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Clinical and sleep profile of children with specific learning disorders, attention deficit hyperactivity disorder, or both: a retrospective multicenter study

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ABSTRACT

Background: Sleep disturbances are frequently reported in children with neurodevelopmental disorders (NDD), particularly attention-deficit/hyperactivity disorder (ADHD). However, sleep in children with specific learning disorders (SLD) remains poorly characterized. This study aimed to compare clinical and polysomnographic (PSG) sleep profiles of children with SLD, ADHD, or comorbid SLD-ADHD presenting with sleep complaints.
Methods: In this retrospective multicenter study, 146 children aged 6–18 years underwent PSG across three sleep centers and were classified into three groups: SLD (n = 60), SLD–ADHD (n = 50), and ADHD (n = 36). Clinical data, sleep complaints, the French Sleepiness Scale adapted for children (FSSA), and PSG parameters were analyzed using multinomial regression and mixed models.
Results: Overall, 38.0% of children reported daytime sleepiness and 39% reported insomnia complaints. FSSA scores were significantly higher in the SLD and SLD–ADHD groups than in the ADHD group (p = 0.01), while insomnia complaints was more frequent in the ADHD group (58.3% vs 26.7%, p = 0.009). On PSG, 25–30% had mild obstructive sleep apnea. The SLD and SLD–ADHD groups showed greater NREM sleep fragmentation, higher respiratory arousal indices, and a higher prevalence of pathological periodic limb movements compared to ADHD. In multivariate analyses, SLD group membership and anxiety were independently associated with higher FSSA scores.
Conclusions: In this sleep-clinic sample, children with SLD exhibited greater subjective daytime sleepiness and NREM sleep fragmentation despite fewer insomnia complaints than those with ADHD, underscoring the importance of systematic sleep assessment in this population.

1. Introduction

Neurodevelopmental disorders (NDD) are a group of conditions characterized by early-onset impairments in cognitive, linguistic, motor, or social functioning, as defined in the DSM-5 [1]. Over the past two decades, their reported prevalence has increased, likely reflecting

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improved diagnostic practices, greater awareness, and evolving classification frameworks [2,3]. Specific learning disorders (SLD) include persistent difficulties in reading, written expression, and mathematics; developmental language disorder (DLD) and developmental coordination disorder (DCD) are classified as distinct NDD; these disorders however, frequently co-occur. SLD represent a substantial subgroup, affecting approximately 3–10% of children in European countries [4], and are frequently encountered in both educational and clinical settings. Notably, SLD commonly co-occur with attention-deficit/hyperactivity disorder (ADHD), with reported comorbidity rates ranging from 33% to 70%, potentially exacerbating both attentional and learning difficulties [5].

Sleep plays a critical role in brain development and in cognitive, emotional, and behavioral functioning. Sleep disturbances are associated with impairments in learning, memory, attention, and emotional regulation in both typically developing (TD) children and those with NDD [6,7]. While approximately 20% of TD children experience sleep disturbances, prevalence rates may reach up to 86% in children with NDD [6,8]. In ADHD, sleep disorders are reported in 55% to 74% of cases, most commonly involving difficulties initiating or maintaining sleep, restless legs syndrome, and delayed sleep phase [9]. Although objective measures tend to show less pronounced alterations than subjective reports, increased sleep fragmentation and reduced sleep efficiency have consistently been observed in children with ADHD compared to TD peers [9].

Sleep in children with SLD remains relatively understudied. Two studies using the Sleep Disturbance Scale for Children (SDSC) [10] reported higher scores for insomnia, sleep-disordered breathing, and arousal disorders in children with dyslexia compared to controls [11]. Another study found that children with comorbid SLD and ADHD exhibited more severe sleep disturbances than those with either condition alone [12]. Objective sleep findings are inconsistent: some studies report alterations in sleep architecture, including changes in non-rapid eye movement (NREM) and rapid eye movement (REM) sleep, as well as associations between sleep spindle activity and reading performance [13,14]. However, more recent data have shown limited differences, with only increased total sleep time (TST) observed in children with dyslexia following a phonologic memory test [15].

Overall, subjective data suggest that sleep disturbances are more frequent in children with SLD and ADHD than in TD peers, particularly in cases of comorbidity [12]. However, objective sleep data remain scarce, and direct comparisons between children with SLD, ADHD, and comorbid SLD–ADHD are lacking. The aim of the present retrospective study was therefore to characterize the clinical and sleep profiles of children with SLD, ADHD, or comorbid SLD–ADHD presenting with sleep complaints.

