

Research Report

**DIFFERENT BRAIN ACTIVATION PATTERNS IN DYSLEXIC
CHILDREN: EVIDENCE FROM EEG POWER AND
COHERENCE PATTERNS FOR THE DOUBLE-DEFICIT
THEORY OF DYSLEXIA**

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Aims: QEEG and neuropsychological tests were used to investigate the underlying neural processes in dyslexia.

Methods: A group of dyslexic children were compared with a matched control group from the Brain Resource International Database on measures of cognition and brain function (EEG and coherence).

Results: The dyslexic group showed increased slow activity (Delta and Theta) in the frontal and right temporal regions of the brain. Beta-1 was specifically increased at F7. EEG coherence was increased in the frontal, central and temporal regions for all frequency bands. There was a symmetric increase in coherence for the lower frequency bands (Delta and Theta) and a specific right-temporocentral increase in coherence for the higher frequency bands (Alpha and Beta). Significant correlations were observed between subtests such as

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Rapid Naming Letters, Articulation, Spelling and Phoneme Deletion and EEG coherence profiles.

Discussion: The results support the double-deficit theory of dyslexia and demonstrate that the differences between the dyslexia and control group might reflect compensatory mechanisms.

Integrative Significance: These findings point to a potential compensatory mechanism of brain function in dyslexia and helps to separate real dysfunction in dyslexia from acquired compensatory mechanisms.

Keywords: Dyslexia; EEG; QEEG; coherence; double-deficit theory.

1. Introduction

Developmental dyslexia is characterized by difficulties with accurate and/or fluent word recognition, by poor spelling and decoding abilities. These difficulties typically result from a deficit in the phonological component of language that is often unrelated to other cognitive abilities [20]. Dyslexia is probably the most common neurobiological disorder affecting children, with prevalence rates ranging from 5 to 10 percent, and is a persistent, chronic condition [33].

Reading problems manifest themselves mainly in the following areas: difficulty in learning to utilize correspondence regularities between graphemes and phonemes [12, 37] poor phonological awareness, i.e., awareness of the sound structure of words, especially phonemic awareness as manifested in the ability to analyze and manipulate sounds within a syllable [34] and poor use of orthographic word reading strategies; and consequently inaccurate and non-fluent word identification [22, 26]. As a result of these difficulties, full alphabetic or phonological reading skills are often not attained. A large body of research has been conducted on the relation between phonological awareness and learning to read. Strong support has been provided that lack of phonological awareness can cause difficulties with the acquisition of reading and writing [29, 36]. Being able to distinguish and identify the different phonemes in a word is part of this awareness. Research in the past decades has provided ample evidence that dyslexic children have problems with phonological awareness and other aspects of phonological processing. There is a general agreement that this phonological processing deficit has to do with problems in phonological encoding [34]. Poor readers are less precise in phonemic discrimination, they have problems on a variety of phoneme segmentation and awareness tasks [40], and they are slower in rapid naming of objects, digits and letters [42, 43], as well as in producing rhyming words [15]. It can be hypothesized that dyslexia is fundamentally a linguistic problem which involves a deficit in phonological encoding. Elbro, Borstrøm and Petersen [6] tested this hypothesis by predicting dyslexia from phonological processing abilities of kindergarteners. It was shown that three language measures contributed independently to predict dyslexia: letter naming, phoneme identification, and distinctness of phonological representations. The results further indicated that the quality of phonological representations in the child's mental lexicon may also be a determinant of the

development of phonemic awareness. Alternatively, there is the claim that reading problems originate from a more general temporal processing deficit [35].

Dyslexia has been attributed to deficiencies in visual, linguistic, and low level sensory functions but most studies have been falsified empirically and logically [38]. Most research emphasizes the phonological deficit in children with dyslexia [1, 20, 33, 34], that is segmenting spoken words into their underlying phonological elements and linking each letter to its corresponding sound. A number of studies also support the double-deficit theory. The double-deficit theory proposes that reading disabilities can be the result of: 1) poor phonological awareness and/or, 2) automatic naming skills. Poor phonological awareness refers to disabilities in identifying and manipulating sounds in speech, whereas poor automatic naming implies the disability to translate visual information into a phonological code. The double-deficit hypothesis proposes that accordingly, subtypes of dyslexia can be distinguished showing a deficit in either one or both of these skills [4, 43]. It is also claimed that a deficit in both skills yields the lowest reading performance. The dyslexic subtypes could be produced by differential processing deficits in the frontal-cerebellar phonological system [4]. The unique contribution of each frontal and cerebellar measure to the classification of dyslexic participants and the prediction of phonological and naming performance support this view.

