific ways network development is disrupted in DD, paving the way for deeper understanding and potential interventions. This research considers to studying auditory processing in children with DD to explore its developmental relationship with different frequency networks.

### *1.2 Hypotheses and Goals of the Study*

Examining pure-tone frequency discrimination is essential for understanding auditory processing deficits in children with DD. Research suggests that the sharpness of high-frequency components is crucial for speech encoding, recognition, and retrieval [71]. This is particularly relevant in Bulgarian, where consonants exhibit distinct high-

frequency acoustic characteristics depending on whether they precede front or back vowels [72,73].

This study investigates the impact of simple pure tones on auditory discrimination in the DD group, specifically exploring the relationship between tone frequency and neural processing. We further examine brain connectivity in the DD group using graph theoretical analysis and the small-world propensity (SWP) model.

Given that no previous research has investigated how pure-tone discrimination affects functional brain networks in the DD group, a key question emerges: Can the analysis of brain functional networks help us understand the neurophysiological origins of DD, as well as the connection between the functional auditory network and specific local brain hubs? Our primary goal is to determine the variability in brain networks between the control and DD groups during a tone discrimination task.

We hypothesize that the DD group will exhibit altered SWP and reduced connectivity within various frequency-specific brain networks compared to typically developing children. We further hypothesize that these alterations may be modulated by tone frequency. We expect that if differences are found in the functional auditory networks of the DD group, they will be manifested as changes in specific local hubs within certain frequency networks during the auditory task.

## 2. Materials and Methods

### 2.1 Auditory Discrimination of Pure Tones

All participants had hearing thresholds better than 20 dB hearing level (HL) in both ears across standard audiometric frequencies (250 Hz to 8 kHz). With the exception of pure-tone audiometry, all auditory stimuli were digitally created in MATLAB R2018a (MathWorks, Inc., Natick, MA, USA) and transformed into analog signals via a 24-bit sound card at a 44 kHz sampling rate. The stimuli consisted of 50 ms pure tones at 900 Hz (low) and 1050 Hz (high). To ensure smooth onset and offset, 5 ms half-Hanning windows were applied. Tones were presented binaurally through Sound Blaster Z SE speakers powered by a Sound Blaster Z Audio Processor (Cat. No. SB1500, Creative Technology Ltd., Singapore) in blocks. Each block contained 40 low and 40 high tones, presented in a pseudo-random order. The interstimulus interval (ISI) varied between 1.5 and 2.5 seconds during each experimental session (**Supplementary Fig. 1**). Stimuli were calibrated to 60 dB SPL using a sound level meter (Brüel & Kjaer, mod. 2238; MicroPrecision, Grass Valley, CA, USA) in a sound-attenuated booth. Participants responded by pressing the right button for a low tone and the left button for a high tone. The percentage of correct responses was displayed to the experimenter on a computer monitor at the end of each session.

### 2.2 Selection of Groups

Eligibility for this quantitative cross-sectional study was determined through a neuropsychological examination [74] administered to all children. The study included a DD group ( $n = 36$ ; 18 boys/girls;  $103 \pm 3$  months) and a norm-lexic control group ( $n = 36$ ; 20 boys/16 girls;  $102 \pm 4$  months). Both groups were composed of second-grade students. All participants were right-handed [75], native Bulgarian speakers with normal or corrected-to-normal vision and no known neurological or psychological deficits.

### 2.3 Instruments

#### 2.3.1 Participant Groups and Initial Assessment

The dyslexic group was formed by selecting children who demonstrated reading difficulties, specifically scoring below the average range (more than one standard deviation) for their age on measures of reading speed and accuracy from both the Dyslexia & Developmental Dysorthography Assessment Battery-Second Edition (DDE-2) battery [76, 77] and the “Reading Abilities” test [78] (**Supplementary Table 1**). Age-matched children from the same socio-demographic backgrounds, who exhibited typical reading performance within the norms of the DDE-2 and “Reading Abilities” assessments, formed the control group that included no children diagnosed with dyslexia or any co-occurring language disorders.

#### 2.3.2 Diagnostic and Psychometric Measures

A standardized test battery, including the DDE-2 for DD [76,77], “Reading Abilities” tests [78], and the Girolami-Boulinier’s test for non-verbal perception with “differently oriented signs” [79,80], was administered to all children. This battery revealed a wide range of reading and writing difficulties in the children with DD (**Supplementary Table 1**). Additionally, all participants completed Raven’s Progressive Color Matrices [81] to assess nonverbal intelligence.

Psychometric testing included a battery designed for primary school-aged children to assess reading, writing, and phonological awareness [78] (**Supplementary Table 1**). The “Reading Abilities” test battery evaluated reading fluency through reading a 133-word text aloud, and writing was assessed via dictation of 30 sentences with missing compound words. Phonological awareness was evaluated through tasks involving word identification and the omission of a word’s initial sound (“without the first sound-letter”) or final syllable (“without the last syllable”). Differences between the control and DD groups were observed in both the accuracy and speed of test completion (**Supplementary Table 1**). Importantly, all children with DD demonstrated normal nonverbal intelligence quotient (IQ) scores on the Raven test, consistent with age-appropriate norms.

## 2.4 EEG Data Acquisition and Processing

### 2.4.1 EEG Data Acquisition

EEG signals were recorded using a 40-channel wireless system ([https://portal.bpo.bg/bpo-registers/utility-modules/view/BG\\_U\\_2023\\_5703](https://portal.bpo.bg/bpo-registers/utility-modules/view/BG_U_2023_5703)) [82] featuring star-shaped dry gold sensors. Data were sampled at 250 Hz. Electrodes were positioned according to both the 10–10 and 10–20 systems (**Supplementary Fig. 2**), encompassing the following locations: 10/10 (AF3–AF4, F7–F8, FT9–F10, FC3–FC4, FC5–FC6, C1–C2, C5–C6, CP1–CP2, CP3–CP4, TP7–TP8, P7–P8, PO3–PO4, PO7–PO8) and 10/20 (Fz, F3–F4, C3–C4, Cz, T7–T8, P3–P4, Pz, O1–O2, Oz; **Supplementary Fig. 2**) [83,84]. Reference electrodes were placed on the processus mastoidei and the ground electrode was positioned on the forehead. Rereferencing to global median reference was performed online to eliminate the volume conduction [85]. Skin impedance was maintained below 5 k $\Omega$  to ensure good signal quality.