2. Methods

2.1. Study design and participants

This retrospective multicenter study was conducted in children with SLD, ADHD, or SLD-ADHD, who were recruited between 2015 and 2024 from three sleep laboratories: the *Hôpital Femme Mère Enfant* (Bron, France; center 1), the *Clinique du Drapeau* (Dijon, France; center 2), and the *Cabinet de l'Aire Familiale* (Bordeaux, France; center 3), which are respectively managed by a pediatric neurologist, an otolaryngologist, and a child psychiatrist. These centers were chosen because they represent the different clinical settings in which children with sleep complaints and NDD are commonly referred. To be included, children had to be aged between 6 and 18 years old, have a confirmed diagnosis of SLD and/or ADHD made by specialists in neurodevelopmental disorders (pediatric neurologists, child psychiatrists, neuropsychologists, speech therapists …) according to DSM-5 criteria, have sleep complaints, and have undergone a sleep assessment using polysomnography (PSG). The diagnosis of SLD with ADHD or not, included the following subtypes: developmental dyslexia, spelling disorder, dysgraphia, and developmental dyscalculia. Children with diagnosed intellectual disability, autism spectrum disorder, epilepsy, or primary hypersomnia (narcolepsy, Kleine-Levin syndrome) were excluded.

2.2. Ethical considerations

Patients and caregivers were informed about the use of their clinical data for research purposes, and those who were not opposed to it were included. The study was approved by the institutional ethics committee of the *Hospices Civils de Lyon* (n° 24-5112, 08/21/2024) with personal data processing compliance commitment in accordance with the national data protection agency (*Comission Nationale de l'Informatique et des Libertés*, CNIL).

2.3. Procedure

Each participant underwent a sleep consultation with a sleep specialist, accompanied by their legal guardian(s). During the consultation, data regarding their medical history were collected using closed-ended questions (yes/no): prematurity, anxiety or depression, emotional dysregulation (difficulties in regulating their emotions, which may manifest as intense and unpredictable emotional reactions), Tic disorders (Tic), obsessive-compulsive disorder (OCD), oppositional defiant disorder (ODD), DLD, DCD, asthma, allergies, adenoid surgery, as well as the use of treatments that can impact sleep (stimulants, anxiolytics, antihistamines, melatonin), when the information was available. Stimulant treatment was stopped 48h before PSG recording in center 1 and 3. The sleep complaint was then investigated. Insomnia complaints (characterized by persistent difficulty in initiating or maintaining sleep, or experiencing early morning awakenings) as wells as sleepiness complaints were searched for by closed-ended questions. The Epworth Sleepiness Scale, adapted for children and validated in French (FSSA) was also completed to evaluate sleepiness complaints [16]. This 8-item scale assesses sleepiness in patients' daily lives; scores above 7 represent excessive daytime sleepiness and are considered pathological [17]. This questionnaire has been validated in a pediatric population comprising 269 non-clinical subjects and 115 clinical subjects admitted for overnight polysomnography and Multiple Sleep Latency Test (MSLT) because of suspected hypersomnia (85 patients with narcolepsy and 30 with other sleep disorders).Distribution of scores for the nonclinical group and the clinical group differed significantly; the FSSA8 had very good screening validity in sleep disorders. A night PSG was then performed and some patients underwent laboratory measurements of blood ferritin levels.

2.4. Polysomnography

Following the sleep consultation, each child underwent a night PSG, according to the clinical practice of each center, using internationally validated systems that are routinely used in both clinical practice and sleep research (Table 1). The EEG, electromyogram (EMG), and electrooculogram (EOG) signal was sampled at 256 Hz; impedances were maintained below 10 kΩ. A centralized reading following the American Academy of Sleep Medicine (AASM) [18,19] criteria was performed by the same sleep expert (AG) to ensure consistent scoring across the three centers. The BrainRT software (OSG, Kontich, Belgium) was used for patients from center 1 and 3 while the Noxturnal software (Nox Medical ehf, Reykjavik, Iceland) was used for patients from centers 2.

The following variables were calculated: sleep latency (min), REM latency (min), TST (min), sleep period (min), duration and percentage of sleep stages (N1, N2, N3, REM) and wake periods (wake after sleep onset [WASO], min), sleep efficiency (TST/time spent in bed; %), longest wake during sleep [min], arousal index (AI) over TST and according to sleep stages (NREM and REM), respiratory indices (apnea-hypopnea index [AHI] whether obstructive [OAHI] or central [CAHI], respiratory

Table 1
Polysomnography recording methods.

	Center 1 (Lyon)	Center 2 (Dijon)	Center 3 (Bordeaux)
Setting	Laboratory recording PSG	Home recording PSG	Laboratory recording PSG or Home recording PSG
Sleep recorder	Morpheus (Micromed, Mogliano Veneto, Italy)	Nox A1 (ResMed ehf, Reykjavik, Iceland)	
EEG channels	10 EEG channels Fp1, Fp2, C3, C4, T3, T4, O1, O2, M1, M2	8 EEG channels F3, F4, C3, C4, O1, O2, M1, M2	4 EEG Channels F3, C3, O1, M2
Other recorded signals	Right and left EOG, chin EMG, bilateral anterior tibialis EMG, thoracic and abdominal efforts, nasal airflow, oronasal airflow, pulse oximetry (SpO_2), ECG		

PSG = polysomnography; EEG = electroencephalogram; EMG = electromyogram; EOG = electrooculogram; ECG = electrocardiogram.

AI, respiratory effort-related Arousal [RERA]), and periodic limb movement index (PLMI) not associated with respiratory events, as recommended by the AASM.