Previous research has also linked dyslexia and reading disabilities to neurological data. There are anatomical studies [7, 13] which show an absence of the usual left-right hemisphere asymmetry of the planum temporale in dyslexia or suggest a possible role of the left inferior frontal gyrus in speech perception and rapid auditory processing, as well as in phonological aspects of reading [13], although no strong effects have been reported [13]. Eckert *et al.* [4] found anatomical anomalies underlying the double-deficit subtype of dyslexia. Their findings suggest that impairments in a frontal-cerebellar network may play a role in delayed reading development in dyslexia.

To study the neural factors of dyslexia, functional neuroimaging has been used. However, there is not much evidence with respect to developmental dyslexia since this research has focused on (young) adults [13]. Only Shaywitz and Shaywitz [32, 33] used children in their neuroimaging studies in order to examine the neural systems for reading during the acquisition of literacy. These reports show a failure of left hemisphere posterior brain systems to function properly during reading [32, 33]. The majority of studies show increased activation in the basal surface of the temporal lobe, the posterior portion of the superior and middle temporal gyri, extending into temporoparietal areas and the inferior frontal lobe during tasks requiring reading and phonological processing [38]. Shaywitz *et al.* [32] supports these findings, however they show evidence of right hemisphere activation in the posterior temporal parietal regions. This could reflect compensatory processes or could indicate that other nonlinguistic factors are related to reading disability [32, 33, 38].

A few studies have focused on event related EEG changes in tasks directly related to the reading difficulties of dyslexic children. Rippon and Brunswick [28] found

that dyslexic children showed increased frontal theta activity in a phonological task, whereas there were no differences between the dyslexic group and the control group in a visual task. Furthermore, there was a marked parieto-occipital right greater than left asymmetry in beta EEG activity in the dyslexic group with respect to the phonological task and the visual task. Klimesch *et al.* [17] found that dyslexics have a lack of attentional control during the encoding of words at left occipital sites and a lack of a selective topographic activation pattern during the semantic encoding of words.

EEG coherence is a measure which displays functional connectivity between brain areas, and could hence be an interesting measure to demonstrate deviation in functional connectivity. To date, few EEG studies have considered EEG coherence. Sklar *et al.* [31] found higher intrahemispheric coherence and lower interhemispheric coherence during text processing in dyslexics compared with normals. This was also supported by Leisman and Ashkenazi [19]. During rest, Shiota, Koeda, and Takeshita [30] reported both increased intra- and inter-hemispheric coherence in dyslexic children. Furthermore, Marosi *et al.* [23] found a frequency-dependent effect on EEG coherence at rest where differences between children with poor reading/writing abilities were compared with children with good reading/writing abilities, with the former showing higher coherence in the delta, theta and beta bands and lower coherence in the alpha bands during rest [39].

Weiss and Mueller [39] proposed that EEG coherence in the different frequency bands played different roles: increased coherence in the theta band correlates with language-related mnemonic processes and theta coherence was increased if task demands increased and more efficient information processing was required. Alpha coherence seemed important for sensory processing and higher alpha coherence for semantic processing. Beta and gamma coherence has been linked with more complex linguistic sub-processes such as syntax or semantics [39].

Our aim was to compare brain function of dyslexic children with non-dyslexic children on different neurophysiological and neuropsychological measures. Our question focused on whether different EEG activation patterns can be found in dyslexia, and to what extent correlations between reading and spelling abilities and specific tasks for rapid naming and phonological awareness, can be found to address the double-deficit theory of dyslexia [43]. We also assessed neuropsychological function in these groups in order to exclude further cognitive differences between the groups potentially confounding the EEG findings. Our hypothesis was that the groups will not show differences on neuropsychological measures, but that children with dyslexia will show increased inter- and intrahemispheric coherence.

