ORIGINAL ARTICLE

Electroencephalogram (EEG) Hemispheric Asymmetry and Regional Power Distribution During Executive Functioning Tasks in Children with Suspected Specific Learning Disabilities: A Preliminary Study

Nootchanart Ruksee, PhD[1], Chutikorn Nopparat, PhD[2], Vorasith Siripornpanich, MD, PhD[3], Vasunun Chumchua, PhD[1], Thirata Khamnong, PhD[1], Sarun Kunwittaya, PhD[1], Adisak Plitponkarnpim, MD, MPH[1,4], Kerry Lee, PhD[5], Khanokporn Donjdee, PhD[1], Rahmat Ryadhush Shalihin, PhD[1], Kannika Permpoonputtana, PhD[1]

[1] National Institute for Child and Family Development, Mahidol University, Nakhon Pathom, Thailand; [2] Innovative Learning Center, Srinakharinwirot University, Bangkok, Thailand; [3] Research Center for Neuroscience, Institute of Molecular Biosciences, Mahidol University, Nakhon Pathom, Thailand; [4] Department of Pediatrics, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand; [5] Yew Chung College of Early Childhood Education, Hong Kong, China

Background: Quantitative spectral analysis of electroencephalogram (EEG) signals has been widely applied to investigate the neurobiological underpinnings of neurodevelopmental disorders, including specific learning disabilities (SpLDs).

Objective: To examine inter-hemispheric asymmetries and EEG power activation during executive task performance in children with suspected SpLDs identified through academic screening.

Materials and Methods: A cross-sectional observational design was employed. Nineteen school-aged children with suspected SpLDs were recruited through teacher referral and academic screening using the Kasetsart Basic Academic Skills Test (KBAST). All participants met the inclusion criteria of average intelligence calculated as IQ of 70 or above, and below-average performance in at least one KBAST domain. EEG signals were recorded using an 8-channel system while participants performed the Tasks of Executive Control (TEC). Each EEG session lasted 15 to 20 minutes. Absolute power across nine frequency bands between 0.5 to 45 Hz, was computed using Fast Fourier Transformation (FFT). Hemispheric asymmetries were analyzed using Wilcoxon signed-rank tests, and regional differences were examined using Kruskal-Wallis tests with Games-Howell post hoc analysis.

Results: The results indicated spectral differences in EEG power across hemispheric and regional sites during executive task performance. Children with suspected SpLDs showed distinct EEG power distribution during executive task performance. Right-hemisphere power was significantly greater than left-hemisphere power in the upper-theta ($p=0.02$) and lower-alpha ($p=0.05$) bands. Frontal regions exhibited significantly higher lower-delta ($p=0.02$) and upper-delta ($p<0.001$) activity than central regions. Low-frequency activity in delta, theta, and lower-alpha was most prominent at prefrontal and frontal sites.

Conclusion: The findings indicate atypical hemispheric lateralization and elevated low-frequency EEG activity during executive task engagement in children with suspected SpLDs. The EEG indicated suboptimal neural resource allocation and clear hemispheric and regional asymmetries during executive task engagement in children with suspected SpLDs, but they should be interpreted cautiously given the small sample size and absence of a control group. Further controlled studies with larger samples are needed to clarify the specificity, developmental significance, and potential clinical relevance of these preliminary observations.

Keywords: Electroencephalogram; Executive function; Specific learning disabilities

Received 20 October 2025 | Revised 12 December 2025 | Accepted 15 December 2025

J Med Assoc Thai 2026;109(1):70-81

Website: http://www.jmatonline.com

Correspondence to:

Permpoonputtana K.

National Institute for Child and Family Development, Mahidol University, Nakhon Pathom 73170, Thailand.

Phone: +66-87-7911213, Fax: +66-2-4410167

Email: kannika.per@mahidol.ac.th

How to cite this article:

Ruksee N, Nopparat C, Siripornpanich V, Chumchua V, Khamnong T, Kunwittaya S, Plitponkarnpim A, Lee K, Donjdee K, Shalihin RR, Permpoonputtana K. Electroencephalogram (EEG) Hemispheric Asymmetry and Regional Power Distribution During Executive Functioning Tasks in Children with Suspected Specific Learning Disabilities: A Preliminary Study. J Med Assoc Thai 2026;109:70-81.

DOI: 10.35755/jmedassocthai.2026.1.03754

Specific learning disabilities (SpLDs) are neurodevelopmental disorders that significantly impair academic achievement in primary school children. They are manifested by persistent difficulties in acquiring skills in reading as dyslexia, writing as dysgraphia, and mathematics as dyscalculia. SpLDs affect an estimated 5% to 15% of school-aged children worldwide and represent one of the most prevalent causes of academic underachievement[1,2]. Environmental and psychosocial factors, such as socio-economic disadvantages, lack of educational

support, and emotional challenges, exacerbate learning difficulties. However, SpLDs are primarily linked to atypical brain development and central nervous system dysfunction, and frequently persist into adolescence and adulthood[3,4].

Prevalent studies in Asia demonstrate that SpLDs are a significant concern beyond Western populations. In India, approximately 5% to 12% of school-aged children are affected, with one in twelve Indian children having SpLDs[5,6]. In Hong Kong, the prevalence of dyslexia is estimated at 9.7%[7]. In Brazil, estimates indicate that the prevalence rates of SpLDs are approximately 7.6% for global impairment. In Turkey, the prevalence rate has been recorded at 13.6%, while in Pakistan, the rate among primary school children is approximately 7.7%[5].

Furthermore, studies indicate that 6% to 10% of children in Thailand experience challenges related to SpLDs, such as dyslexia and dyscalculia[8]. A study in Bangkok found that 21.76% of students in six schools had SpLDs[9]. SpLDs are strongly linked to broader impairments in cognitive processing, particularly in executive functions (EFs). However, research in Thailand has not yet systematically examined how executive dysfunction presents at the neural level in children with SpLDs. Besides, the deficits in inhibition, working memory, and cognitive flexibility have been consistently documented in children with SpLDs[10-12]. These disabilities can hinder attention regulation, task monitoring, and adaptive learning[13,14].

The frontal lobes are the central component in EFs, including attention regulation, working memory, cognitive shifting, and inhibitory control[15,16]. Dysfunctions in these regions have been reported in children with SpLDs and contribute to difficulties in planning, regulating, and executing goal-oriented behaviors[17,18]. However, there has been limited research investigating the neurophysiological correlations of executive dysfunction, particularly regarding localized brain activation during executive-related tasks. Previous studies have primarily focused on behavioral assessments that establish a link between EF deficits and SpLDs[19,20].