### 2.4.2 EEG Data Preprocessing

Individual trials were segmented into 800-ms epochs locked to stimulus onset. EEG signals underwent bandpass filtering (1–70 Hz; Chebyshev II) and a 50 Hz notch filter was applied. Before we analyzed the data set was band-pass filtered by using a 4th-order Butterworth filter in forward and backward directions to avoid artificial phase shifts in each frequency band:  $\delta$  (1.5–4 Hz),  $\theta$  (4–8 Hz),  $\alpha$  (8–13 Hz),  $\beta_1$  (13–20 Hz),  $\beta_2$  (20–30 Hz),  $\gamma_1$  (30–48 Hz), and  $\gamma_2$  (52–70 Hz). Artifact-contaminated EEG trials (e.g., eye blinks, muscle activity) with amplitudes exceeding  $\pm 200$   $\mu$ V were removed. Each trial also had to meet a signal-to-noise ratio (SNR) criterion [86]. We calculated SNR by dividing the mean signal's peak-to-peak amplitude by twice the standard deviation of the noise. To get the noise for a sensor, we subtracted the average signal from each trial, and then found the standard deviation of these resulting residuals. Only trials meeting these noise removal criteria and correct behavioral responses were used in further analysis.

### 2.4.3 Analyzed Data and Frequency Bands

Following preprocessing, the average number of artifact-free data epochs per condition and subject (mean  $\pm$  sd) was  $34 \pm 5$  for controls and  $25 \pm 8$  for children with DD. To compensate for this imbalance, additional sessions were conducted with children with DD to equalize the number of epochs. The EEG data were further analyzed within the following frequency bands:  $\delta$ ,  $\theta$ ,  $\alpha$ ,  $\beta_1$ ,  $\beta_2$ ,  $\gamma_1$ , and  $\gamma_2$  Hz.

## 2.5 Functional Connectivity

Functional connectivity between brain regions was assessed by computing the weighted phase lag index (wPLI) [87,88]. For all possible pairs of EEG sensors, an adjacency matrix was constructed, with separate computations performed for each time series and for each frequency band.

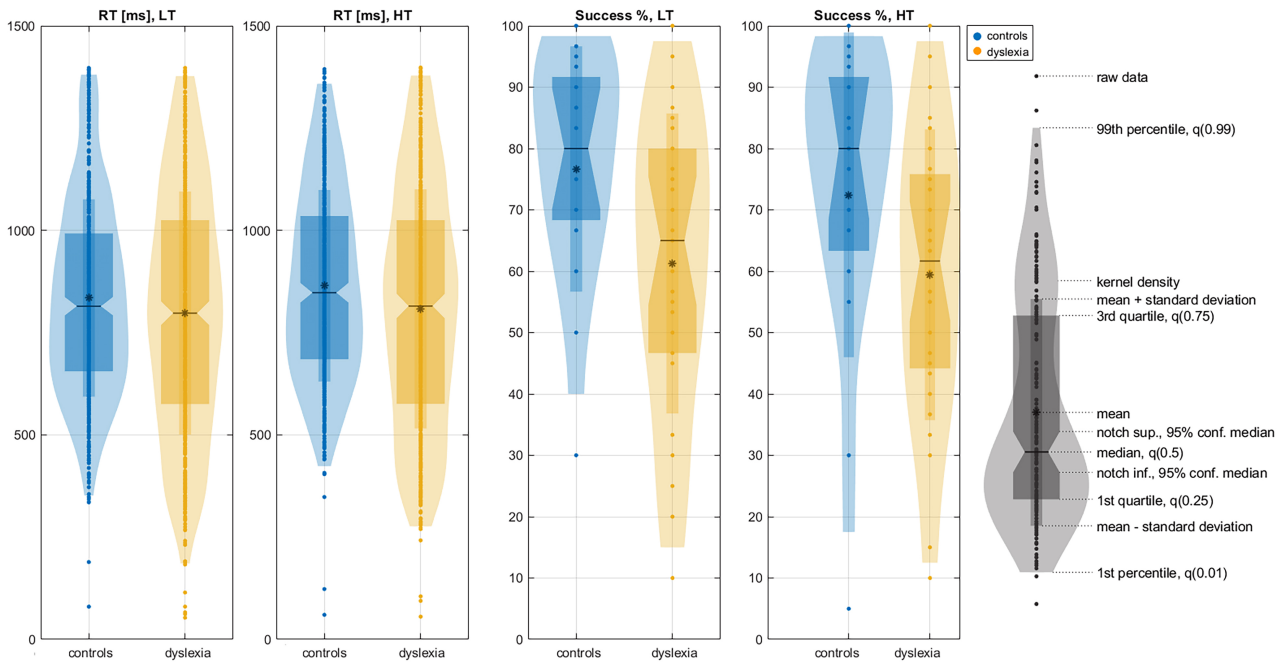
The wPLI quantifies the extent of phase synchronization between two brain regions and reduces the bias introduced by volume conduction [87,88]. The wPLI is calculated as a fraction: the numerator highlights the magnitude of phase lag asymmetry, while the denominator scales the result, ensuring the wPLI value always falls between 0 and 1. A wPLI value near 0 indicates an absence of a consistent phase lag, whereas a value closer to 1 indicates strong and consistent phase synchronization. The wPLI was used to determine the weighted adjacency matrix, which was then applied to calculate the network's SWP [89].

The efficiency of information transfer within the network was quantified using SWP ( $\phi$ ) [89]. SWP is a measure that evaluates a network's clustering coefficient and characteristic path length. Small-world networks are defined by exhibiting both a high clustering coefficient (signifying dense local connections) and a short characteristic path length (indicating efficient global communication). This balance allows for efficient local information processing alongside fast global communication [89].

Small-world characteristics are quantitatively defined by the deviation of the observed network's clustering coefficient ( $\Delta C$ ) and characteristic path length ( $\Delta L$ ) from those of reference random (Crand, Lrand) and regular (Clatt, Llatt) models. In empirical networks, high SWP ( $\phi$ ) values are indicative of robust small-world attributes. Networks with  $\phi$  values near 1 show high Cobs and short to moderate Lobs, or moderate Cobs and short Lobs. Conversely, lower  $\phi$  values (close to 0) correspond to larger deviations ( $\Delta L$ ,  $\Delta C$ ) from null models, indicating a weaker small-world propensity [89].