2. Materials and Methods

2.1. Subjects

Nineteen children with dyslexia (11 males and 8 females; average age = 10.33; range 8.0–15.98) and nineteen control children (matched on age, gender and education;

11 males and 8 females; average age 10.34; range 8.01–16.03) were used to investigate the differences in brain function and neuropsychological performance. All dyslexic children went to regular schools. They were diagnosed with dyslexia by their remedial teachers, who worked with a structured protocol for diagnosing children with dyslexia on the basis of their reading and spelling development from grade 1 [44]. The control group was drawn from the Brain Resource International Database (BRID: www.brainresource.com, for more details also see [9, 10]) and children were chosen from this database who did not have dyslexia or learning disorders.

Exclusion criteria included a personal or family history of mental illness, brain injury, neurological disorder, serious medical condition, drug/alcohol addiction; and a family history of genetic disorder. All subjects voluntarily gave written informed consent.

Subjects were seated in a sound and light attenuated room, controlled at an ambient temperature of 22°C/72°F. Electroencephalographic and neuropsychological assessments were completed in order.

2.2. Language tests

The group of children with dyslexia was submitted to a range of tests to investigate correlations between EEG and neuropsychological findings of dyslexia. The included tests were measures of tasks related to reading: Rapid Naming of Letters, Articulation, Phoneme deletion [16] and Spelling [8].

2.3. Electroencephalographic data acquisition

Participants were seated in a sound and light attenuated room, controlled at an ambient temperature of 22°C. EEG data were acquired from 28 channels: Fp1, Fp2, F7, F3, Fz, F4, F8, FC3, FCz, FC4, T3, C3, Cz, C4, T4, CP3, CPz, CP4, T5, P3, Pz, P4, T6, O1, Oz and O2 (Quikcap; NuAmps; 10–20 electrode international system). Data were referenced to averaged mastoids with a ground at Fpz. Horizontal eye-movements were recorded with electrodes placed 1.5 cm lateral to the outer canthus of each eye. Vertical eye movements were recorded with electrodes placed 3 mm above the middle of the left eyebrow and 1.5 cm below the middle of the left bottom eye-lid. Skin resistance was < 5 K Ohms and above 1 K Ohm for all electrodes. A continuous acquisition system was employed and EEG data were EOG corrected offline [11]. The sampling rate of all channels was 500 Hz. A low pass filter with attenuation of 40 dB per decade above 100 Hz was employed prior to digitization.

The EEG data were recorded for two minutes with eyes open (EO). Subjects were asked to sit quietly. Subjects were asked to fix their eyes on a red dot presented on a computer screen.

2.4. *Electroencephalographic variables*

Each two minute epoch was divided into adjacent intervals of four seconds. Power spectral analysis was performed on each four second interval by first applying a Welch window to the data, and then performing a Fast Fourier Transform (FFT), next the average power spectra were calculated.

The power was calculated in the following frequency bands delta (1.5–3.5 Hz), theta (4–7.5 Hz), alpha (8–13 Hz), alpha1 (8–11 Hz), alpha2 (11–13 Hz), SMR (12–15 Hz), beta (14.5–30 Hz), beta1 (14.5–20 Hz), beta2 (20–25 Hz) and beta3 (25–30 Hz). The data were then square-root transformed to approximate the normal distributional assumptions required by parametric statistical methods.

2.5. *Neuropsychology*

Neuropsychological assessment was done using a touch screen monitor. Besides the subtests for dyslexia, other neuropsychological tests were included in order to establish that the children were otherwise completely normal. Measures included: memory recall and memory recognition (number of correctly reproduced words on trials 1, 5, 6, 7; number or correctly recognized words), verbal interference test — equivalent to the Stroop test (Number correct text and color condition), tapping test (Number of taps with the dominant and nondominant hand), timing test (proportional bias) and switching of Attention test part A and B (equivalent to the WAIS Trails A and B; time to complete the A and B form) (see [9, 10] for details of these tests). All tests were fully computerized and subjects' responses were recorded via touch-screen presses. Reliability and validity data of these tasks are reported elsewhere [2, 10, 25].

3. Statistical Analysis

3.1. *Missing values*

If missing values were present for a given statistical test, those cases were excluded for that analysis. The number of missing values per group are included in the results sections.