The following pathological standards defined by the AASM [17,18] were used: obstructive sleep apnea (OSA) was defined as mild if OAHI ≥1.5/h, moderate if OAHI ≥5/h, and severe if OAHI ≥10/h; upper airway resistance syndrome (UARS) was defined as RERA ≥1/h and OAHI <1.5/h; high periodic limb movement (HPLM) was defined as PLMI ≥5/h; sleep fragmentation was defined as AI >15/h.

2.5. Data collection

Overall, the following data were collected from the medical files: age, sex, weight, height, body mass index (BMI, defined as weight/height squared), BMI Z-score (overweight and obesity were defined as BMI Z-score ≥1.6 and >2, respectively), SLD type, blood ferritin levels, and information regarding comorbidities and treatments, which were collected during the consultation. The sleep and respiratory variables were obtained from the PSG reports.

2.6. Statistical analyses

Descriptive analyses were conducted for the three different groups: SLD, ADHD, and SLD-ADHD. Continuous variables were described using median (range) and categorical variables using number and percents. Group comparisons were carried out using non-parametric tests, after checking the normality of the distribution by Shapiro test (Kruskal-Wallis with Benjamini-Hochberg correction, Chi-square test) followed by pairwise Wilcoxon tests with Benjamini-Hochberg correction to account for multiple testing. Only a descriptive analysis was performed for the ferritin data due to the small sample size (mean, percentage of patients with values < 50 μg/L).

Three multivariate analyses were performed using a multinomial logistic regression model to study the association between daytime sleepiness (FSSA score) and clinical groups (ADHD, SLD, and SLD-ADHD). The ADHD group was used as the reference category. The models were adjusted for confounding factors identified based on literature and directed acyclic graphs [20]. The first model was adjusted on center and sex. The second model was adjusted on anxiety and emotional dysregulation as well as on a combined comorbidity score that was calculated based on medical history. The last model was adjusted on treatment: stimulant treatment (y/n), melatonin (y/n), anxiolytic (y/n). Multicollinearity was verified (Variance Inflation Factor <3 for all variables), and results were reported as odds ratios (OR) with their 95% confidence intervals (95% CI) and p-values.

Then, exploratory multivariate regression analyses were performed to identify factors explaining daytime sleepiness (FSSA) according to the clinical group. Explored variables were prematurity, age, allergies, anxiety, BMI Z-score, insomnia symptoms, HPLM, OAHI, stimulant treatment, AI in NREM, and center. Models were compared using the Akaike Information Criterion (AIC)/Bayesian Information Criterion (BIC). The final model was selected based on parsimony and with interactions between the explored variables and the clinical group that were searched for, with the main explanatory factors being group, anxiety and center. Finally, analyses were replicated using mixed models with center as random intercept in order to take into account the hierarchical structure of the data (participants recruited from three centers). Model estimates are reported as regression coefficients (β) with their standard errors (β ± SE) and p-values. The significance threshold was set at $p < 0.05$. All analyses were performed using RStudio software version 4.3.2 (nnet, car, dplyr, and ggplot2 packages).

3. Results

3.1. Patients' characteristics

A total of 146 children were included in the study (36.3% girls; median age = 10.7 years [range: 6–18]), divided into three groups: SLD (41.1%), SLD-ADHD (34.2%), and ADHD (24.7%); Fig. 1). Children with ADHD were slightly younger than those with SLD and SLD-ADHD (10.1 years vs. 10.8 and 11 years, respectively, p = 0.07). The proportion of overweight or obese children was 27.8% in ADHD, 28.3% in SLD, and 34% in SLD-ADHD. Among children with SLD, 15% had associated DCD and 8.3% had associated DLD. In contrast, among those with both SLD and ADHD, the prevalence of associated DCD and DLD was higher, at 38% and 12%, respectively. In the SLD-ADHD group, a higher proportion of children had multi-SLD and DCD compared to those from the SLD group (64% vs 45%, p 0.056 and 38% vs 15%, p = 0.008, respectively). In terms of treatments, a significantly higher proportion of children with SLD-ADHD reported the use of psychostimulants (methylphenidate) compared to the SLD group (44% vs 6.7%, $p < 0.001$). In children with ADHD, the proportion of emotional dysregulation and history of respiratory disorders was significantly higher than in children with SLD and SLD-ADHD (47.2% vs 11.7 and 20%, $p < 0.001$ and 38.9% vs 18.3 and 16%, p = 0.026, respectively; Table 2).

3.2. Subjective sleep and wake quality

In the total population of children, 38.4% had a sleepiness complaint, 39% had insomnia complaints, and 13.7% had both types of complaints. Children in the ADHD group reported significantly more insomnia complaints than those in the SLD group (58.3% vs 26.7%, p = 0.009). The median score on the FSSA was significantly higher in the SLD group compared to the ADHD group (10 vs 4, p = 0.01). The proportion of children with a pathological score was significantly higher in the SLD versus ADHD group (50% vs 19.4%, p = 0.01; Table 2).