For each frequency-specific functional network, brain connectivity characteristics, such as SWP ( $\phi$ ),  $\Delta L$ , and  $\Delta C$ , were extracted from each subject's single-trial full wPLI matrix. This encompassed data for specific frequencies and tones (MATLAB functions from the Brain Connectivity Toolbox (<http://www.brain-connectivity-toolbox.net>); [89]). These global measures were then statistically compared using nonparametric methods.

Local node measures included strength (defined as the sum of weights to all other nodes) and betweenness centrality (BC), which is the proportion of all shortest paths in the network that pass through a given node [90–92]. Both strength and BC values were calculated for each node using the weights within the adjacency matrix. These values were then normalized for each subject by dividing a node's score by the average of the local measures across all nodes. Nodes exhibiting high strength (and consequently high BC) are crucial for information processing within the network, as they are involved in a large number of the shortest paths between other nodes [93]. The most crucial nodes in a network are often termed hubs [91]. A key indicator of a network's integration is a higher maximum BC or strength [90,92]. Hubs were defined as nodes whose strength or BC, averaged across all participants, was at least one standard



**Fig. 1. Significantly different behavioral characteristics and reaction times in ms and successful discrimination of tones in % between the control group (blue) and children with DD (yellow).** RT, reaction time; LT, low tone; HT, high tone. In the color plots, the mean is marked by an asterisk (\*), and the median by a dash (—), as indicated by the black violin legend.

deviation greater than the group’s average for that measure. The most important links were then determined by identifying edges whose BC, averaged across all participants, was at least one standard deviation above the mean group edge BC.

The characteristics of individual brain networks, summarized at the participant level for each frequency band and tone, were established by combining anatomical parcellation methods, brain mapping techniques, and connectivity measures using the BrainNet Viewer functional package 1.7 (<http://www.nitrc.org/projects/bnv>, [93]). This package was then used to map nodes (brain regions) and edges (connections) onto a brain template, with their properties represented by size and color.

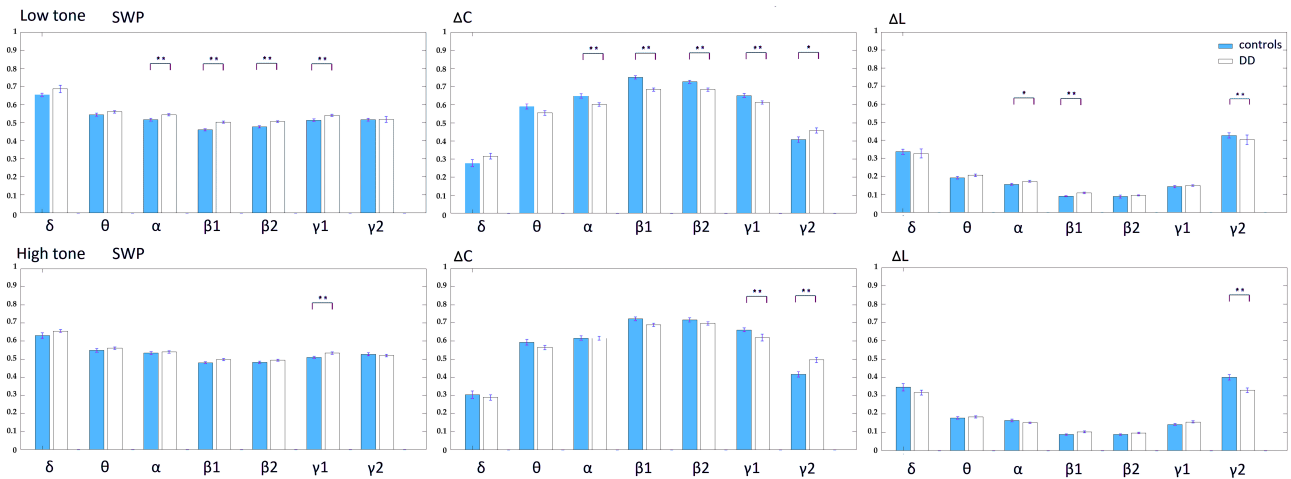
## 2.6 Statistical Analysis

During EEG recording sessions, Kruskal-Wallis non-parametric tests (KW test) were used to compare reaction times and performance accuracy between control and DD groups for each tone condition (low/high).

A multi-way analysis of variance (ANOVA) was conducted on the global measures, with the within-subject factors of tone (low, high) and frequency ( $\delta$ ,  $\theta$ ,  $\alpha$ ,  $\beta_1$ ,  $\beta_2$ ,  $\gamma_1$ ,  $\gamma_2$ ) and the between-group factor of group (control vs. children with DD) [94]. Subsequently, a nonparametric procedure with 1000 permutations was employed for between-group pairwise comparisons of each global SWP measure ( $\phi$ ,  $\Delta L$ ,  $\Delta C$ ) across each frequency band and tone [95,96]. The Bonferroni correction was applied to control the family-wise error rate (FWER) in multiple hypothesis

testing. The adjusted significance level was calculated as  $p = \alpha/3 = 0.05/3 \approx 0.0167$  to account for the three dependent measures. Furthermore, the Benjamini & Hochberg’s procedure [97] was also used to control the false discovery rate (FDR) within a family of hypothesis tests.

A multi-way ANOVA was conducted on the local measures, employing the same factors as described for the global measures. Local measures of individual nodes were assessed using statistical methods based on non-parametric permutation analyses with cluster-mass tests [94,96]. Hubs were identified as nodes where the maximum strength or BC value was at least one standard deviation greater than their respective group’s average for that measure. Significant clusters were identified by applying a critical value to the maximum cluster statistic, with the false alarm rate controlled through multiple comparison corrections. The nodes within these clusters were indexed according to their corresponding EEG sensors, and the medians of their distributions were used to examine hemispheric differences. Permutation tests with cluster-mass tests were employed to effectively control the FWER and address the multiple comparisons problem for the dependent local measures (adjusted significance level:  $p = \alpha/2 = 0.025$ ). An additional procedure [97] was used to control the FDR within a family of hypothesis tests. The same selective criteria were applied to the links and edges presented in the figures, highlighting those with specific values of BC and strength (Str).