3.2. *Statistical analyses*

Since we expected quite local effects on some measures due to the localized differences in brain function for dyslexia, we did not perform the traditional GLM, since small localized effects could average out in the overall tests. Therefore, we performed one-way ANOVA's but used very stringent alpha correction. Significance levels were set as follows: for the EEG power data, the significance level was set to $p < 0.05$ and for the coherence data, significance levels were set to $p < 0.001$. For EEG coherence there were many more data points per frequency band (> 100 coherence values), hence the lower p value for coherence compared to EEG power.

The obtained significant differences between the dyslexia and control group were then submitted to a bivariate correlation analysis together with the severity questionnaire data, and a correlation matrix was obtained for correlations between variables within the group of dyslexic children.

4. Results

4.1. EEG power

An overview of Delta and Theta power for all sites is depicted in Fig. 1 and the significant differences are indicated.

The following differences between the two groups were found:

Delta: increased Delta power for the dyslexia group at Fp1 ($F = 6.315$, $df = 1, 33$, $p = 0.017$), Fp2 ($F = 4.861$, $df = 1, 34$, $p = 0.034$), F7 ($F = 4.806$, $df = 1, 34$, $p = 0.035$) and T6 ($F = 6.193$, $df = 1, 35$, $p = 0.018$).

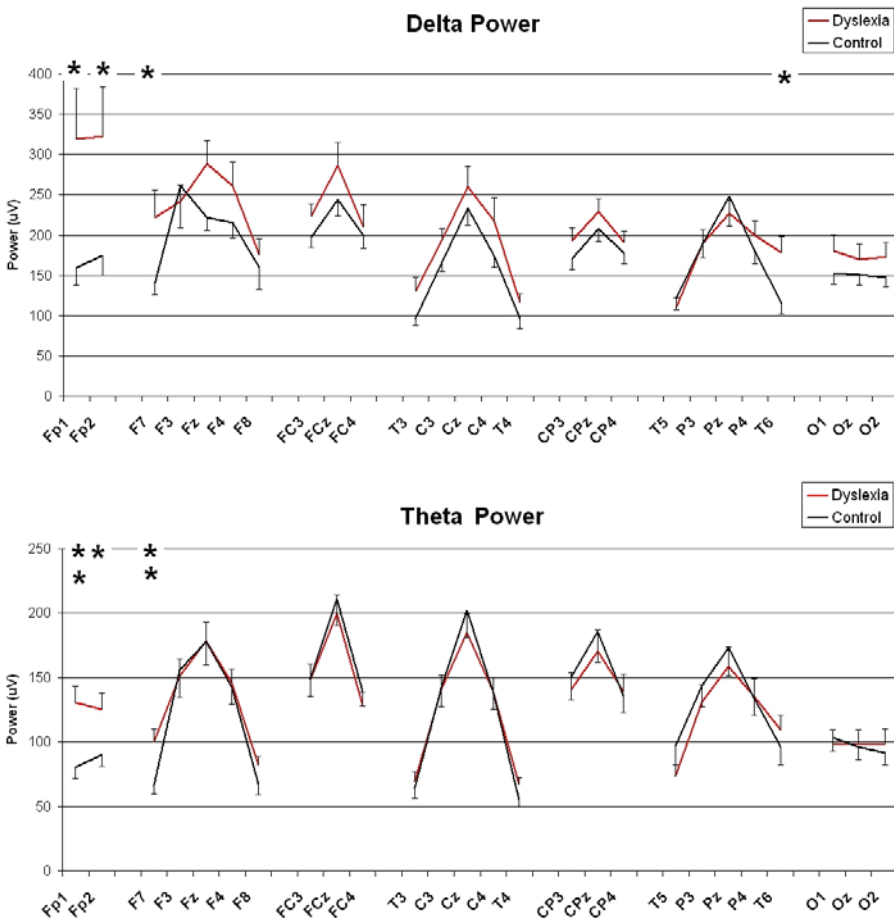


Fig. 1. Mean EEG power for Delta and Theta for the dyslexic group (in red) and the control group (in black). All findings have $p < 0.05$.