3.3. Polysomnography characteristics

The analysis of sleep found no significant difference in TST, sleep stages, or sleep efficiency among the three groups (Table 3). Overall, 30.3%, 26%, and 25% of children with SLD, SLD-ADHD, and ADHD, respectively, had mild OSA. Sleep fragmentation with an AI greater than 15/h was present in 60%, 54%, and 38.9% of the SLD, SLD-ADHD, and ADHD groups; the AI in NREM was significantly higher in both the SLD (14.5 vs 10.8, p = 0.04) and SLD-ADHD (14.1 vs 10.8, p = 0.04) groups compared to the ADHD group. Respiratory AI was significantly higher in the SLD and SLD-ADHD groups than in the ADHD group (4 vs 2.4, p = 0.002 and 4 vs 2.4, p = 0.009, respectively). Among the 75 children with ferritin testing, 54/75 (72%) had ferritin levels <50 μg/ml; 26% of them had ADHD, 37% had SLD, and 37% had SLD-ADHD.

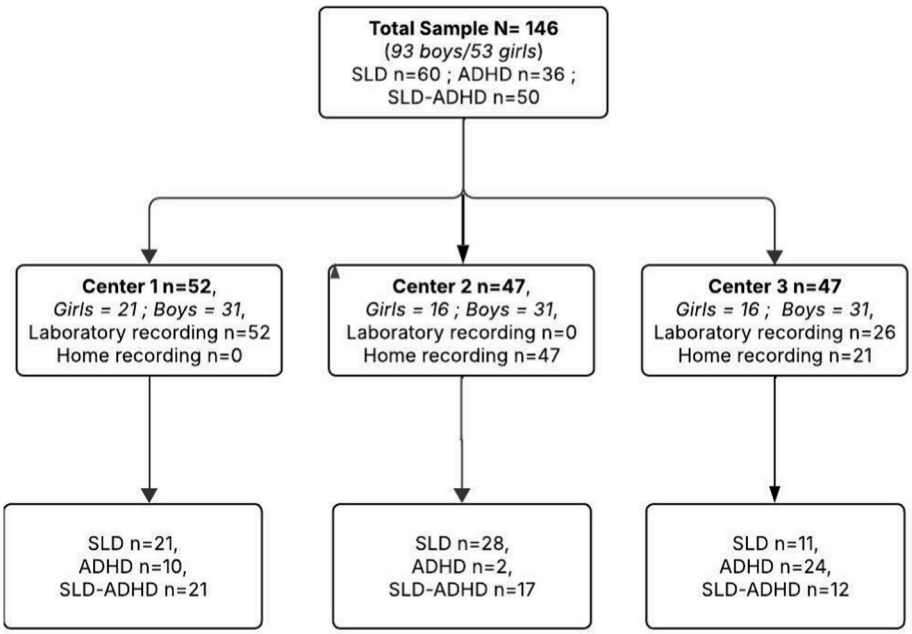

Fig. 1. Study flowchart.

Table 2
Patients' characteristics.

	SLD N = 60	SLD-ADHD N = 50	ADHD N = 36	*p-value*	Missing values
Demographics					
Age, years, median (range)	10.8 (6.9–17.2)	11 (6.2–18)	10.1 (7.1–16.2)	*0.07*	0
Sex, girls	23 (38.3)	19 (38)	11 (30.6)	0.71	0
BMI Z-score, median (range)	0.6 (−2.3–7.5)	0.3 (−1.2–9.3)	0 (-3–8.8)	*0.23*	0
Overweight or obesity	17 (28.3)	17 (34)	10 (27.8)	*0.76*	0
Treatments					
Psychostimulants	4 (6.7)	22 (44)	6 (16.7)	*< 0.001*[d]	0
Melatonin	2 (3.3)	6 (12)	4 (11.1)	*0.19*	0
Anxiolytics	2 (3.3)	3 (6)	0 (0%)	*0.32*	0
Antihistamines	16 (26.7)	7 (14)	7 (19.4)	*0.25*	0
Medical History					
Prematurity	1 (1.7)	5 (10)	2 (5.6)	*0.16*	0
Allergy	17 (28.3)	5 (10)	10 (27.8)	*0.04*[d]	0
Asthma	18 (30)	7 (14)	9 (25)	*0.13*	0
Respiratory disorders	11 (18.3)	8 (16)	14 (38.9)	*0.026*[e, f]	0
Adenotonsillectomy	11 (18.3)	13 (26)	10 (27.8)	*0.48*	0
Anxiety/Depression	13 (21.7)	12 (24)	10 (27.8)	*0.79*	0
Emotional dysregulation	7 (11.7)	10 (20)	17 (47.2)	*< 0.001* [e f]	0
Tic/OCD/ODD	3 (5)	5 (10)	2 (5.6)	*0. 5*	0
SLD subtype					
Multi-SLD	27 (45)	32 (64)		*0.056*	0
Developmental Dyslexia	48 (80.0%)	39 (78.0%)		0.81	0
Spelling Disorder	27 (45.0%)	23 (46.0%)		1	0
Developmental Coordination Disorder	9 (15.0%)	19 (38.0%)		0.008	0
Dysgraphia	17 (28.3%)	21 (42.0%)		0.16	0
Developmental Dyscalculia	12 (20.0%)	8 (16.0%)		0.62	0
Developmental Language Disorder	5 (8.3%)	6 (12.0%)		0.54	0
Sleep complaint					
Insomnia	16 (26.7)	20 (40)	21 (58.3)	*0.009* [e]	0
Sleepiness complaint	26 (43.3)	19 (38)	11 (30.6)	*0.45*	0
FSSA, median (range)	10 (0–22)	7 (0–21)	4 (0–24)	*0.017*[b]	25
	N=49	*N=43*	*N=29*		
FSSA >10	25 (41.7)	15 (30)	5 (13.9)	*0.008* [e]	25
FSSA >7	30 (50)	19 (38)	7 (19.4)	*0.011* [e]	25