Although there has been limited emphasis on brainwave activation during EF tasks, electro-encephalography (EEG) provides an effective means of non-invasive[21] for examining the temporal[22] and spectral aspects of neural activity related to these processes[23]. EEG is a sensitive tool for examining spectral dynamics that underpin EF processes and contribute to the spatial and temporal organization of neural activity. It indicates that the electrical functioning of the brain during EF tasks varies between students with learning disabilities and those who are typically developing[24]. In addition, children with SpLDs often exhibit atypical EEG signatures, such as elevated frontal theta and altered alpha rhythm, which are associated with delayed cortical maturation[22] and inefficient cognitive control[25,26].

Unfortunately, previous studies have focused solely on resting-state[25,27] or generalized activity[22]. Theta activity and cognitive functioning is integrating evidence from resting-state and task-related developmental EEG research, with relatively lack of focus given to task-related activation patterns. Hence, the present study aimed to investigate inter-hemispheric asymmetries and regional EEG power activation during executive task performance in children with suspected SpLDs. The present study employed the Tasks of Executive Control (TEC) as a validated measure designed to elicit working memory and inhibitory control states[28]. The direct elicitation aimed to capture neurophysiological responses associated with these specific cognitive states.

The analysis of localized EEG activation during these tasks has the potential to deepen understanding of the neurophysiological mechanisms underlying executive dysfunction in SpLDs and to provide new insights into atypical hemispheric specialization. Therefore, the findings of this study will contribute to the development of targeted interventions. The neurophysiological biomarkers can aid in the early identification and tailored educational support for children.

Materials and Methods

The present study employed a cross-sectional observational design to examine executive functioning and neural activity in school-aged children with suspected SpLDs. All assessments and EEG recordings were conducted within the same session for each participant to ensure data consistency and temporal alignment of cognitive and neurophysiological measures.

Participants and procedure

Twenty-three children were initially screened for participation. Four were excluded due to incomplete consent or EEG artefacts. The sample size of 19 with suspected SpLDs was determined based on feasibility and alignment with prior EEG pilot studies involving similar populations, which

were from 15 to 25 patients, where such a sample size was sufficient to detect large within-group effects[25]. All participants were right-handed, had normal or corrected-to-normal vision, and reported no history of neurological or psychiatric conditions beyond their academic difficulties. Children were referred to the study by their teachers due to ongoing learning problems and were voluntarily recruited. Data was collected across three provinces in central Thailand, which were Phitsanulok, Phichit, and Phetchabun, in collaboration with local primary schools. All assessments took place in a quiet and distraction-free environment within the school setting to ensure optimal concentration. To ensure the exclusion of intellectual disability, participants were required to have an average intelligence quotient (IQ) score of 70 or above on the Test of Non-verbal Intelligence, Fourth Edition (TONI-4)[29]. Intellectual disability is formally defined by an IQ score of 70 or below, coupled with lower-than-expected adaptive functioning and an onset in early childhood[30]. The exclusion criteria were a history of neurological or psychiatric disorders, except for the diagnosis of SpLDs. In addition, no participants withdrew after data collection commenced, and all variables had complete data with no missing value.

Meanwhile, the inclusion criteria consisted of participants of all genders, an average IQ score, and enrolment in grades 4 to 6. The participants did not exhibit any psychological concerns beyond their SpLDs. In the present study, SpLDs were defined as the Kasetsart Basic Academic Skills Test (KBAST) scoring below average in at least one domain. Besides, the ethics approval was granted by the Mahidol University Central Institutional Review Board (COA No. MU-CIRB 2019/067.2602). Prior to testing, written informed consent was obtained from all parents for their child's participation. Parents were also required to complete the demographic questionnaires. The participants were assessed by the TONI-4 and the KBAST on the first day. SpLDs were suspected in cases where participants scored poorly in at least one domain and demonstrated below-average performance in another domain such as word reading, word spelling, sentence comprehension, or math computation. On the following day, the EEG was recorded while participants performed executive tasks in a distraction-free environment to facilitate their concentration on the tasks[31].

Instruments

The research instruments comprised the validated instruments TONI-4, KBAST, and a brief demographic questionnaire. Caregivers completed the demographic questionnaire, which included questions regarding gender, age, marital status, educational background, therapy, and current school accommodations. The TONI-4 assessed fluid intelligence in individuals aged 6 to 89 years, 11 months[29]. The test comprises 45 matrix thinking questions with raw scores ranging from 0 to 45. These scores were then transformed into deviation quotients using the test manual. The rater records the score as 0 or 1, on the answer sheet. The TONI-4 is norm-referenced and produces an index (quotient) with a mean of 100 and standard deviation (SD) of 15. Higher index scores indicate better fluid intelligence[29]. Meanwhile, the KBAST is a basic academic skills test used for students in grades 1 to 6 that evaluates four domains, including word reading (RKBAST), word spelling (SKBAST), sentence comprehension (UKBAST), and mathematics computation (MKBAST). In the present study, the KBAST has good internal consistency, with a Cronbach's alpha greater than 0.8. The raw scores are transformed into percentiles according to the norms of the KBAST. A standard score below 90 is classified as below average in basic academic skills compared with the same age. Students who scored below average in at least one domain are suspected of having SpLDs.

EEG recording

Before the EEG recording in executive task condition, all parents and children were instructed about the procedure, and informed consent was obtained. An 8-channel EEG device (eego, ANT Neuro, Berlin, Germany) was used to record EEG signals, which were then digitized at a sampling rate of 1,000 Hz. Electrodes as Fpz, Fz, F3, F4, Cz, C3, C4, and Pz, were placed over the frontal and parietal areas in accordance with the extended 10 to 20 international system[32]. All electrode impedances were kept below 10 kΩ, and the EEG signal was digitized at 256 Hz with a 30 Hz low-pass filter. Environmental bias was controlled by conducting all EEG sessions in quiet rooms. Immediately after resting EEG, the recording of EEG continued as the executive tasks were administered. Participants with suspected SpLDs were asked to concentrate on computer-based executive tasks displayed on the screen. EEG data were stored, and real-time data were shown on a computer. The Fast Fourier Transformation (FFT) was used to compute absolute power data for the following frequency bands as

lower delta for 0.5 to 2 Hz, upper delta for 2 to 4 Hz, lower theta for 4 to 6 Hz, upper theta for 6 to 8 Hz, lower alpha for 8 to 10 Hz, upper alpha for 10 to 12 Hz, lower beta for 12 to 16 Hz, upper beta for 16 to 30 Hz, and gamma for 30 to 45 Hz. In addition, the powers corresponding to each of these frequency bands were also derived.

