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# Mobile sleep EEG suggests delayed brain maturation in adolescents with ADHD: A focus on oscillatory spindle frequency

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ABSTRACT

*Background:* Attention-deficit/hyperactivity disorder (ADHD) is a prevalent neurodevelopmental disorder. Although data show ADHD is associated with sleep problems, approaches to analyze the association between ADHD and sleep electrophysiology are limited to a few methods with circumscribed foci.
*Aims:* Sleep EEG was analyzed by a mixed-radix FFT routine and power spectrum parametrization in adolescents with ADHD and adolescents not at-risk for ADHD. Spectral components of sleep EEG were analyzed employing a novel, model-based approach of EEG power spectra.
*Methods and procedures:* The DREEM mobile polysomnography headband was used to record home sleep EEG from 19 medication-free adolescents with ADHD and 29 adolescents not at-risk for ADHD (overall: $N = 56$, age range 14–19 years) and groups were compared on characteristics of NREM sleep.
*Outcomes and results:* Adolescents with ADHD exhibited lower frequency of spectral peaks indicating sleep spindle oscillations whereas adolescents not at-risk for ADHD showed lower spectral power in the slow sleep spindle and beta frequency ranges.
*Conclusions and implications:* The observed between-groups difference might indicate delayed brain maturity unraveled during sleep in ADHD.

## What does this paper add?

Although ADHD is often associated with sleep disorders, ADHD and sleep electrophysiology are limited to a few methods with circumscribed foci. This paper adds indirect evidence of delayed cortical maturation in ADHD as indexed by data-driven spectral parameters of sleep EEG.

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## 1. Introduction

Attention-deficit/hyperactivity disorder (ADHD) is an early-onset, chronic, costly, and prevalent neurodevelopmental disorder (Hámori et al., 2023). ADHD is characterized by developmentally inappropriate inattention, hyperactivity, and impulsivity symptoms and corresponding functional impairment (American Psychiatric Association, 2022). Evidence shows that adolescents with ADHD have more sleep problems and higher level of sleepiness than their typically developing peers and that these atypical sleep patterns are associated with later affective and mood disturbances (Becker et al., 2019a, 2019b; Becker et al., 2020). More precisely, approximately 50–80 % of children and adolescents with ADHD exhibit difficulties with sleep which, in turn, are associated with impaired daytime cognitive functioning and assumed cerebral maturational delay (Hobson et al., 2019; Biancardi et al., 2021). This inappropriate neural maturation may be linked to chronic sleep deprivation, which predicts later comorbid externalizing and internalizing symptoms in adolescents with ADHD (Becker et al., 2015). Experimental restriction of nighttime sleep has also been shown to exacerbate ADHD symptoms and correlates in adolescents, including inattention, oppositionality, greater daytime sleepiness and sluggish cognitive tempo (Becker et al., 2019a, 2019b). The prevalence and consequences of ADHD-related disturbances in sleep underscore the need to examine ADHD-related atypicalities in sleep, via reliance on neurophysiological studies.

Nighttime sleep electroencephalogram (EEG) data indicate that delta (1–4 Hz) and theta (4–8 Hz) power is characterized by a consistent reduction as a function of age during non-rapid eye movement (NREM) sleep in children and adolescents (Campbell & Feinberg, 2009). These findings suggest that slow-wave activity (SWA: 0.75–4.5 Hz) might be considered a developmental marker. As such, SWA may also be an ADHD diagnostic biomarker, given that SWA is associated with cortical maturation (Buchmann et al., 2011) and that ADHD is associated with delayed cortical maturation (Shaw et al., 2007; Kakuszi et al., 2020). Indeed, children and adolescents with ADHD exhibit greater SWA and theta power relative to their typically developing age-matched peers (Scarpelli et al., 2019), or altered developmental pattern of SWA (Biancardi et al., 2021). Further, recent findings indicate elevated low-frequency (3–10 Hz) oscillations during NREM, within upper SWA, low alpha, and theta ranges in ADHD, which is consistent with the delayed maturation hypothesis and suggest an atypical sleep homeostasis, including higher sleep pressure, in ADHD (Castelnovo et al., 2022).

Besides SWA, other sleep-specific correlates of cognitive functioning, such as sleep spindles are promising biomarkers of individual differences (Ujma et al., 2016). Sleep spindles are involved in sleep maintenance by inhibiting sensory input during sleep via disruption of the thalamic relay system (De Dea et al., 2018).