Variables are presented as n (%) unless otherwise specified.
Overweight = Z-score ≥1.6; Obesity = Z-score >2.
Respiratory disorders: OSA = OAHI >1.5; UARS = Respiratory AI > 1/h and OAHI <1.5/h.
Significant results using pairwise Wilcoxon test: a = SLD - SLD/ADHD; b = SLD - ADHD; c = SLD/ADHD – ADHD.
Significant results using pairwise Chi^2 test: d = SLD - SLD/ADHD; e = SLD - ADHD; f = SLD/ADHD – ADHD.
Tic = Tic disorder; OCD = Obsessive-Compulsive Disorder; ODD = Oppositional Defiant Disorder; OSA = Obstructive Sleep Apnea; OAHI = Obstructive Apnea Hypopnea Index; UARS = Upper Airway Resistance Syndrome; Respiratory AI = Respiratory Arousal Index; BMI = Body Mass Index.

Table 3
Polysomnography results.

	SLD N = 60	SLD-ADHD N = 50	ADHD N = 36	*p-value*	Missing Value
Sleep architecture					
NREM 1	7.1 (3.4–27.9)	7.9 (1.8–18.4)	6.5 (3.7–17.7)	*0.14*	0
NREM 2	40.6 (9.8–59.3)	43.3 (14.3–62.1)	43.4 (26.7–62)	*0.17*	0
NREM 3	28.9 (12.9–56.5)	26.9 (9.7–64.5)	26.9 (14.4–37.7)	*0.32*	0
NREM 3 > 25%	40 (66.7)	32 (64)	25 (69.4)	*0.86*	
REM	22.5 (15.1–33.3)	22.4 (11.5–34)	22.8 (14.6–29.2)	*0.94*	0
Wake after sleep onset (min)	44.8 (8.5–275)	49.8 (15.5–164.5)	43 (9.5–250)	*0.37*	0
Longest wake period (min)	8.8 (1-148)	12.5 (1.5-92)	8.25 (1.5-218.5)	0.33	0
Efficiency	91 (55.8–98.4)	90.6 (72.6–97.2)	91.9 (58.2–98.3)	*0.41*	0
% efficiency <90%, n (%)	27 (45)	21 (42)	13 (36.1)	*0.69*	0
Sleep latency (min)	20 (1.5–118)	15 (2.1–62.2)	14.1 (0–119.3)	*0.62*	4
% latency <30 min, n (%)	15 (25)	15 (30)	6 (16.7)	*0.37*	4
REM latency (min)	112.2 (43.5–368.5)	132 (47.5–241.5)	137.5 (52–235.5)	*0.10*	4
TST (min)	502.2 (332–607)	495.5 (309–682.5)	494 (347–589)	*0.92*	0
Sleep period (min)	553.2 (409–718.5)	563.5 (332.5–723.5)	542 (328.5–659)	*0.73*	0
Respiratory					
OAHI (/h)	1.4 (0–68.6)	1.2 (0–49.2)	0.9 (0–12.2)	*0.13*	0
Mild OSA, n (%)	20 (33.3)	13 (26)	9 (25)	*0.59*	0
Moderate OSA, n (%)	5 (8.3)	5 (10)	1 (2.8)	*0.4*	0
				0.3	
Severe OSA, n (%)	3 (5)	2 (4)	1 (2.8)	*86*	0
UARS, n (%)	31 (51.7)	26 (52)	21 (58.3)	*0.79*	0
Respiratory arousal index (/h)	4 (0.9–52.3)	4 (0–32.6)	2.4 (0–11.5)	*0.002* [b, c]	0
Fragmentation					
Arousal index (/h)	15.9 (7.1–60.5)	16.2 (6.6–48.9)	13.8 (6.6–27.6)	*0.1*	0
Arousal index in NREM (/h)	14.5 (6.2–61.3)	14.1 (6.7–47.1)	10.8 (6.1–22.3)	*0.046* [b, c]	1
Arousal index in REM (/h)	23.2 (7.5–64.2)	22.9 (4.2–56.6)	21.6 (6.4–46.6)	*0.76*	1
Sleep fragmentation (AI > 15/h), n (%)	36 (60)	27 (54)	14 (38.9)	*0.13*	0
PLMI (/h)	0.6 (0–12.4)	1.4 (0–36.5)	1.1 (0–9.6)	*0.069*	7
PLMS (PLMI ≥5/h), n (%)	6 (10)	9 (18)	1 (2.8)	*0.067*	7
PLMI >5/h with ferritin <50 μg/ml)	1/2 (50)	5/7 (83)	0 (0)		137
Laboratory measures					
Ferritin (μg/ml)	28.5 (4–124)	33 (6–212)	34.5 (14–121)		71
Ferritin <50 μg/ml	20/26 (76.9)	20/27 (74.1)	14 (63.6)		71