Statistical analysis

EEG data were processed utilizing FFT to derive absolute power values across nine frequency bands, which were 0.5 to 45 Hz, for eight electrode sites. Descriptive statistics were calculated for each frequency band and electrode region and are presented as the median (interquartile range, IQR). To examine hemispheric asymmetries, non-parametric Wilcoxon signed-rank tests were employed to compare power spectra between the left (F4) and right (F3) frontal regions. Regional differences across frontal, central, and parietal electrodes were analyzed using the Kruskal-Wallis test, followed by Games-Howell post hoc comparisons to accommodate unequal variances and sample sizes. Statistical significance was set at p-value less than 0.05. All analyses were performed using IBM SPSS Statistics, version 25.0 (IBM Corp., Armonk, NY, USA).

Results

Characteristics of participants

Nineteen school-aged children, including eleven males and eight females, aged 9 to 12 years, with a mean age of 11.2 and standard deviation (SD) of 0.99, identified as having suspected SpLDs participated in this study. All were volunteers from grades 4 to 6 across six elementary schools and had been referred by their teachers because of academic difficulties. Participants' IQ scores and academic skills are summarized in Table 1. On the KBAST, with mean standard scores of 100 (SD 15), the sample showed clear weaknesses across multiple domains. Mean scores, and proportion scoring below 90, for the 19 students were 75.83 for word reading with 14 (73.68% below average), 75.67 for word spelling with 16 (84.21% below average), 82.94 for sentence comprehension with 15 (78.95% below average), and 72.55 for math computation with 16 (84.21% below average). This pattern of overlapping below-average performance across literacy and numeracy domains supports describing the group as children with suspected SpLDs rather than assigning them to single SpLD subtypes.

Table 1. The distribution of the participants with complaints of learning disabilities according to their demographic data

Participants' characteristics	n=19
Child age (years); mean	9.2
Sex (male:female)	11:8
Grade; n (%)	
Grade 4	3 (15.79)
Grade 5	2 (10.53)
Grade 6	14 (73.68)
IQ scores; mean	92.52
Kasetsart Basic Academic Skills Test; mean, n (%)	
Word reading skill, standard score <90	75.83, 14 (73.68)
Word spelling skill, standard score <90	75.67, 16 (84.21)
Sentence comprehension skill, standard score <90	82.94, 15 (78.95)
Math computation skill, standard score <90	72.55, 16 (84.21)

Hemispheric asymmetries in frontal EEG power

The data revealed hemispheric asymmetries that were most pronounced in the theta and alpha frequency bands. Upper-theta power was significantly higher in the right hemisphere compared with the left ($z=-2.19$, $p=0.02$). It indicated atypical right-hemispheric dominance during executive processing. A similar trend occurred for lower-alpha activity, which was greater in the right hemisphere ($z=-1.93$, $p=0.05$). These findings indicate altered frontal lateralization, as typical development often showed left-hemispheric engagement in executive and linguistic processing. Table 2 presents the comparison of EEG power spectra between the left (F4) and right (F3) frontal regions. Therefore, there were no significant hemispheric differences in delta, beta, or gamma frequency bands.

Regional differences in EEG power across frontal, central, and parietal electrodes

In Figure 1, the mean EEG power spectra at 0.5 to 45 Hz, were presented for eight scalp electrodes located over prefrontal, frontal, central, and parietal regions while children with SpLDs performed executive control tasks. The x-axis represents frequency (Hz) and the y-axis represents absolute spectral power (μV^2), illustrating the distribution of power across frequency bands at each electrode and the relative predominance of low-frequency activity in frontal sites. Regional differences were most evident in the delta, theta, and alpha ranges. In particular, lower-delta power was significantly higher in the frontal region than in the central region, with a mean difference of 85.41 μV^2 ($p=0.02$). Similarly, upper-delta power was significantly greater in the

Table 2. Frontal EEG power spectral density in the left (F4) and right (F3) hemispheres during executive function in children with suspected specific learning disabilities

EEG	Right hemisphere (F3); mean (IQR)	Left hemisphere (F4); mean (IQR)	z	p-value
Lower delta	133.53 (77.28, 308.87)	164.50 (90.85, 493.35)	–0.53	0.59
Upper delta	96.20 (67.03, 168.99)	96.83 (72.16, 187.13)	–0.15	0.87
Lower theta	37.90 (28.87, 53.91)	32.29 (29.35, 37.86)	–1.10	0.27
Upper theta	20.38 (15.03, 23.15)	15.66 (13.29, 18.08)	–2.19	0.02*
Lower alpha	10.25 (8.91, 15.81)	8.63 (6.62, 12.42)	–1.93	0.05*
Upper alpha	8.57 (5.42, 13.09)	5.61 (4.11, 11.63)	–1.76	0.07
Lower beta	10.30 (5.91, 18.24)	5.79 (4.65, 13.09)	–1.83	0.06
Upper beta	12.93 (8.80, 24.60)	7.82 (6.71, 22.47)	–1.45	0.15
Gamma	1.75 (1.37, 3.35)	2.21 (1.25, 3.14)	–0.03	0.98

IQR=interquartile range

* Statistical significance, $p<0.05$

frontal region than in the central region, with a mean difference of 47.36 ($p<0.001$), with a trend towards higher power relative to the parietal region ($p=0.06$). In the theta band, lower-theta power was significantly elevated in the frontal compared with the central region, with a mean difference of 8.21 ($p=0.007$), whereas upper-theta activity showed no significant regional variation. Table 3 summarizes the post-hoc comparisons of EEG power between frontal, central, and parietal regions across frequency bands.

In the alpha range, a significant effect emerges for upper-alpha power, where central regions demonstrate greater activity than frontal regions, with a mean difference of 4.69 ($p=0.04$). Lower-alpha differences were not significant, and no regional differences occurred for either beta, lower or upper, or gamma frequencies. These results indicated that spectral differences across cortical regions were concentrated in low-frequency, at delta and theta, and mid-frequency, at alpha bands. Specifically, frontal regions exhibit stronger delta and theta activity. In addition, the central regions showed relative dominance in upper-alpha power and indicate differential cortical engagement during executive processing in children with suspected SpLDs.