Theta: increased Theta at Fp1 ($F = 11.072$, $df = 1, 33$, $p = 0.002$), Fp2 ($F = 5.074$, $df = 1, 34$, $p = 0.031$) and F7 ($F = 8.267$, $df = 1, 34$, $p = 0.007$).

Beta 1: increased beta-1 at F7 ($F = 4.450$, $df = 1, 34$, $p = 0.042$).

4.2. EEG coherence data

Figure 2 gives an overview of the significant increases in coherence per frequency band. All increases have p values of $p < 0.001$. Due to the many significant findings, no detailed statistics are given. All coherence values were increased for the dyslexia group *red* connections are from homologous pairs (both right and left hemisphere) and *purple* are uniquely right or left hemispheric increased coherences. Note the specific patterns which include mainly frontal, central and temporal sites. Also note

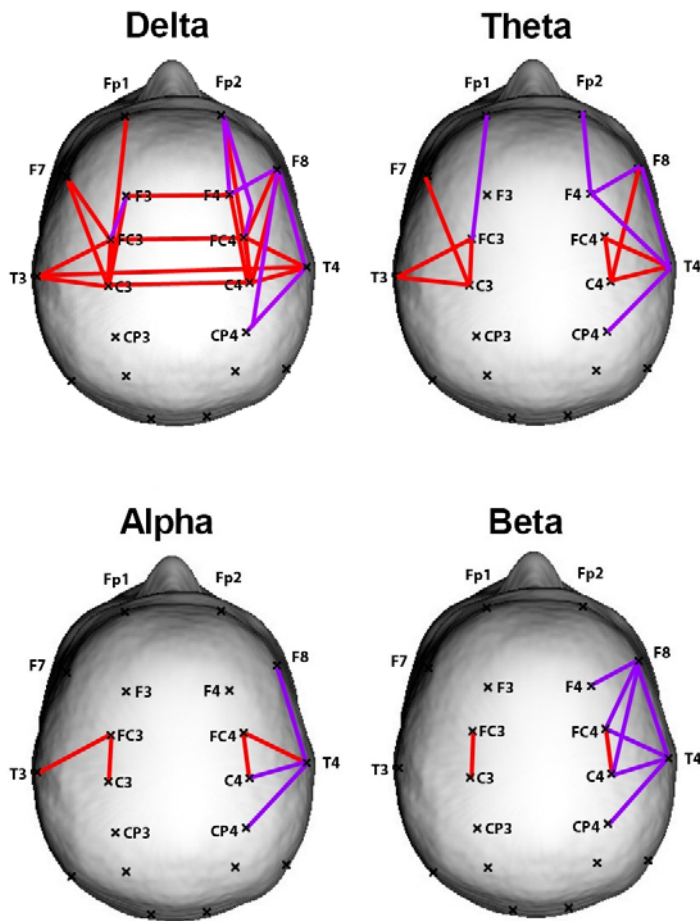


Fig. 2. Significant increased coherences for the dyslexic vs. the control group for the different frequency bands. *Red* connections are from homologous pairs (equal right and left) and *purple* connections are unique right or left hemispheric increased coherences. All p values's < 0.001 .