In typically developing boys, age is positively correlated with fast spindle amplitude, indicating that this measure could be conceptualized as a marker of sleep maturation (Ujma et al., 2016). Furthermore, findings of a longitudinal study showed oscillatory wave frequency of sleep spindles exhibited a linear increase between the ages of 6 and 18 years (Zhang et al., 2021). ADHD is associated with atypical left hemispheric power spectra activity following spindle events (De Dea et al., 2018), as well as selective power reduction in the sigma (12–15 Hz) band during stage 2 sleep (Saletin et al., 2017). Children with ADHD exhibit a greater number and density of slow spindles than fast spindles in frontal areas (Ruiz-Herrera et al., 2021), as well as higher activity, amplitude, density, and duration of these oscillatory episodes over the frontal regions (Özbudak et al., 2022).

Psychostimulants including amphetamines and methylphenidate (MPH) are first-line pharmacotherapies in ADHD (Mechler et al., 2022). Yet, in youth, beneficial daytime effects of pharmacotherapy are coupled with greater risk for insomnia and sleep problems (Biancardi et al., 2021). Further, although in one study ADHD was associated with a 20.5 % SWA reduction across the scalp, individuals with ADHD who were taking medication did not differ from typically developing controls in SWA (Furrer et al., 2019). Taken together, the ADHD-medication-sleep relation is controversial and data indicate medication status is important to account for in analyses of ADHD and sleep. Accordingly, only medication-free adolescents were included in the current study.

### 1.1. Aims

Previously, sleep EEG in ADHD has been examined using analytical methods relying on band-limited power spectra (Philipsen et al., 2005; Darchia et al., 2021) or specific spindle detection procedures (Saito et al., 2019; Merikanto et al., 2019), without considering the overall picture of electrodynamic features. Here, we examine the spectral components of sleep EEG in ADHD by employing a novel, model-based approach of EEG power spectra. The model assumes power law and oscillatory peak features as composite descriptors of the EEG power spectra (Bódizs et al., 2021). Specifically, we aim to evaluate sleep EEG patterns analyzed by FFT routine and power spectrum parametrization obtained for selected EEG derivations in adolescents with ADHD and adolescents not at-risk for ADHD. Using this analytic approach, we aim to obtain a more reliable picture of sleep characteristics in ADHD by transcending inherent redundancies in power law type spectra. We hypothesize that adolescents with ADHD will exhibit atypical SWA as indexed by model-based parameters (spectral slope, spectral intercept, spectral peak frequency, spectral peak amplitude).

## 2. Methods

### 2.1. Procedure and participant recruiting

Data for this study were obtained in the context of a larger, longitudinal study (BLINDED) whose aim was to assess behavioral and biological, affective-motivational protective and risk factors of ADHD-relevant outcomes in adolescents. For the larger study, a community sample of adolescents (oversampled for ADHD) between the ages of 14–17 years and with a range of ADHD symptoms (no-risk, at-risk for ADHD, and diagnostic) were recruited. In our present study an additional exclusion criterion was applied. Namely, all

participants in our study were at least medication-free, which meant that they could not take any psychostimulant medication at least 72 h before the tests or were medication-naïve.

ADHD classification was determined using parent-report on the ADHD Rating Scale-5 (ARS-5) (DuPaul et al., 2016). For an ADHD diagnosis, adolescents had to present ≥6 (youth <17 years old) or 5 (youth ≥17 years old) Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR) IA or H/I symptoms and exhibit impairment (i.e., rating of ≥2 =moderate impairment) in at ≥3 areas of functioning. Unanimous agreement by a child clinical psychologist and a psychiatrist was required for ADHD diagnoses.

Following informed assent and consent procedures, before EEG recordings all participants received information on the proper use of DREEM2 mobile polysomnography headband (https://dreem.com/; Arnal et al., 2020) at home, including a 10-minute training session. To ensure standardization of EEG recordings, participants were also provided with recommendations related to the circumstances of the nocturnal EEG recording; they were asked to go to bed and get up at their usual time and to preferably sleep for 7–8 h. In addition, during the study night a telephone support was provided to the participants to solve technical problems that could arise during the night.