Discussion

The present study provides preliminary evidence of atypical hemispheric distribution of EEG activity during EF tasks in children with suspected SpLDs, with a relative predominance of theta and alpha activity in right-hemispheric regions. This pattern appears to differ from the typical left-lateralized frontal specialization for executive and language-related functions reported in typically developing children, which has been linked to minimal hypo arousal and readiness for task activation[25]. Children with dyslexia, in contrast, frequently do not demonstrate the typical left-hemispheric specialization and may display atypical patterns of hemispheric lateralization[25]. Tabiee et al. (2023) also suggest that alpha synchronization and increased theta activity may be associated with poor attention and memory processes in dyslexic individuals[33]. Similarly, a relative predominance of right-hemispheric alpha2 and beta activity in the resting state has been reported in children with dyslexia[25], and impairment of right-hemisphere frontoparietal attention circuitry has been proposed as a potential contributing mechanism in developmental dyslexia[34]. Elevated right-hemispheric theta has also been linked to delayed cortical maturation and hypo arousal mechanisms in dyslexia and related learning disorders[25]. However, findings across studies are not uniform. For example, another study reported a predominance of left-hemispheric theta activity at rest in dyslexia[35].

Previous work has therefore not yet converged on a single pattern that reliably differentiates children with dyslexia from typically developing peers during reading-related tasks[34,35]. Within this context, the present study results add preliminary observations of atypical hemispheric asymmetry during executive states in children with suspected SpLDs. Rather than indicating a definitive diagnostic pattern, these findings may be consistent with hypotheses of underlying inefficiencies in executive-network recruitment, learning unreadiness, for example, delayed maturation or hypo arousal, and possible alterations in functional lateralization. Given the absence of a typically developing control group in the present study, these interpretations should be regarded as tentative and hypothesis-generating.

Similarly, increased alpha asymmetry has been

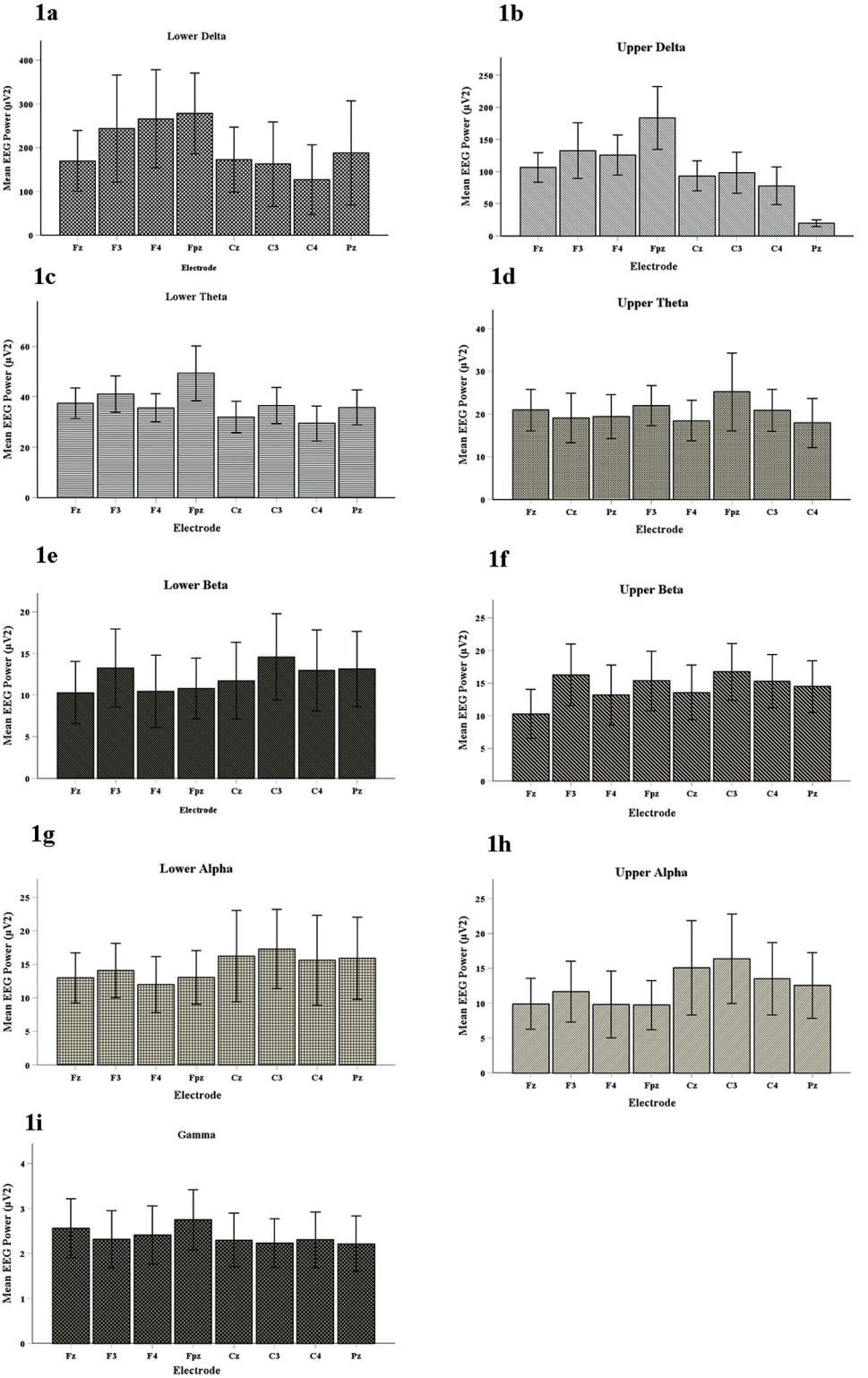

Figure 1. Mean EEG power spectra (0.5 to 45 Hz) recorded from eight scalp electrodes (Fpz, Fz, F3, F4, Cz, Pz, P3, P4) over prefrontal, frontal, central, and parietal regions while children with suspected SpLDs performed executive control tasks. The x-axis represents frequency (Hz), and the y-axis represents absolute spectral power (μV^2), showing the average distribution of power across frequency bands for each electrode.

Table 3. Multiple comparisons of EEG power between brain regions using Games-Howell post-hoc test