**Fig. 2.** The global measures ( $\phi$ ,  $\Delta C$ ,  $\Delta L$ ; means  $\pm$  s.e.) of the frequency networks of children with DD (white bars) versus controls (blue color) in the discrimination of: a low tone; a high tone. There was a significant difference ( $* p < 0.05$ ,  $** p < 0.01$ ) between controls and children with DD (Supplementary Tables 2,3). SWP, small-world propensity; DD, developmental dyslexia;  $\Delta C$ , deviation of network's clustering coefficient;  $\Delta L$ , deviation of network's characteristic path length.

### 3. Results

#### 3.1 Behaviour Parameters in Pure Tone Discrimination

Children with DD were significantly faster than controls only for the high-tone condition (mean  $\pm$  s.e.: children with DD,  $796.82 \pm 10.39$  ms; controls,  $844.4 \pm 7.78$  ms;  $p = 2.01 \times 10^{-6}$ ;  $\chi^2 = 22.59$ ), but not for the low-tone condition (children with DD,  $777.69 \pm 10.13$  ms; controls,  $812.64 \pm 7.58$  ms;  $p = 0.35$ ,  $\chi^2 = 0.84$ ; Fig. 1).

The control group gave better success discrimination compared to the children with DD for low tone (children with DD,  $64.54 \pm 2.62\%$ ; controls,  $88.4 \pm 2.55$ ,  $p = 3.54 \times 10^{-9}$ ;  $\chi^2 = 34.86$ ), and for high tone (children with DD,  $59.92 \pm 2.64\%$ ; controls,  $84.4 \pm 3.29\%$ ;  $p = 2.65 \times 10^{-8}$ ;  $\chi^2 = 30.9$ ; Fig. 1).

When comparing two tones in a series of trials, performance is influenced by an implicit memory of previously presented tones [98]. Both control children and those with DD form this implicit memory, which they use to aid in subsequent tone discrimination. However, children with DD benefit less from this effect, particularly for low tones. This deficit is reflected in faster reaction times, a finding observed exclusively in the high-tone condition.

#### 3.2 Comparison of Global SWP Measures of Functional Connectivity

Analysis of global measures showed a significant within-group effect for tones ( $F(1, 1036) = 8.79$ ,  $p \leq 0.003$ ) and frequencies ( $F(6, 1036) = 78.66$ ,  $p \leq 0.000001$ ). A significant between-group main effect was also observed for group ( $F(1, 980) = 19.36$ ,  $p \leq 0.00001$ ).

Significant differences in global network measures between children with DD and controls in low- and high-tone conditions showed (Fig. 2; Supplementary Tables 2,3) that the controls exhibited statistically higher  $\Delta C$  and smaller SWP ( $\phi$ ),  $\Delta L$  than children with DD in low-tone in: (1) the  $\alpha$ - $\beta_1$  frequency networks ( $\phi$ ,  $\Delta C$ ,  $\Delta L$ :  $\chi^2 > 6.115$ ;  $p < 0.013$ ; Supplementary Table 2); (2) the  $\beta_2$ - $\gamma_1$  frequency networks ( $\phi$ ,  $\Delta C$ :  $\chi^2 > 10.006$ ;  $p < 0.00156$ ; Supplementary Table 2); (3) the  $\gamma_2$  frequency network ( $\Delta C$ ,  $\Delta L$ :  $\chi^2 > 5.842$ ;  $p < 0.015$ ; Supplementary Table 3).

Significant differences between groups were found in the high tone at: (1)  $\gamma_1$ -frequency: lower SWP, higher  $\Delta C$  for controls than children with DD ( $\chi^2 > 8.73$ ,  $p < 0.003$ ; Fig. 2, Supplementary Table 3); (2)  $\gamma_2$ -frequencies,  $\gamma_2$ : lower  $\Delta C$ , higher  $\Delta L$  for controls than children with DD ( $\chi^2 > 12.145$ ,  $p < 0.001$ ; Fig. 2, Supplementary Table 3).

A difference was found between the groups in the  $\alpha$ ,  $\beta_1$ ,  $\beta_2$ , and  $\gamma_1$  frequency networks in global measures in low-tone discrimination. The group of children with DD showed larger values of small-world tendency SWP and larger change in characteristic path length  $\Delta L$  and smaller changes in cluster coefficients  $\Delta C$  of frequency functional networks (Fig. 2, Supplementary Table 2). The difference between the functional networks was in  $\gamma$  frequencies (Fig. 2, Supplementary Table 3). An EEG study using graph analysis revealed altered global characteristics of functional brain networks in children with DD during a task involving low- and high-tone discrimination.

These global features suggest that these frequency networks in dyslexic children are more integrated than in controls and not as well segregated as in controls. Heterogeneous trajectories of cortical network maturation during childhood underlie variations associated with disruptions in brain network development across the studied groups.

### 3.3 Comparison of Local Measures of Functional Connectivity

Analysis of EEG graphs revealed changes in the local characteristics (hub strength, hub BC, and connections between them) of functional brain networks in control children and those with DD. Analysis of local measures showed a significant within-group effect for tones ( $F(1, 1484) = 4.55, p \leq 0.033$ ) and frequencies ( $F(6, 1456) = 3.71, p \leq 0.0012$ ). A significant between-group main effect was also observed for group ( $F(1, 1484) = 9.25, p \leq 0.0024$ ).

Different rates of cortical network maturation between groups during childhood contribute to variations in local measures (BC, Str) of certain frequency networks associated with disruptions in brain network development. These local measures indicate that certain frequency networks in children with DD are more integrated compared to controls and are not as well segregated as in controls.