Variables are presented as median (range) unless otherwise specified.
OSA = OAHI >1.5; UARS = Respiratory Arousal Index >1/h and OAHI <1.5/h.
Significant results using pairwise Wilcoxon test: a = SLD - SLD/ADHD; b = SLD - ADHD; c = SLD/ADHD – ADHD.
Significant results using pairwise Chi^2 test: d = SLD - SLD/ADHD; e = SLD - ADHD; f = SLD/ADHD – ADHD.
Tic = Tic Disorders; OCD = Obsessive-Compulsive Disorder; ODD = Oppositional Defiant Disorder; OSA = Obstructive Sleep Apnea; OAHI = Obstructive Apnea Hypopnea Index; TST = Total Sleep Time; UARS = Upper Airway Resistance Syndrome; BMI = Body Mass Index; PLMI = Periodic Limb Movement Index; PLMS = Periodic Legs Movement Syndrome; AI = Arousal Index; Respiratory AI = Respiratory Arousal Index.

3.4. Factors associated with SLD or SLD-ADHD

In the first model, FSSA scores and clinical group were associated with increased odds of belonging to the SLD group compared with the ADHD group (OR = 1.12, 95% CI 1.01–1.24, p = 0.031), whereas no significant association was found for the SLD-ADHD group (OR = 1.06, 95% CI 0.95–1.17, p = 0.29). Center effect was significant for center 3 in the SLD group (OR = 0.23, 95% CI 0.07–0.83, p = 0.024), but not for center 2. Sex was not significantly associated with clinical group.

In the second model, FSSA remained significantly associated with higher odds of belonging to the SLD group (OR = 1.17, 95% CI 1.04–1.31, p = 0.008). In the SLD-ADHD group, a similar association did not reach statistical significance (OR = 1.11, 95% CI 0.99–1.25, p = 0.066). Among clinical variables, anxiety was associated with lower odds of belonging to the SLD group (OR = 0.43, 95% CI 0.10–1.87, p = 0.26), while a significant association was observed for emotional dysregulation in the SLD-ADHD group (OR = 0.06, 95% CI 0.01–0.54, p = 0.012).

The third model adjusted on treatments showed unstable estimates for anxiolytic and melatonin use, with very wide confidence intervals, suggesting sparse data and limited interpretability for these predictors. No significant association was observed for stimulant treatment. FSSA remained weakly associated with clinical group membership, while treatment variables should be interpreted with caution due to model instability. In these models, 120 children were included: 27 with ADHD, 49 with SLD, and 44 with SLD-ADHD. Collinearity diagnostics indicated low multicollinearity across predictors (all VIF <3.5).

3.5. Factors associated with daytime sleepiness

The exploratory analysis carried out to identify factors explaining daytime sleepiness (FSSA) according to the clinical group showed that the best model included anxiety and center as random effect. Children with SLD and SLD-ADHD had significantly higher FSSA scores than those with ADHD (β = 3.95±1.3 points, p = 0.01 and β = 5.1±1.5, p = 0.002, respectively). Interaction between anxiety and group significantly improved the model fit and showed that while anxiety and FSSA score was strongly positively associated with ADHD (β = 7.3±2.2, p = 0.001), children in the SLD and SLD-ADHD groups with anxiety showed lower FSSA scores than those with ADHD and anxiety (β = −4.1±3, p = 0.17 and β = −7.1±2.8, p = 0.01, respectively; Table 4 and Fig. 2). In addition, the random variance for the center (variance = 0.82±0.91), with residual variance (27.8±5.27), indicated moderate inter-center variability. The analyses including center as random intercept provided similar effects in magnitude and significance.

4. Discussion

Compared with the ADHD group, children with SLD, particularly those without comorbid ADHD, reported more complaints of daytime

Table 4
Linear mixed effect model evaluating factors explaining daytime sleepiness (FSSA).

Variable	Estimate	SE	df	t	p
Intercept	3.957	1.377	12.745	2.874	0.01326 *
Group SLD	5.115	1.578	54.448	3.243	0.002 **
Group SLD-ADHD	4.007	1.587	85.388	2.525	0.01342 *
Anxiety	7.332	2.241	113.989	3.273	0.0014 **
Group SLD × Anxiety	−4.193	3.045	113.989	−1.377	0.17111
Group SLD-ADHD × Anxiety	−7.142	2.892	113.050	−2.470	0.01500 *