EEG	(I) Electrode	(J) Electrode; median (IQR)	SE	p-value	Mean difference (95% CI)
Lower delta	Frontal	Central 103.76 (60.72, 272.73)	33.05	0.02	85.41* (6.98 to 163.84)
		Parietal 112.34 (63.95, 176.95)	61.49	0.68	51.65 (–102.15 to 205.46)
	Central	Frontal 139.83 (76.09, 228.83)	33.05	0.02	–85.41* (–163.84 to –6.98)
		Parietal 112.34 (63.95, 176.95)	61.03	0.84	–33.76 (–186.74 to 119.21)
	Parietal	Frontal 139.83 (76.09, 228.83)	61.49	0.68	–51.65 (–205.46 to 102.15)
		Central 103.76 (60.72, 272.73)	61.03	0.84	33.76 (–119.21 to 186.74)
Upper delta	Frontal	Central 79.08 (52.75, 139.62)	12.20	0.000	47.36* (18.40 to 76.32)
		Parietal 78.91 (51.86, 150.21)	16.81	0.06	39.04 (–2.12 to 80.22)
	Central	Frontal 101.70 (67.83, 135.05)	12.20	0.000	–47.36*** (–76.32 to –18.40)
		Parietal 78.91 (51.86, 150.21)	15.90	0.86	–8.32 (–47.66 to 31.02)
	Parietal	Frontal 101.70 (67.83, 135.05)	16.81	0.06	–39.04 (–80.22 to 2.12)
		Central 79.08 (52.75, 139.62)	15.90	0.86	8.32 (–31.02 to 47.66)
Lower theta	Frontal	Central 28.32 (25.89, 34.40)	2.65	0.007	8.21** (1.91 to 14.51)
		Parietal 31.86 (25.00, 48.52)	3.78	0.38	5.05 (–4.27 to 14.38)
	Central	Frontal 33.75 (29.80, 44.54)	2.65	0.007	–8.2** (–14.51 to –1.91)
		Parietal 31.86 (25.00, 48.52)	3.76	0.68	–3.15 (–12.45 to 6.14)
	Parietal	Frontal 33.75 (29.80, 44.54)	3.78	0.38	–5.05 (–14.38 to 4.27)
		Central 28.32 (25.89, 34.40)	3.76	0.68	3.15 (–6.14 to 12.45)
Upper theta	Frontal	Central 14.16 (12.64, 22.68)	2.08	0.51	2.30 (–2.63 to 7.25)
		Parietal 15.75 (12.96, 20.92)	2.84	0.72	2.19 (–4.81 to 9.20)
	Central	Frontal 17.32 (14.69, 24.88)	2.08	0.51	–2.30 (–7.25 to 2.63)
		Parietal 15.75 (12.96, 20.92)	2.86	0.99	–0.11 (–7.16 to 6.94)
	Parietal	Frontal 17.32 (14.69, 24.88)	2.84	0.72	–2.19 (–9.20 to 4.81)
		Central 14.16 (12.64, 22.68)	2.86	0.99	0.11 (–6.94 to 7.16)
Lower alpha	Frontal	Central 10.30 (6.39, 22.89)	1.97	0.21	–3.35 (–8.07 to 1.36)
		Parietal 9.62 (8.72, 23.35)	3.05	0.62	–2.88 (–10.59 to 4.82)
	Central	Frontal 9.50 (7.91, 17.50)	1.97	0.21	3.35 (–1.36 to 8.07)
		Parietal 9.62 (8.72, 23.35)	3.39	0.98	0.47 (–7.88 to 8.83)
	Parietal	Frontal 9.50 (7.91, 17.50)	3.05	0.62	2.88 (–4.82 to 10.59)
		Central 10.30 (6.39, 22.89)	3.39	0.98	–0.47 (–8.83 to 7.88)
Upper alpha	Frontal	Central 8.19 (3.70, 28.10)	1.92	0.04	–4.69* (–9.27 to –.11)
		Parietal 8.81 (5.63, 14.75)	2.42	0.62	–2.28 (–8.35 to 3.78)
	Central	Frontal 6.69 (4.81, 12.62)	1.92	0.04	4.69* (0.11 to 9.27)
		Parietal 8.81 (5.63, 14.75)	2.78	0.66	2.40 (–4.38 to 9.19)
	Parietal	Frontal 6.69 (4.81, 12.62)	2.42	0.62	2.28 (–3.78 to 8.35)
		Central 8.19 (3.70, 28.10)	2.78	0.66	–2.40 (–9.19 to 4.38)
Lower beta	Frontal	Central 9.43 (4.30, 15.67)	1.64	0.48	–1.90 (–5.80 to 2.00)
		Parietal 9.37 (6.65, 17.55)	2.34	0.68	–1.94 (–7.79 to 3.89)
	Central	Frontal 7.08 (5.00, 14.33)	1.64	0.48	1.90 (–2.00 to 5.80)
		Parietal 9.37 (6.65, 17.55)	2.51	1.00	–0.04 (–6.22 to 6.12)
	Parietal	Frontal 7.08 (5.00, 14.33)	2.34	0.68	1.94 (–3.89 to 7.79)
		Central 9.43 (4.30, 15.67)	2.51	1.00	0.048 (–6.12 to 6.22)
Upper beta	Frontal	Central 10.37 (6.09, 21.80)	1.54	0.85	–0.83 (–4.50 to 2.82)
		Parietal 10.82 (7.86, 23.26)	2.14	0.99	–0.10 (–5.42 to 5.20)
	Central	Frontal 9.37 (7.28, 18.38)	1.54	0.85	0.83 (–2.82 to 4.50)
		Parietal 10.82 (7.86, 23.26)	2.20	0.94	0.73 (–4.68 to 6.15)
	Parietal	Frontal 9.37 (7.28, 18.38)	2.14	0.99	0.10 (–5.20 to 5.42)
		Central 10.37 (6.09, 21.80)	2.20	0.94	–0.73 (–6.15 to 4.68)
Gamma	Frontal	Central 2.25 (1.29, 3.24)	0.21	0.54	0.23 (–0.28 to 0.75)
		Parietal 1.76 (1.17, 2.92)	0.33	0.65	0.29 (–0.52 to 1.11)
	Central	Frontal 2.52 (1.24, 3.63)	0.21	0.54	–0.23 (–0.75 to 0.28)
		Parietal 1.76 (1.17, 2.92)	0.33	0.98	0.06 (–0.76 to 0.88)
	Parietal	Frontal 2.52 (1.24, 3.63)	0.33	0.65	–0.29 (–1.11 to 0.52)
		Central 2.25 (1.29, 3.24)	0.33	0.98	–0.06 (–0.88 to 0.76)

IQR=interquartile range; SE=standard error; CI=confidence interval

* Statistical significance, * p<0.05, ** p<0.01, *** p<0.001

interpreted by others as a possible compensatory reliance on right-hemispheric networks when left-frontal processing is less efficient[36]. In the present study data, the pattern of right-hemispheric predominance in low-frequency and alpha activity is broadly in line with this view and with prior quantitative EEG studies reporting excess theta power in children with learning difficulties. Such excess theta has been associated with lagged functional brain development, academic underachievement[37], and executive difficulties[26]. More severe learning problems have also been associated with lower IQ in some samples[37-39].