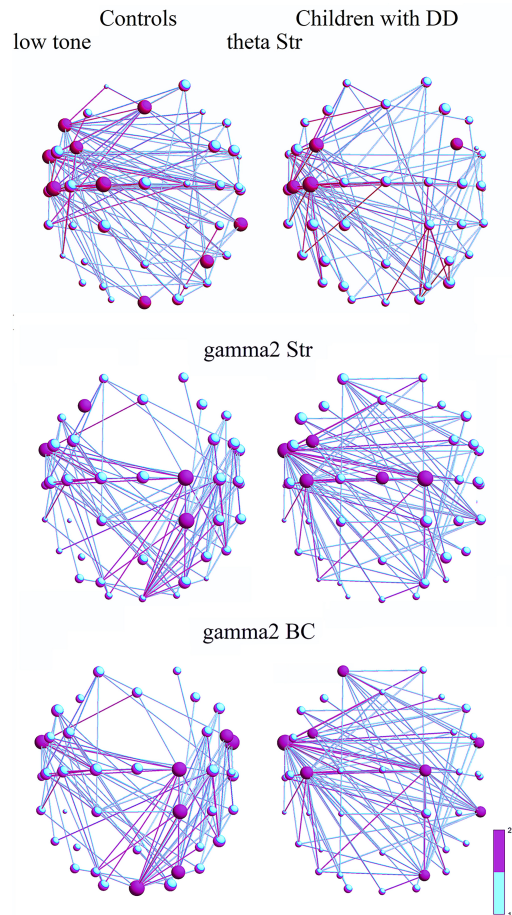
#### 3.3.1 Low Tone Discrimination

In the low-tone condition, the distribution of the strongest hubs in the  $\theta$ -network differed significantly between groups ( $\chi^2 = 6.188, p = 0.012$ ; Table 1; Fig. 3). In controls, these hubs were located in the medial frontal cortex (MFC: Fz), inferior frontal gyrus (IFG: F7), middle frontal gyrus (MFG: FC3), anterior inferior temporal gyrus (aITG: FT9), precentral gyrus (PreCG: C1), postcentral gyrus (PstCG: C5), middle temporal gyri (MTG: TP8 BA21/22/37/20), superior parietal lobe (SPL: P4), and occipital lobe (OL: Oz). In children with DD, the strongest hubs were found in the middle frontal gyrus (MFG: FC3–4) and postcentral gyrus (PstCG: C3, C5).

In the  $\gamma_2$ -network, hub strength was greatest in controls at MFG (intermediate frontal: F3), aITG (FT9), PreCG (C2), and SPL (CP2), and in children with DD at MFG (including premotor/pre-SMA, supplementary motor area, SMA: FC3), aITG (FT9), PreCG (C2, Cz), and PstCG (C3). The group difference was statistically significant ( $\chi^2 = 44.110, p = 1.116 \times 10^{-16}$ ; Table 2; Fig. 3). Controls showed the highest hub BC at aITG (FT9–10), IFG (FC6), PreCG (C2), SPL (CP2), superior occipital lobe (SOG: PO4), and OL (Oz), while children with DD showed it at superior frontal cortex (SFC or dorsolateral prefrontal cortex DLPC: AF3), aITG (FT9–10), and PreCG (C2) ( $\chi^2 = 15.935, p = 0.00016$ ; Table 2; Fig. 3).

#### 3.3.2 High Tone Discrimination

The distribution of hubs with the greatest strength in the  $\delta$ -network for a high tone differed significantly between controls and children with DD ( $\chi^2 = 13.360, p = 0.0002$ ; Table 2; Fig. 4). In controls, these hubs were located at SFC (AF3), PstCG (C6), posterior Superior Temporal Gyrus (pSTG: T7), and OL (Oz), while in children with DD, they were at SFC (AF3–4), IFG (F7), aITG (FT9), PstCG (C5), pSTG (T7), SOG (PO4 BA19/18/39), pITG (P8), and SPL (Pz).



**Fig. 3. Frequency networks of controls (1st column) and children with DD (2nd column) for a low tone.** The 3D spatial network topology of nodes is shown in scalp coordinates, using a color gradient from purple to cyan. Purple nodes and connections represent main hubs and strong connections that are above the significant threshold; their largest size corresponds to a value of 2 on the color scale. Cyan nodes and links are below the significant threshold, with the smallest size representing a value of 1: (1) Strength (Str) of hubs in the  $\theta$ -network (controls: Fz, F7, FC3, FT9, C1, C5, TP8, P4, Oz; children with DD: FC3–4, C3, C5); (2) Str of hubs in the  $\gamma_2$ -network (controls: F3, FT9, C2, CP2; children with DD: FC3, FT9, C2, Cz, C3); BC of hubs in the  $\gamma_2$ -network (controls: FT9–10, FC6, C2, CP2, PO4, Oz; children with DD: AF3, FC3, FT9–10, C2, C3, TP8, PO4).

In the  $\gamma_1$ -network, there were statistically significant differences in the distribution of hubs with the greatest BC between groups ( $\chi^2 = 5.302, p = 0.021$ ; Table 2; Fig. 4). Controls showed these hubs at IFC (F7–8), aITG (FT9–10), PstCG (C5), middle temporal gyrus (MTG: TP8), SOG (PO4), and middle occipital gyrus (MOG: PO7), while children with DD showed them at IFG (F7), PreCG (C2), PstCG (C5), pSTG (T7–8), and MTG (TP7).

In the  $\gamma_2$ -network, hub strength and BC differed significantly between controls and children with DD. The

**Table 1. Statistically different local measures between groups for low-tone discrimination are indicated in bold and correspond to Fig. 2 ( $\theta$ , hub strength).**