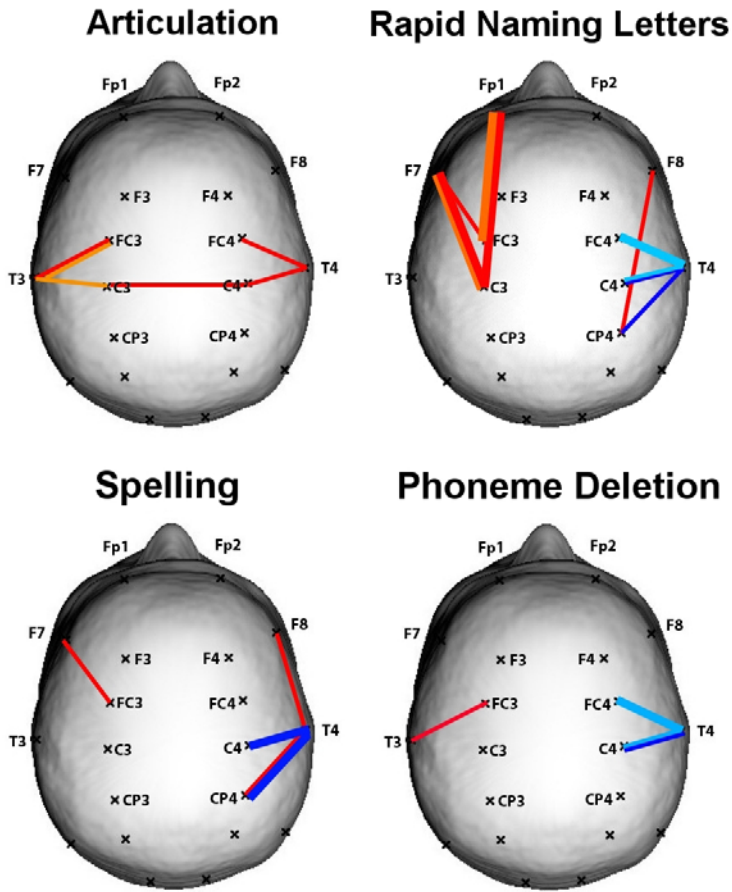


Fig. 3. Significant correlations between dyslexia subscales and significant EEG coherences in the different EEG bands. Red = Delta Coherence; Orange = Theta Coherence; Light Blue = Alpha Coherence; Dark Blue = Beta Coherence. *Thick* lines represent significances of $p < 0.001$ and *thinner* lines of $p < 0.05$. Note the specific independent patterns for some of the patterns especially Articulation with a centro-temporal pattern but also the continuous involvement of the right temporal region for all measures. Also note the clear differences between the slow (Delta and Theta; Red and Orange) vs. higher (Alpha and Beta; Light and Dark Blue) EEG frequencies and the similarities between Delta/Theta and Alpha/Beta.

the bi-laterally increased delta coherence fronto-central and the right fronto-central increased coherence specifically in the alpha and beta band.

4.3. Neuropsychology

The dyslexia group named fewer words in the word condition of the verbal interference test ($F = -6.994$, $df = 36$, $p = 0.012$) but there was no difference on the color condition of the ($F = 2.330$, $df = 36$, $p = 0.136$). The dyslexia group recognized fewer words as compared to the control group ($F = 8.914$, $df = 36$, $p = 0.005$) on the memory recognition task.

Table 1. Significant correlations between coherence values for the different frequency bands vs. the 4 dyslexia subtests. Note that all correlations are rather high and positive, indicating that increased coherence between a given electrode pair is related to better performance on that test.

	Location vs. Subtest	Correlation and Sign
Delta	C4-C3 vs. ART	$r = 0.568$; $df = 17$; $p = 0.017$
	T4-FC4 vs. ART	$r = 0.508$; $df = 17$; $p = 0.037$
	C4-T4 vs. ART	$r = 0.527$; $df = 17$; $p = 0.030$
	T3-FC3 vs. ART	$r = 0.541$; $df = 17$; $p = 0.025$
	T3-FC3 vs. PD	$r = 0.520$; $df = 17$; $p = 0.033$
	C3-F7 vs. RNL	$r = 0.638$; $df = 17$; $p = 0.006$
	C3-Fp1 vs. RNL	$r = 0.662$; $df = 16$; $p = 0.005$
	CP4-F8 vs. RNL	$r = 0.527$; $df = 18$; $p = 0.025$
	FC3-F7 vs. RNL	$r = 0.576$; $df = 17$; $p = 0.015$
	T4-F8 vs. SPL	$r = 0.529$; $df = 17$; $p = 0.029$
	CP4-T4 vs. SPL	$r = 0.491$; $df = 17$; $p = 0.045$
Theta	C3-F7 vs. RNL	$r = 0.598$; $df = 17$; $p = 0.011$
	FC3-Fp1 vs. RNL	$r = 0.772$; $df = 16$; $p < 0.000$
	T3-FC3 vs. ART	$r = 0.527$; $df = 17$; $p = 0.030$
	C3-T3 vs. ART	$r = 0.532$; $df = 17$; $p = 0.028$
Alpha	T4-FC4 vs. RNL	$r = 0.576$; $df = 17$; $p = 0.015$
	T4-FC4 vs. PD	$r = 0.653$; $df = 17$; $p = 0.005$
	C4-T4 vs. RNL	$r = 0.508$; $df = 17$; $p = 0.038$
	C4-T4 vs. PD	$r = 0.565$; $df = 17$; $p = 0.018$
Beta	C4-T4 vs. RNL	$r = 0.501$; $df = 17$; $p = 0.041$
	C4-T4 vs. SPL	$r = 0.617$; $df = 17$; $p = 0.008$
	C4-T4 vs. PD	$r = 0.602$; $df = 17$; $p = 0.011$
	CP4-T4 vs. RNL	$r = 0.521$; $df = 17$; $p = 0.032$
	CP4-T4 vs. SPL	$r = 0.637$; $df = 17$; $p = 0.006$