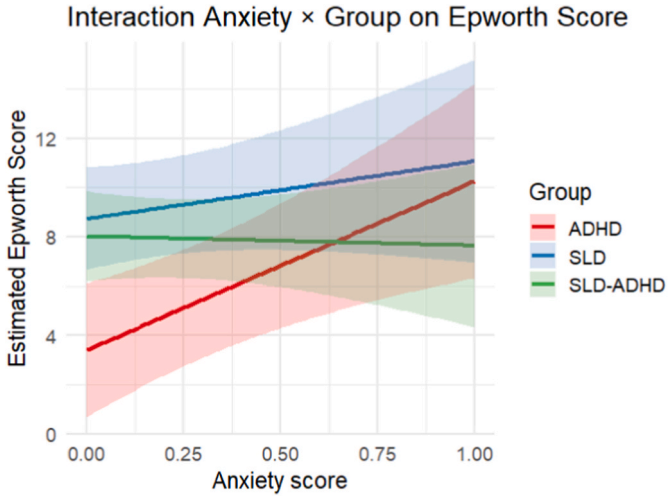

Fig. 2. Anxiety × Group interaction on FSSA score in the linear mixed model.

sleepiness and higher FSSA scores, without a corresponding increase in insomnia complaints, despite greater NREM sleep fragmentation and higher respiratory AI on PSG recordings. High rates of anxiety and obesity were observed across all three groups.

Age differed slightly between groups, with ADHD patients being younger. This difference likely reflects recruitment patterns, as many ADHD patients were enrolled at one center (center 3) where early ADHD diagnosis was systematically combined with sleep evaluation. The sex ratio was balanced across groups, with 30.6-38.3% of females in each group. The proportion of overweight or obese children was relatively high in all groups compared to the 21% population estimate among adolescents [21]. This observation is consistent with previous studies reporting increased obesity in children with NDD [22]. In SLD, academic stress, emotional vulnerability, and cognitive fatigue may contribute to dysregulated eating behaviors and reduced physical activity [23,24]. In ADHD, impulsivity and emotional dysregulation may promote disorganized eating patterns [25,26]. In both conditions, sleep disturbances may further contribute to metabolic dysregulation [27].

Marked anxiety was observed across the three groups, consistent with previous literature highlighting the high prevalence of anxiety disorders in children with NDD [28–31]. In ADHD, anxiety may partly reflect a neurobiological vulnerability involving emotional dysregulation and alterations in fronto-limbic circuits [32,33]. Consistent with this interpretation, the ADHD group herein showed a higher prevalence of emotional dysregulation, a finding frequently reported in this population and reflecting their affective vulnerability [34–36]. In contrast, anxiety in children with SLD appears more reactive, often related to repeated academic difficulties and reduced academic self-esteem [35,37]. Sleep disturbances frequently associated with dyslexia may further exacerbate these symptoms [36]. Overall, the levels of anxiety and depressive symptoms observed in the present cohort were higher than those reported in the general pediatric population, supporting the notion of increased emotional vulnerability in children with NDD [38].

Treatment characteristics also differed between groups. Children with ADHD were more frequently treated with psychostimulants, consistent with the predominance of hyperactivity–impulsivity symptoms in this population. The higher rate of adeno-tonsillectomy may also reflect the greater burden of respiratory or Ear Nose and Throat (ENT)-related conditions reported in children with ADHD [35]. In addition, allergic conditions were more frequently observed in children with ADHD and in those with isolated SLD, as previously reported [39,40]. These observations are consistent with studies suggesting possible links between allergic diseases and neurodevelopment, supporting the hypothesis of shared biological or developmental mechanisms.

Regarding sleep complaints, insomnia symptoms were significantly more frequent in children with ADHD, consistent with previous studies reporting frequent difficulties in sleep initiation and maintenance in this population [9]. These disturbances may reflect hyperarousal, circadian rhythm alterations, and the effects of stimulant treatments, as suggested by previously described sleep phenotypes in ADHD [41]. In contrast, although complaints of daytime sleepiness were similar in the three groups, higher scores on the FSSA scales were found in the SLD and SLD-ADHD groups compared with the ADHD group. In contrast, studies in children with dyslexia have reported higher sleep disturbance scores—particularly for insomnia complaints—on subjective sleep questionnaires compared with TD children [11,12]. A direct comparison with the present results is complicated by the difference in assessment methods. In prior studies, sleep disturbances were evaluated using standardized sleep questionnaires, whereas herein, insomnia was assessed based on clinical complaints rather than scale-derived scores. For daytime sleepiness however, FSSA scores in the present cohort were higher in children with SLD, particularly those without comorbid ADHD, while subjective complaints did not differ significantly between groups. This finding suggests that excessive daytime sleepiness in this population may be underreported clinically and better captured by standardized sleepiness scales. Another important difference with previous studies, which warrants caution when interpreting the results, relates to the fact that all the children in the present study were referred for sleep evaluation. This referral bias may have increased the overall prevalence of sleep complaints across all groups and modified the clinical presentation, with a higher baseline level of sleep-related concerns irrespective of diagnosis. One possible hypothesis is that children with SLD initially present with insomnia symptoms that may progressively evolve into excessive daytime sleepiness, resulting in referral to a specialized sleep center.

From an objective perspective, PSG did not reveal major differences in global sleep architecture between groups as they shared neurodevelopmental vulnerabilities. However, analyses of sleep microstructure showed that NREM sleep fragmentation and respiratory AI were significantly higher in the SLD and SLD-ADHD groups than in the ADHD group. These alterations might contribute to non-restorative sleep and increased daytime sleepiness in SLD patients, even in the absence of prominent insomnia complaints.