Frequency band	Local measures	Controls	Dyslexia	Kruskal-test	Wallis
		Main hubs	Main hubs	$p$ -values	$\chi^2$ -statistics
$\delta$	strength	AF3–4, Fz, F3, FC5, C5, CP1, T8	AF3, F7, F3, FC3, CP1, T8	0.693	0.154
	BC	Fz, FT9–10, T8, PO4	F7, FT9, C3, CP2	0.246	1.342
$\theta$	strength	Fz, FC3, F7, FT9, C1, C5, TP8, P4, Oz	FC3–4, C3, C5	<b>0.012</b>	6.188
	BC	F7, FT9, C3, T7, PO8	Fz, FC3, FT9, C3, T7, PO8, O2	0.130	2.291
$\alpha$	strength	Fz, C1, C3, C5, CP1, P8, Pz, PO8, O1–2	C3, CP3, TP7, P3, PO7	0.043	4.090
	BC	Fz, FT9, C3, C5, PO4, PO8, Oz, O2	Fz, F7, FT9–10, C4, PO7–8, O2	0.094	2.803
$\beta_1$	strength	Fz, C1, Cz, C3, PO3	F3, C1–2, C3–4, CP4, Pz, Oz	0.033	4.535
	BC	FT9, C3, T7, PO4, PO8, O2	FT9, T7, PO4, P8, PO7–8, Oz	0.194	1.682
$\beta_2$	strength	FT9, C3, T7, PO4, PO8, O2	FC4, FT9, C3–4	0.183	1.771
	BC	Fz, FC4, C2, Cz, C4, PO7	F7, FT9–10, C3, TP7	0.531	0.392
$\gamma_1$	strength	FC4, FT9–10, C2, C5, T7–8, TP8	FC4, C4, C6, T8, TP8, PO8	0.043	4.090
	BC	FC4, C4, T7, Oz, O2	AF3, FT9, C6, T7–8, PO4, PO8, O2	0.094	2.803
$\gamma_2$	strength	F3, FT9, C2, CP2	FC3, FT9, C2, Cz, C3	<b>&lt;0.001</b>	44.110
	BC	FC6, FT9–10, C2, CP2, PO4, Oz	AF3, FC3, FT9–10, C2, C3, TP8, PO4	<b>&lt;0.001</b>	15.935

Statistically different local measures between groups for low-tone discrimination are indicated in bold. BC, betweenness centrality.

greatest hub strength was observed at MFG (F3), PreCG (C2), PstCG (C3, C5), and SPL (CP2) for controls, and at anterior temporal gyrus (ATG: FT9), PreCG (C2, Cz), and PstCG (C3) for children with DD ( $\chi^2 = 68.752, p = 1.116 \times 10^{-16}$ ; Table 2; Fig. 4). The highest BC was found at aITG (FT9–10), PstCG (C5), SPL (CP2), posterior part of inferior temporal gyrus (pITG: P8), and superior occipital lobe (SOG: PO4) for controls, and at SFC (AF3), aITG (FT9–10), and PreCG (C2) for children with DD ( $\chi^2 = 14.242, p = 0.00016$ ; Table 2; Fig. 4).

## 4. Discussion

The multifaceted relationship between DD and auditory processing offers a deeper understanding of this learning disorder. A central debate revolves around whether auditory deficits in dyslexia are specific to speech or reflect more general auditory processing challenges. Dyslexia is linked to alterations in auditory cortex anatomy and function. Specifically, children with DD exhibit alterations in auditory sampling within the delta/theta [99,100] and low-frequency gamma bands [101,102] in auditory cortical areas [103] during early stages of processing.

### 4.1 Global Measures of Network Connectivity During Tone Discrimination

The small-world propensity ( $\phi$ ) vary between groups. Lower  $\phi$  values indicate substantial deviations from null model predictions for clustering and path length, suggesting the network is less “small-world”. A low  $\phi$  results from high  $\Delta C$  (lack of local clustering) while maintaining a small path length and increased randomness. Even a small  $\Delta L$  can decrease  $\phi$ , though a higher  $\Delta C$  can also drive this de-

crease [89]. In response to a low tone, controls showed increased local network connectivity and decreased global integration in auditory  $\alpha$  and  $\beta_1$  networks. This was characterized by a weak small-world propensity ( $\phi$ ), small deviations in  $\Delta L$ , and a large deviation in  $\Delta C$ . Compared to children with DD, controls also exhibited lower  $\phi$  and smaller  $\Delta L$  in the  $\beta_1$  (low-tone) and  $\gamma_1$  (both tones) networks. A substantial  $\Delta C$  (0.75 for controls in  $\beta_1$ ) indicates strong divergence from a lattice network, while a very low  $\Delta L$  for controls in  $\beta_1$  highlights its similarity to a random network. This behavior suggests a rewiring mechanism that can transform the network between these two topologies. The observed “small-world” properties are thus almost entirely driven by short path length. This suggests the network does not genuinely exemplify small-world principles due to deficient local clustering, indicating topological reorganization and a minimal tendency towards a true small-world structure. A genuine small-world structure is characterized by high local clustering and short path lengths, which facilitates information flow. In contrast, children with DD exhibited larger deviations in path lengths ( $\alpha, \beta_1$ ), indicating greater variability in connectivity, and smaller deviations in clustering ( $\alpha, \beta, \gamma_1$ ), suggesting increased global integration of these networks. The more balanced network segregation and integration, characterized by moderate clustering and path length, observed in the controls’  $\gamma_2$  frequency network, might explain the optimal network structure supporting their better performance.

**Table 2. Statistically different local measures between the groups for high-tone discrimination are indicated in bold and correspond to Fig. 2 ( $\delta$ ,  $\gamma$ , strength, and BC of the hubs).**

Frequency band	Local measures	Control	Dyslexia	Kruskal-test	Wallis
		Main hubs	Main hubs	<i>p</i> -values	$\chi^2$ -statistics
$\delta$	strength	AF3, C6, T7, Oz	AF3–4, F7, FT9, C5, T7 PO4, P8, Pz	<b>&lt;0.001</b>	13.360
	BC	C6, T7, TP7, PO8, Oz, O2	F3, FT9, T7, PO4, P8	0.525	0.402
$\theta$	strength	FC3, FC5, Fz, PO7, Oz	F3, FC3, FC5, FT9, C3, C5	0.150	2.069
	BC	FC3, FC5, FT9, C2, PO7–8, P8, Oz, O2	Fz, FT9–10, PO4, PO8	0.079	3.078
$\alpha$	strength	AF3, F3, FC3, C3, CP1, Pz, P3, O1	CP1, CP3, PO3–4, P3, Pz	0.240	1.376
	BC	AF3, PO4, PO8, O1–2, Oz	Fz, FT9, T7, PO4, PO7–8	0.923	0.009
$\beta_1$	strength	Fz, FC4, C1, PO3, CP2	AF3, F3, FC3, F7, FC5, C3, C5	0.069	3.291
	BC	Fz, FT10, T7–8, PO4, PO8, Oz	AF4, F7, FT9–10, PO4, P8	0.666	0.185
$\beta_2$	strength	AF4, FT9, C1, TP7, P7, Oz	Fz, C1, CP1–2, CP3, TP7, C4	0.355	0.852
	BC	FC4, FT9–10, T7, TP7, PO8, Oz	Fz, FC4, FT10, C2, T7–8, TP7	0.040	4.177
$\gamma_1$	strength	FT10, CP1, TP8, CP4, P4, PO7	FC6, C4, C5–6, TP8, CP1, CP4, P8	0.788	0.072
	BC	F7–8, FT9–10, C5, TP8, PO4, PO7	F7, C2, C5, T7–8, TP7	<b>0.021</b>	5.302
$\gamma_2$	strength	F3, C2, C3, C5, CP2	FT9, C2, Cz, C3	<b>&lt;0.001</b>	68.752
	BC	FT9–10, C5, CP2, P8, PO4	AF3, FT9–10, C2	<b>&lt;0.001</b>	14.242

Statistically different local measures between groups for low-tone discrimination are indicated in bold.