ART = Articulation
 PD = Phoneme Deletion
 RNL = Rapid Naming Letters
 SPL = Spelling

4.4. *Within group correlations*

The within group correlations were performed on 18 dyslexic children only (one subject was removed from the analysis due to his age). This child was 16 years old whereas the majority of the group was around 10 years of age, and his inclusion may have lead to spurious age-related correlations.

Figure 3 and Table 1 shows the significant correlations between the obtained significant measures reported in the previous section and the sub-tests used to measure the severity of dyslexia. All significant EEG power and EEG coherence measures (63 measures: 8 EEG and 55 coherence) were submitted to the correlation analysis with the four dyslexia sub-tests: Rapid Naming Letters — RNL; Phoneme Deletion — PD, Articulation — ART and Spelling SPL. The results are depicted in

four different colors, each depicting a significant correlation between that variable, between those locations for the given frequency band. The thickness of the line also depicts the significance level (thin $p < 0.05$; thick $p \leq 0.001$).

Interestingly, there were no significant correlations between the EEG power data and the EEG coherence data within frequency bands, hence the increased coherence for dyslexic children cannot be explained by the increased delta and theta frontally.

There was only one significant correlation, between EEG power and the severity of dyslexia: the power of Theta at FP1 and spelling ($r = 0.510$; $df = 16$; $p = 0.044$).

For coherence the significant differences are depicted in Table 1 and are also visually depicted in Fig. 3.

5. Discussion

This study focused on brain function patterns and neuropsychological findings in children with developmental dyslexia and aimed to establish a link between EEG parameters and dyslexia relevant constructs. EEG findings showed an increased (left) frontal and right temporal slow activity in the Delta and Theta bands and increased Beta 1 power at F7. Since all EEG data have been EOG corrected using Gratton *et al.* [11], it is very unlikely the frontal increased Delta and Theta is due to residual EOG. EEG Coherence data showed increased coherence in frontal, central and temporal regions. However, the increased coherences seemed to show a frequency specific effect, where the slower frequencies (delta and theta) showed a more symmetrical increase in right and left frontal, central and temporal networks, whereas the higher frequencies (alpha and beta) showed a more specific right-hemispheric effect originating at T4 and F8. Correlational analysis showed that these increased coherences were an effect in itself, since there were no correlations between the increased delta and theta power on the one hand and the increased coherence in the according band on the other hand; hence the increased delta and theta power were not the cause of the increased coherence findings. High coherence between two EEG signals suggests high cooperation and synchronization between underlying brain regions within a certain frequency band [39]. Increased coherence can thus be interpreted as increased functional connectivity. This could implicate that dyslexic children have increased activity within frontal, central and temporal networks.

There were also significant differences between the dyslexic and the control group on the verbal interference tests (similar to the Stroop test) and the memory recognition test. These findings are directly related to the dyslexic problems experienced by this group, since dyslexic children have decoding problems. The dyslexic children named fewer words on the verbal interference test than the control children but had no impairment when required to name the color of the word relative to normal children. Dyslexic children also recognized fewer words on a memory recognition test whereas spontaneous memory recall was not affected at all. These findings suggest that interpretation of neuropsychological data derived from these specific tests should always be treated with caution, and may be that dyslexic status should be

incorporated into the interpretation of neuropsychological data to safeguard false positive findings on these tests.