The present findings confirm a dissociation between subjective and objective sleep disturbances across neurodevelopmental profiles. In ADHD, subjective insomnia complaints were prominent despite relatively modest PSG alterations [42,43]. Such discrepancies have been widely reported in the literature and may reflect altered perception of sleep or dysregulation of arousal mechanisms [9,24]. Several models propose instability of cortical activation and altered vigilance regulation in ADHD. In the hypo-arousal model described by Hegerl and colleagues [44], behavioral hyperactivity may represent a compensatory mechanism aimed at maintaining wakefulness. Dysregulation of dopaminergic and noradrenergic pathways involved in vigilance regulation [45,46] may further contribute to an atypical alteration of sleep perception despite relatively preserved sleep architecture [47]. In contrast, the greater sleep fragmentation observed in children with SLD may contribute more directly to perceived daytime sleepiness. Respiratory

events were also more frequent in the SLD groups. Although sleep-disordered breathing are commonly associated with ADHD [48], earlier clinical recognition and treatment in this population may partly explain the lower residual respiratory indices observed in the ADHD group herein [9,35].

Beyond these classical sleep disorders, additional mechanisms may contribute to daytime sleepiness in SLD. Children with SLD reported greater daytime sleepiness despite relatively limited differences in macrostructural sleep parameters. One possible explanation may involve increased sleep fragmentation observed during NREM sleep. Even subtle micro-fragmentation could potentially interfere with the restorative functions of slow-wave sleep, which plays a central role in cognitive recovery, synaptic plasticity, and memory consolidation [49]. Another potential explanation relates to the increased cognitive load experienced during the day. Children with SLD often require sustained compensatory effort to perform academic tasks, leading to increased mental fatigue [30,31]. In this context, even modest sleep disturbances may have disproportionate effects on daytime alertness. However, several other factors known to influence daytime sleepiness in children and adolescents, including habitual sleep duration, sleep schedules, circadian preferences, screen exposure, mood symptoms, and academic or socioeconomic stressors, were not assessed in the present study. Importantly, anxiety and sleep-disordered breathing did not explain the differences in daytime sleepiness observed between groups in the adjusted analyses. This suggests that sleepiness in children with SLD may reflect a broader vulnerability of sleep-dependent cognitive processes rather than the presence of specific sleep disorders alone. Alterations in sleep microstructure, including slow-wave activity or sleep spindle dynamics, have previously been reported in dyslexic children and could contribute to non-restorative sleep in this population [14]. Multivariate analyses confirmed herein that higher FSSA scores were independently associated with belonging to the SLD and SLD-ADHD groups compared with ADHD and that anxiety influences sleepiness in children with ADHD more than in those with SLD.

The present study has several strengths, including its multicenter design, the use of full PSG scored using standardized procedures, and the integration of clinical, behavioral, and sleep parameters. However, several limitations must be acknowledged. The retrospective design resulted in some missing data, notably for the FSSA and ferritin levels, as well as the unavailability of certain parameters of interest, such as school reports, screen time, or sleep schedules. Limitations related to the multicenter design must also be taken into account, such as the variations in PSG procedures, although the centralized scoring helped reduce this variability. Moreover, the neurodevelopmental profile of children varied greatly according to the recruiting center; this clinical heterogeneity however, reflects real-world pediatric sleep clinic populations. In addition, the variability in treatments (stimulants, melatonin, or anxiolytics) may have introduced a source of bias: although the majority of children stopped their medication 48 h before the PSG recording (primarily stimulants and melatonin), an effect of these treatments on sleepiness and/or insomnia complaints remains likely. Since insomnia complaints were not evaluated using standardized scales, the findings should be interpreted as reflecting parent- and patient-reported insomnia complaints rather than formally diagnosed insomnia disorders. Importantly, a selection bias preventing the generalizability of the findings to the entire SLD and ADHD must be acknowledged, since only children referred for sleep evaluation were included herein. Finally, the absence of a TD control group limits conclusions regarding the specificity of the observed sleep alterations.

In this sleep-clinic sample, children with SLD showed greater daytime sleepiness and NREM fragmentation than those with ADHD alone. These findings support routine sleep assessment in children with SLD and sleep-related or daytime complaints, but require confirmation in prospective controlled studies. These findings highlight the importance of comprehensive sleep assessment in children with NDD and suggest that sleep disturbances may represent an integral component of the neurodevelopmental phenotype in SLD.

CRediT authorship contribution statement

Julien Lioret: Formal analysis, Investigation, Writing – original draft, Writing – review & editing. **Marion Comajuan:** Writing – review & editing. **Sabine Plancoulaine:** Formal analysis, Methodology. **Daniel Gerard:** Writing – review & editing. **Christophe Gauld:** Writing – review & editing. **Marine Thieux:** Writing – review & editing. **Nicolas Beck:** Writing – review & editing. **Émilie Denechere:** Writing – review & editing. **Patricia Franco:** Conceptualization, Methodology, Supervision, Validation, Writing – review & editing. **Aurore Guyon:** Conceptualization, Supervision, Validation, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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