#### 4.2 Local Measures of Tone-Dependent Functional Connectivity

The study revealed that tone differences were reflected in functional networks by variations in the strength and BC of major hubs across frontal, parietal, and primary sensory cortices, alongside the diverse contributions of individual nodes.

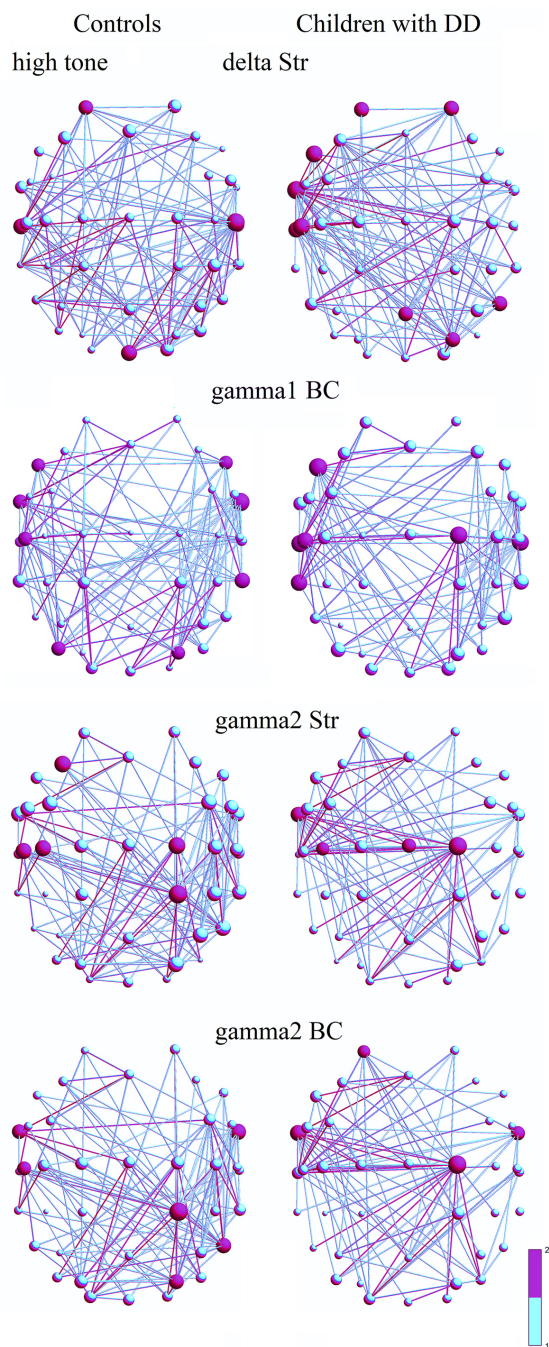
In the low-tone  $\theta$ -frequency network, controls showed their strongest hubs in the frontal lobes (medial, middle, and inferior), the anterior inferior temporal region, and the primary somatosensory and auditory cortices. These hubs were connected through the superior parietal lobe to primary sensory cortices, including the right middle temporal cortex and the occipital lobe. Conversely, children with DD showed their strongest hubs only in the middle frontal cortex of both hemispheres and the primary somatosensory and auditory cortices. These DD-specific hubs lacked high BC, and no connections through the parietal lobe to primary sensory areas were observed.

In the control group's low-tone  $\gamma_2$  network, stronger, more central hubs were prominent in the frontal lobes, particularly the right inferior frontal lobe. These hubs connected via the superior parietal lobe to other sensory areas, including the superior occipital lobe and occipital cuneus. Conversely, in children with DD, the frontal lobes (prefrontal and middle) showed the highest strength in the  $\gamma_2$  network but connected only through primary somatosensory and motor areas to the right middle temporal and superior occipital regions. Notably, these primary sensory regions lacked strong  $\gamma_2$  connectivity in the DD group (Fig. 3). Additionally, the pre-supplementary motor areas (pre-SMA) showed increased engagement during low-tone processing.

These findings suggest the middle frontal cortex, particularly the pre-SMA, plays a tone-structure-modulated role in DD children's  $\theta$ - and  $\gamma_2$  networks, independent of specific auditory frequency. This points to the pre-SMA's primary contribution in movement prediction and temporal control, driven more by temporal information than auditory tone.

In the high-tone  $\delta$  network, children with DD exhibited widespread bilateral activity across prefrontal, frontal, temporal, parietal, and occipital regions, though the left superior temporal gyrus (STG) and posterior superior parietal lobe had weak primary sensory connections. Conversely, controls' strongest primary somatosensory and auditory cortices (right hemisphere) and left STG showed high BC with other sensory cortices, including the occipital lobe, with the right auditory cortex appearing to favor high-tone processing. Both groups had robust left posterior superior temporal lobe (pSMG) strength. The prefrontal region (left dorsolateral prefrontal cortex (dLPFC) for controls; bilateral for DD) is implicated in temporal processing and action planning [104,105]. DD children's bilateral "ramping activity" suggests cognitive interval tuning, separate from motor responses. Attention and arousal significantly modulate temporal processing: reduced attention shortens perceived duration and alters activity in limbic, medial cortical, and cortico-thalamic-basal ganglia circuits, as well as frontal, temporal, and parietal cortices [105].