Correlational analyses revealed significant correlations between the obtained significant EEG findings and the tests: articulation, rapid naming of letters, spelling and phoneme deletion. The correlated patterns (as depicted in Fig. 3) showed quite specific patterns for all these 4 sub-tests. Interestingly, all these correlations were positive and high (explaining $> 30\%$ of the variance), suggesting that better performance on these tests was associated with increased coherence. Given the fact that all the coherence findings were increased in comparison to the control group, it might be concluded that these patterns reflect compensatory mechanisms and do not explain the deficit *per se* (where negative correlations would be expected). The EEG in this study was not recorded during the completion of the dyslexia specific tests, hence we recorded resting state eyes open EEG which correlated highly with these tests. This further supports the fact that these patterns can be considered compensatory patterns since they are also present at rest. Furthermore, this demonstrates that clear associations can be found between passive brain states and deviant behavior, demonstrating the utility of integrative approaches.

There seems to be a clear distinction between delta coherence on the one hand and beta coherence on the other. The increased coherence for dyslexic children was prominent and symmetric for the delta band; but localized to the left hemisphere for the beta band. The correlations also demonstrate this; a slow (delta and theta) coherent network over left frontal and central regions, and a faster (alpha and beta) network originating at T4. Although EEG coherence between different cortical regions is largely established by cortico-cortical and thalamo-cortical interactions [24], subcortical brain areas also contribute to both inter- and intra-hemispheric functional communication [3]. Lower bandwidths such as delta frequency in the EEG coherence spectrum have particularly been associated with limbic contributions to cortico-cortical coupling [18], hence these increased low-frequency coherences could indicate a limbic contribution.

The core dysfunction in dyslexia seems to consist of increased slow activity at left frontal and right temporal (T6) regions, and bilateral increased coherence in the slower frequency bands (delta and theta), as opposed to acquired-compensatory mechanisms consisting of right-hemispheric increased coherences in the higher frequency bands (alpha and beta) and a left frontal increased coherence in slower bands originating from C3 and FC3. The increases in coherence in the delta band fronto-central suggest a strong limbic involvement as part of the core deficit in dyslexia, although this requires further study.

In this study, children showed delays in both rapid naming and phonological awareness. These delays correlated with the activation in the frontal-cerebellar phonological system. The EEG findings in our study showed an increased activation pattern in dyslexic children, mainly in frontal and temporal lobes. Furthermore, the correlation analyses showed significant correlations with spelling, phonological skills and rapid naming with quite different topographical representations, suggesting

involvement of different neural mechanisms. It can tentatively be concluded that the frontal-cerebellar network may be critical to the precise timing of mechanisms that underlie the double-deficit theory of dyslexia, suggesting the existence of three subtypes of reading disability: dyslexics with deficiencies in phonological skills, poor rapid naming skills or a combination of both types. Thus, the present study supports the theory of Eckart *et al.* [4] hypothesizing that impairments in a frontal-cerebellar network may play a role in delayed reading impairment in dyslexia. These authors reported that anomalies in a cerebellar-frontal circuit are associated with rapid automatic naming and phonological processing.

Previous EEG studies have shown different findings. Rippon and Brunswick [28] found no specific activation patterns with respect to dyslexic children. Weiss and Mueller [39] have proposed several roles for coherence in the different frequency bands (also see introduction), however in this study, we did not use a task-related protocol, making comparison to this study difficult.

This study contributes to the theory that neurobiological causes underlie dyslexia. The increased activation patterns of dyslexic children seem to be associated with the double deficit type of dyslexia. In future research, it will be important to examine the relation between EEG data and the phonological or orthographic deficits. Outcomes of these studies might further contribute to the diagnosis of subtypes of dyslexia.

Finally, this study demonstrated that increased EEG power could not explain the increased coherence findings in dyslexia, suggesting these measures reflect different neural networks. The positive correlations between coherence and the different tests demonstrated that these increased coherences might reflect compensatory mechanisms rather than being part of the real core dysfunction in dyslexia, whereas the increased slow activity might be part of the core dysfunction in dyslexia. This should be taken into account in future studies to elucidate dysfunctional networks in dyslexia. These dysfunctional networks can be dissociated from acquired compensatory mechanisms. Also, treatments focused on normalizing brain function (e.g., rTMS, EEG Biofeedback or Neurofeedback) will benefit from this given they could target the deficit rather than target acquired compensatory mechanisms.

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