In contrast, all participants in the present study were required to demonstrate at least average non-verbal intellectual functioning, with an IQ of 70 or above, on the TONI-4. This methodological choice was intended to minimize the contribution of global intellectual disability and to focus on learning difficulties related to SpLD-specific neurocognitive mechanisms such as executive dysfunction and atypical neural processing, rather than generalized cognitive limitations. This approach aligns with the growing view that SpLDs are not inherently determined by IQ and that routine IQ testing may have limited added value for identification in many cases[40]. The present study findings support the notion that children with SpLDs can show learning difficulties despite broadly age-appropriate non-verbal cognitive potential, which is relevant for how assessment and educational planning are conceptualized, even though direct clinical implications cannot be drawn from this preliminary dataset alone.

The regional distribution of EEG spectral power observed here may also provide insight into executive-processing demands in this group. Frontal regions showed stronger low-frequency, of delta and theta activity than central regions, particularly during executive task demands. This pattern is broadly consistent with previous reports linking frontal slow-wave activity to inefficient executive control and delayed cortical maturation in children with learning difficulties[25,41]. Elevated frontal delta activity has been associated with reduced cortical arousal and diminished task-related alertness[42], although it may additionally contribute to the suppression of irrelevant neural activity and the filtering of distractions during cognitive engagement[43]. Increased frontal theta power has long been implicated in memory encoding, retention, and working memory processes[44-46]. In contrast, the relatively greater upper-alpha activity in central regions compared with frontal regions is consistent with the proposed role of alpha oscillations in attentional control, cortical inhibition, and sensory-motor integration[47].

Developmentally, delta, theta, and lower-alpha activity tend to decline with age, whereas upper-alpha power increases as the brain matures[48]. Within this framework, the elevated low-frequency power observed in the present study suspected SpLD sample may be compatible with delayed or altered neurodevelopmental trajectories, particularly in frontal networks. This interpretation is also in line with evidence highlighting the central role of the prefrontal cortex in EFs such as working memory, inhibitory control, and cognitive flexibility[49-51]. The presence of right-lateralized increases in upper-theta and lower-alpha power in the present study data may suggest that some children with suspected SpLDs rely more heavily on right-hemispheric prefrontal mechanisms during executive processing, potentially reflecting compensatory strategies when left-hemispheric recruitment is suboptimal. This pattern differs from the predominantly left-lateralized activation reported in typical children during phonological working-memory tasks[52,53], and resonates with prior findings of abnormal hemispheric asymmetry in dyslexia and SpLDs, particularly in circuits supporting phonological working memory and inhibitory control[54,55].

Right prefrontal regions have been specifically linked to supervisory control and response inhibition[15,56], and imbalances in frontal alpha activity have been associated with impulsivity and executive dysfunction[57]. In light of this literature, the present study findings tentatively suggest that children with suspected SpLDs in this sample may exhibit a distinct pattern of cortical engagement characterized by excessive low-frequency frontal activity, atypical hemispheric asymmetry, and altered alpha modulation during executive states. These patterns may reflect a combination of maturational lag and compensatory neural recruitment and could help to explain some of the observed inefficiencies in executive functioning. However, because this was a preliminary study without a control group and with a small sample size, the observed EEG signatures in the delta, theta, and alpha bands should currently be viewed as candidate neurophysiological correlates rather than established biomarkers. Future research incorporating larger samples, longitudinal designs, and direct comparisons with typically developing peers will be essential to determine the specificity,

stability, and potential clinical relevance of these EEG patterns in SpLDs.

Limitation

Limitations warrant caution in interpreting these findings. First, the absence of a typically developing control group prevents us from determining whether the observed spectral and asymmetric patterns are specific to SpLDs or reflect more general features of childhood neurodevelopment. Second, the small sample size with 19 students limits statistical power and generalizability and may partly account for the lack of robust effects in higher-frequency bands. Consequently, the present results should be regarded as exploration and hypothesis-generating. Future studies with larger samples, well-matched control groups, and longitudinal designs are needed to clarify whether these spectral characteristics reflect maturational delay, persistent atypical organization, or compensatory mechanisms.

Within these constraints, the present study findings add to the emerging literature on the neurophysiological correlates of SpLDs and highlight the potential usefulness of EEG as a non-invasive method for probing executive-related brain activity. Quantitative EEG (qEEG) measures, particularly in the delta, theta, and alpha ranges, may contribute to a more nuanced understanding of how executive networks function in children with SpLDs. However, further research, integrating EEG with behavioral and other neuroimaging data, is essential before any firm conclusions can be drawn about their specificity, stability, or possible clinical utility in assessment or intervention planning.

Implication

Hence, the present study contributes to the neurophysiological correlations of SpLDs and highlights the potential of EEG as a non-invasive tool for detecting atypical executive-related brain activity. The identification of qEEG markers offers potential for early neurophysiological screening of SpLDs. These findings support the use of non-invasive qEEG-based assessments to complement behavioral evaluations, enabling individualized educational interventions and targeted neurocognitive training such as neurofeedback or EF enhancement programs.

Recommendation

The authors emphasize the importance of a multidisciplinary approach, including psychological assessment, academic achievement, and qEEG for early identification of learning difficulties. As SpLDs involve a wide range of learning challenges, reliance on single assessment modality is insufficient. The authors believe that a combination of objective and subjective measures is essential to ensure diagnostic accuracy and to capture the full complexity of each learner's profile. Clinically, qEEG-based assessments could guide individualized educational planning and guide interventions such as neurofeedback or cognitive training programs aimed to strengthen the EFs. Thus, future studies with larger samples and typically developing control groups are necessary to confirm these findings.

Conclusion

In summary, the present preliminary study suggests that children with suspected SpLDs show relatively elevated absolute low-frequency power at delta, theta, and lower-alpha, compared with higher-frequency activity at upper-alpha, beta, and gamma, during executive tasks, with the strongest effects observed in prefrontal and frontal regions. The authors also observed marked hemispheric asymmetries, characterized by reduced left-hemispheric dominance, which may indicate atypical lateralization of executive processing in this group. Taken together, these EEG patterns can be viewed as candidate neurophysiological correlations that may be associated with executive functioning difficulties in children with SpLDs, rather than as established diagnostic indicators.

What is already known about this topic?

SpLDs affect approximately 5% to 15% of school-aged children globally and are characterized by persistent difficulties in reading, writing, or mathematics due to atypical brain development and executive dysfunction. Children with SpLDs often show frontal cortex abnormalities and altered EEG patterns, particularly elevated frontal theta and atypical alpha rhythms, which reflect delayed cortical maturation and inefficient cognitive control. However, previous EEG studies have focused primarily on resting-state conditions rather than task-related neural activation. It leaves a limited understanding of how executive control processes are represented at the neurophysiological level during active cognitive engagement.

What does this study add?

This preliminary study presents novel findings on localized EEG activation during executive tasks.