In the high-tone  $\gamma_1$  network, children with DD showed the highest BC in the left IFG, left primary somatosensory and auditory cortices (connected to both hemispheric pSTGs), and the left MTG. Conversely, controls exhibited their highest BC in both hemispheric IFGs and aITGs, extending through the left somatosensory and auditory cor-



**Fig. 4. Frequency networks in controls (1st column) and children with DD (2nd column) for a high tone:** (1) Str of hubs in the  $\delta$ -network (controls: AF3, C6, T7, Oz; children with DD: AF3–4, F7, FT9, C5, T7, P8, PO4, Pz); (2) BC of hubs in the  $\gamma_1$ -network (controls: F7–8, FT9–10, C5, TP8, PO4, PO7; children with DD: F7, C2, C5, T7–8, TP7); Str of hubs in the  $\gamma_2$ -network (controls: F3, C2, C3, C5, CP2; children with DD: FT9–10, C2, Cz, C3); BC of hubs in the  $\gamma_2$ -network (controls: FT9–10, C5, CP2, P8, PO4; children with DD: AF3, FT9–10, C2).

tex to primary sensory regions like the right MTG, right SOG, and left MOG. Unlike controls, children with DD lacked high-BC hubs in the aITG in both hemispheres at the

$\gamma_1$  network, mirroring findings in patients with specific lesions [106]. The  $\gamma_2$  network in controls featured the highest strength in the middle frontal cortex, primary somatosensory, motor, and auditory cortices, and the SPL. These areas, especially the auditory cortex and SPL, also had high BC with bilateral aITG and primary sensory regions (ITG, SOG). In contrast, children with DD showed less developed parietal lobes with poor connections to the frontal lobe and bilateral aITGs. Instead, their bilateral aITG had the highest BC, but only with the primary somatosensory and premotor cortices. In controls, the IPL consistently connected to main hubs across low- and high-frequency auditory conditions, reinforcing its role in integrating sensory inputs for higher-order timing and transmitting information to prefrontal areas for pitch prediction [107]. This IPL connectivity with primary somatosensory and auditory cortices was tone-dependent only in controls, with connections from the left primary somatosensory/auditory cortices (high-tone) and connections from the right primary motor/premotor/somatosensory cortices (low-tone). In these conditions, sensory processing largely remained in early sensory areas, with weak prefrontal recruitment in the  $\delta$  band. However, high tones ( $\gamma_1$ ) potentially involved bilateral Broca's areas, while low tones ( $\theta$ ) involved only the left side, suggesting weaker auditory network engagement. This aligns with studies on sensory-motor coupling in rhythm tracking [108,109]. This  $\delta$ -frequency network pattern was only observed for the high-tone in children with DD. In summary, when timing is predictable, the core network is functionally affected by development, indicated by weak IPL–prefrontal interactions. When predictability is low, children with DD exhibited a localized high-tone auditory network strategy, with left somatosensory and auditory regions primarily influenced by the SPL and substantial prefrontal involvement. This tone-dependent connectivity highlights the importance of top-down prefrontal control for temporal predictions and the adaptive, yet compensatory, role of sensory regions when predictability is diminished. A stronger functional connectivity both between hemispheres of the auditory network and between the auditory system and prefrontal/parietal areas in the controls than in the dyslexics tended to be associated with facilitation in the encoding and discrimination of the tone sequence, a relationship less evident in dyslexics [110–112]. The task-driven bilateral frontotemporal auditory network encoding tones showed frequency-specific activity: in the  $\theta$ -frequency range for low tones, in  $\delta$ - and  $\gamma_1$ -frequency ranges for high tones, and  $\gamma_2$ -frequency ranges for both tone frequencies.

Additional hubs observed in controls for high tone included primary somatosensory, auditory, and supra-marginal cortex (right in  $\delta$ , left in  $\gamma_1$ ), and right occipitotemporal gyrus ( $\gamma_2$ ), while for low tone—right IFG and occipital lobe ( $\gamma_2$ ). In contrast, children with DD only showed additional hubs for low tone in the left dLPFC,

left pre-SMA, SMA, and right MTG ( $\gamma_2$ ). Strong inter-hemispheric connections between auditory cortices in typically developing children likely support accurate tone perception and retention, crucial for speech decoding and phonetic categorization [113,114]. The dLPFC is key for working memory, maintaining and manipulating sensory information from posterior sensory areas [115]. Consequently, weakened connectivity between the DLPFC and sensory regions can impair both fine detail and overall auditory feature processing. Reduced frontotemporal connectivity, observed in DD, is linked to difficulties retrieving tonal information [116–119], potentially indicating poorer auditory processing [119].

A potential neurophysiological mechanism for dyslexia's deficits may involve impaired functional communication between sensory regions and the prefrontal cortex, alongside excessive communication between homologous auditory areas. The strength of interhemispheric functional connections within relevant sensory networks could underpin specific neurophysiological mechanisms leading to perception-cognitive biases or frequency discrimination difficulties.

## 5. Conclusion

The study reveals distinct local and global functional network properties in children with DD during auditory processing. It highlights differences in integration, segregation, hub organization, and connectivity, particularly involving the parietal and prefrontal cortices. It is important to interpret scalp-level EEG network results with caution, even though the estimations of functional connectivity are highly reliable [120]. These findings suggest neurophysiological differences that may underlie the auditory processing difficulties associated with DD.

## Availability of Data and Materials

The data are not publicly available due to privacy and confidentiality concerns but are available from the corresponding author on reasonable request.

## Author Contributions

TT and JD designed and performed the research. TT was responsible for data analysis. Both authors wrote the initial draft. Both authors contributed to editorial changes in the manuscript. Both authors read and approved the final manuscript. Both authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

## Ethics Approval and Consent to Participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of the Institute of Neurobiology, Bulgarian Academy of Sciences (BAS, approval No. 02-41/12-07-2019), on 12

July 2019. Ethical approval, ensured through informed consent from the children and their parents, was a prerequisite for the children's participation in the EEG study.

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## Conflict of Interest

The authors declare no conflict of interest. Given the role as the Editorial Board member, Dr. Juliana Dushanova had no involvement in the peer-review of this article and has no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to Bettina Platt.

## Supplementary Material

Supplementary material associated with this article can be found, in the online version, at <https://doi.org/10.31083/JIN42398>.

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