Using eight electrode sites and nine frequency bands, from 0.5 to 45 Hz, significant hemispheric and regional differences were observed. Specifically, upper-theta power was significantly higher in the right hemisphere ($z=-2.19$, $p=0.02$), and lower-alpha power also demonstrated right-dominant activation ($z=-1.93$, $p=0.05$). In regional analyses, the frontal region showed markedly higher lower-delta ($p=0.02$) and upper-delta ($p<0.001$) power than the central region. These data reveal atypical right-hemispheric dominance and elevated low-frequency activity in prefrontal regions (Fpz, Fz, F3, F4). It indicates suboptimal neural resource allocation and delayed cortical maturation during executive processing.

Acknowledgement

The present research was funded by the Health Systems Research Institute (HSRI), Ministry of Public Health, Thailand.

Authors contribution

NR: ideas, design experiment, research practice, and manuscript writing, CN: data storage and processing, VS: criticism of results, VC: data collection, TK: data collection, SK: data collection, AP: ideas and hypotheses, KL: participation in manuscript writing, KD: data storage and processing, RRS: participation in manuscript writing, and KP: data analysis & processing, criticism of results, and manuscript writing.

Conflicts of interest

The authors declare no conflict of interest.

References

1. Karande S, Kulkarni M. Poor school performance. Indian J Pediatr 2005;72:961-7.
2. Maehler C, Schuchardt K. Working memory in children with specific learning disorders and/or attention deficits. Learn Individ Differ 2016;49:341-7.
3. Lyon GR, Shaywitz SE, Shaywitz BA. A definition of dyslexia. Ann Dyslexia 2003;53:1-14.
4. Hynd GW, Marshall R, Gonzalez J. Learning disabilities and presumed central nervous system dysfunction. Learn Disabil Q 1991;14:283-96.
5. Scaria LM, Bhaskaran D, George B. Prevalence of Specific Learning Disorders (SLD) among children in India: A systematic review and meta-analysis. Indian J Psychol Med 2023;45:213-9.
6. American Psychiatric Association. Neuro-developmental disorders. In: Desk reference to the diagnostic criteria from DSM-5. Washington, DC: APA Publishing; 2013.
7. Chan DW, Ho CSh, Tsang Sm, Lee Sh, Chung KKH. Prevalence, gender ratio and gender differences in reading-related cognitive abilities among Chinese children with dyslexia in Hong Kong. Educ Stud 2007;33:249-65.
8. Jamsai P, Boonnak S, Ruangcham C. Studying the current situation of screening, providing assistance, and monitoring elementary school students with learning disabilities in Thailand. Humanities Arts and Social Sciences Studies 2024;24:689-97.
9. Suvarnakich K, Rohitsuk W, Phattharayuttawat S, Ariyanuchitkul S, Patoommas P. Academic problems in primary schools in Bangkok. J Psychiatr Assoc Thailand 1999;44:55-64.
10. Booth JN, Boyle JM, Kelly SW. Do tasks make a difference? Accounting for heterogeneity of performance of children with reading difficulties on tasks of executive function: findings from a meta-analysis. Br J Dev Psychol 2010;28:133-76.
11. Henry LA, Messer DJ, Nash G. Executive functioning in children with specific language impairment. J Child Psychol Psychiatry 2012;53:37-45.
12. Moll K, Göbel SM, Gooch D, Landerl K, Snowling MJ. Cognitive risk factors for specific learning disorder: Processing speed, temporal processing, and working memory. J Learn Disabil 2016;49:272-81.
13. Ebert KD, Kohnert K. Sustained attention in children with primary language impairment: a meta-analysis. J Speech Lang Hear Res 2011;54:1372-84.
14. Sharfi K, Rosenblum S. Executive functions, time organization and quality of life among adults with learning disabilities. PLoS One 2016;11:e0166939.
15. Stuss DT, Alexander MP. Is there a dysexecutive syndrome? Philos Trans R Soc Lond B Biol Sci 2007;362:901-15.
16. Alvarez JA, Emory E. Executive function and the frontal lobes: a meta-analytic review. Neuropsychol Rev 2006;16:17-42.
17. Krause B, Cohen Kadosh R. Can transcranial electrical stimulation improve learning difficulties in atypical brain development? A future possibility for cognitive training. Dev Cogn Neurosci 2013;6:176-94.
18. Chan RC, Shum D, Toulopoulou T, Chen EY. Assessment of executive functions: review of instruments and identification of critical issues. Arch Clin Neuropsychol 2008;23:201-16.
19. El Wafa HEA, Ghobashy SAEL, Hamza AM. A comparative study of executive functions among children with attention deficit and hyperactivity disorder and those with learning disabilities. Middle East Current Psychiatry 2020;27:64. doi: https://doi.org/10.1186/s43045-020-00071-8.
20. Mousavi ST, Jamali G, Raji P. The relationship between executive function and activities of daily living in children with specific learning disorders. Appl Neuropsychol Child 2025;14:100-6.
21. Hamedi N, García-Salinas JS, Berry BM, Worrell GA, Kucewicz MT. Anterior prefrontal EEG theta activities

indicate memory and executive functions in patients with epilepsy. Epilepsia 2025;66:1274-87.
22. Tan E, Troller-Renfree SV, Morales S, Buzzell GA, McSweeney M, Antúnez M, et al. Theta activity and cognitive functioning: Integrating evidence from resting-state and task-related developmental electroencephalography (EEG) research. Dev Cogn Neurosci 2024;67:101404. doi: 10.1016/j.dcn.2024.101404.
23. Wang RWY, Chen YC, Liu IN, Chuang SW. Temporal and spectral EEG dynamics can be indicators of stealth placement. Sci Rep 2018;8:9117. doi: 10.1038/s41598-018-27294-4.
24. Ji L, Zhao Q, Gu H, Chen Y, Zhao J, Jiang X, et al. Effect of executive function on event-based prospective memory for different forms of learning disabilities. Front Psychol 2021;12:528883. doi: 10.3389/fpsyg.2021.528883.
25. Papagiannopoulou EA, Lagopoulos J. Resting state EEG hemispheric power asymmetry in children with dyslexia. Front Pediatr 2016;4:11. doi: 10.3389/fped.2016.00011.
26. Bosch-Bayard J, Galán-García L, Fernandez T, Lirio RB, Bringas-Vega ML, Roca-Stappung M, et al. Stable sparse classifiers identify qEEG signatures that predict learning disabilities (NOS) severity. Front Neurosci 2017;11:749. doi: 10.3389/fnins.2017.00749.
27. Benwell CSY, Davila-Pérez P, Fried PJ, Jones RN, Travison TG, Santarnecchi E, et al. EEG spectral power abnormalities and their relationship with cognitive dysfunction in patients with Alzheimer's disease and type 2 diabetes. Neurobiol Aging 2020;85:83-95.
28. Isquith PK, Roth RM, Gioia GA. Assessment of executive functioning using tasks of executive control. In: Goldstein S, Naglieri JA, editors. Handbook of Executive functioning [Internet]. New York, NY: Springer; 2014 [cited 2025 Aug 20]. p. 333-57. Available from: https://link.springer.com/10.1007/978-1-4614-8106-5_19.
29. Ritter N, Kilinc E, Navruz B, Bae Y. Test Review: Brown L, Sherbenou RJ, Johnsen SK. Test of Nonverbal Intelligence-4 (TONI-4). Austin, TX: PRO-ED; 2010. J Psychoeduc Assess 2011 Oct;29(5):484–8.
30. Mungkhetklang C, Crewther SG, Bavin EL, Goharpey N, Parsons C. Comparison of measures of ability in adolescents with intellectual disability. Front Psychol 2016;7:683. doi: 10.3389/fpsyg.2016.00683.
31. Pieper K, Spang RP, Prietz P, Möller S, Paajanen E, Vaalgamaa M, et al. Working with environmental noise and noise-cancelation: A workload assessment with EEG and subjective measures. Front Neurosci 2021;15:771533. doi: 10.3389/fnins.2021.771533.
32. Elshenaway A, Guirguis S. Adaptive thresholds of EEG brain signals for IoT devices authentication. IEEE Access 2021;9:100294-307.
33. Tabiee M, Azhdarloo A, Azhdarloo M. Comparing executive functions in children with attention deficit hyperactivity disorder with or without reading disability: A resting-state EEG study. Brain Behav 2023;13:e2951.
34. Cainelli E, Vedovelli L, Carretti B, Bisiacchi P. EEG correlates of developmental dyslexia: a systematic review. Ann Dyslexia 2023;73:184-213.
35. Guhan Seshadri NP, Agrawal S, Kumar Singh B, Geethanjali B, Mahesh V, Pachori RB. EEG based classification of children with learning disabilities using shallow and deep neural network. Biomed Signal Process Control 2023;82:104553. doi: https://doi.org/10.1016/j.bspc.2022.104553.
36. Cavalli E, Chanoine V, Tan Y, Anton JL, Giordano BL, Pegado F, et al. Atypical hemispheric re-organization of the reading network in high-functioning adults with dyslexia: Evidence from representational similarity analysis. Imaging Neurosci (Camb) 2024;2:imag-2-00070. doi: 10.1162/imag_a_00070.
37. Martínez-Briones BJ, Fernández T, Silva-Pereyra J. Electroencephalographic power spectrum patterns related to the intelligence of children with learning disorders. PeerJ 2025;13:e19138.
38. Cárdenas SY, Silva-Pereyra J, Prieto-Corona B, Castro-Chavira SA, Fernández T. Arithmetic processing in children with dyscalculia: an event-related potential study. PeerJ 2021;9:e10489.
39. Roca-Stappung M, Fernández T, Bosch-Bayard J, Harmony T, Ricardo-Garcell J. Electroencephalographic characterization of subgroups of children with learning disorders. PLoS One 2017;12:e0179556.
40. Fletcher JM, Miciak J. Assessment of specific learning disabilities and intellectual disabilities. Assessment 2024;31:53-74.
41. Fernández T, Herrera W, Harmony T, Díaz-Comas L, Santiago E, Sánchez L, et al. EEG and behavioral changes following neurofeedback treatment in learning disabled children. Clin Electroencephalogr 2003;34:145-52.
42. Knyazev GG. EEG delta oscillations as a correlate of basic homeostatic and motivational processes. Neurosci Biobehav Rev 2012;36:677-95.
43. Neuner I, Arrubla J, Werner CJ, Hitz K, Boers F, Kawohl W, et al. The default mode network and EEG regional spectral power: a simultaneous fMRI-EEG study. PLoS One 2014;9:e88214.
44. Gevins A, Smith ME. Neurophysiological measures of working memory and individual differences in cognitive ability and cognitive style. Cereb Cortex 2000;10:829-39.
45. Jensen O, Tesche CD. Frontal theta activity in humans increases with memory load in a working memory task. Eur J Neurosci 2002;15:1395-9.
46. Raghavachari S, Lisman JE, Tully M, Madsen JR, Bromfield EB, Kahana MJ. Theta oscillations in human cortex during a working-memory task: evidence for local generators. J Neurophysiol

2006;95:1630-8.
47. Klimesch W. α-band oscillations, attention, and controlled access to stored information. Trends Cogn Sci 2012;16:606-17.
48. Somsen RJ, van't Klooster BJ, van der Molen MW, van Leeuwen HM, Licht R. Growth spurts in brain maturation during middle childhood as indexed by EEG power spectra. Biol Psychol 1997;44:187-209.
49. Braver TS, Cohen JD, Nystrom LE, Jonides J, Smith EE, Noll DC. A parametric study of prefrontal cortex involvement in human working memory. Neuroimage 1997;5:49-62.
50. Casey BJ, Tottenham N, Liston C, Durston S. Imaging the developing brain: what have we learned about cognitive development? Trends Cogn Sci 2005;9:104-10.
51. Moriguchi Y, Hiraki K. Longitudinal development of prefrontal function during early childhood. Dev Cogn Neurosci 2011;1:153-62.
52. Thomason ME, Race E, Burrows B, Whitfield-Gabrieli S, Glover GH, Gabrieli JD. Development of spatial and verbal working memory capacity in the human brain. J Cogn Neurosci 2009;21:316-32.
53. Reuter-Lorenz PA, Jonides J, Smith EE, Hartley A, Miller A, Marshuetz C, et al. Age differences in the frontal lateralization of verbal and spatial working memory revealed by PET. J Cogn Neurosci 2000;12:174-87.
54. Simmonds DJ, Pekar JJ, Mostofsky SH. Meta-analysis of Go/No-go tasks demonstrating that fMRI activation associated with response inhibition is task-dependent. Neuropsychologia 2008;46:224-32.
55. Xu M, Yang J, Siok WT, Tan LH. Atypical lateralization of phonological working memory in developmental dyslexia. J Neurolinguistics 2015;33:67-77.
56. Nee DE, Wager TD, Jonides J. Interference resolution: insights from a meta-analysis of neuroimaging tasks. Cogn Affect Behav Neurosci 2007;7:1-17.
57. Gable PA, Mechin NC, Hicks JA, Adams DL. Supervisory control system and frontal asymmetry: neurophysiological traits of emotion-based impulsivity. Soc Cogn Affect Neurosci 2015;10:1310-5.