### 2.2. Participants

Participants included in the current study were $N = 56$ medication-free adolescents (46 were medication-naïve, 10 took medication in the past), who at the time of sleep EEG recording were 14–19 years old. A total of $n = 19$ participants met criteria for ADHD (adolescents with ADHD) ($n = 13$ primarily inattentive, $n = 1$ primarily hyperactive/impulsive, and $n = 4$ combined; $M_{age}$=16.304, $SD$=1.392, 12 male). Of those who did not meet criteria for an ADHD research diagnosis ($n = 37$; $M_{age}$=16.491, $SD$=1.104), eight met criteria for being at-risk for ADHD (i.e., exhibited >=four symptoms), and were therefore excluded from further analysis. The remaining 29 participants ($M_{age}$=16.535, $SD$=1.121, 19 male) were not at-risk for ADHD (i.e., exhibited <=3 symptoms of either domain (e.g., Hámori et al., 2023)). Sleep EEG was recorded using the DREEM 2 mobile polysomnography headband in participants' homes (© 2022 Dreem; Arnal et al., 2020). The study was approved by the United Hungarian Ethical Committee for Research in Psychology (EPKEB: 2020–100) and has been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments.

### 2.3. Measures

#### 2.3.1. ADHD rating scale-5

The ARS-5 is a 30-item parent- and teacher-report measure of the past 6-month presence and severity of DSM-5 ADHD symptoms (9 inattentive symptom items and 9 hyperactivity/impulsivity symptom items) and functional impairment across six domains: relationship with significant others (family members for the home version), relationship with peers, academic functioning, behavioral functioning, homework performance and self-esteem (2 × 6 impairment items, with one set corresponding to inattention and one to hyperactivity/impulsivity). Parents and teachers rate items on a four-point scale ranging in case of symptoms from 0 (never or rarely) to 4 (very often) and in case of impairment from 0 ("no problem") to 3 ("severe problem"), with higher scores indicating more severe symptoms and impairment. The ARS-5 is comprised of two symptoms scales, Inattention and Hyperactivity-Impulsivity, and a Total Scale. The ARS-5 is suitable for ages 5–17 years, with separate forms for children (5–10 years) and adolescents (11–17 years) and age-appropriate and DSM-5 compatible descriptions of symptoms. In the current study, the adolescent home (i.e., parent-report) version was used. The ARS-5 has well-established reliability of the adolescent, home version (e.g., internal consistency and 6-week test-retest reliability) and validity (i.e., factor structure; concurrent validity and predictive validity and clinical utility) (DuPaul et al., 2016).

For purposes of the current study, the English version of the ARS-5 was translated into Hungarian following the evidence-based procedures described (Hámori et al., 2023; Sebők-Welker et al., 2023). The original author approved the translated measure (G. DuPaul, personal communication, June 5, 2020). The Hungarian translation exhibited acceptable internal consistency and convergent validity. In the current sample, internal consistency of the ARS-5 was 0.965, with McDonald's omega values of 0.949 for the Inattention subscale and 0.916 for the Hyperactivity/impulsivity subscale.

#### 2.3.2. EEG recordings

Sleep EEG signal was recorded with DREEM2 mobile polysomnography headband, including five dry-EEG sensors (F7, F8, Fp1, O1, O2), from which data were obtained bipolarly, and a built-in 3D accelerometer located over the head (which measures head position, respiratory rate/trace, and movements during sleep), as well as a red-infrared pulse oximeter for measuring heart rate, which is embedded in the front band of the device. The ground electrode was placed at Fpz.

The sampling frequency for the EEG was 250 Hz/channel. Raw data were filtered offline by a finite impulse response (FIR) bandpass filter with a low cut off frequency of 0.4 Hz and a high cut off frequency of 48 Hz, filter order was 698. DREEM includes an automatic sleep stage scoring which was manually overridden on 30 s basis [Wake, sleep stages 1 (N1), 2 (N2), and 3 (N3), and REM sleep] according to the American Academy of Sleep Medicine (AASM) by a trained expert (Berry et al., 2018). Given excessive artefacts, we did not include EEG data from occipital channels. In case of the three frontal channels (F7-F8, Fp1-F8, Fp1-F7), artefacts were detected in 5 s windows employing visual inspection.

### 2.4. Power spectral analysis

For each participant, data were analyzed from the artefact-free periods of NREM sleep (stage N2 and N3). Minimum criterion for

NREM data was having at least 500 4 s windows per participant. Analysis was performed by mixed-radix FFT routine (4 s, Hanning taper). The power spectrum was calculated between 0.5–48 Hz, with a resolution of 0.25 Hz, obtained for select EEG derivations: Fp1-F8, Fp1-F7 and F7-F8. The overlap of the FFT windows was 50 %. We defined SWA as the power spectral density of 0.75–4.5 Hz EEG activity (sum of the bin power values).

### 2.5. Spectrum parametrization

The methodology employed in the current study provides a comprehensive characterization of broadband NREM sleep EEG by parametrization of the power spectra (Bódizs et al., 2021). Here we present a brief summary of this approach which is based on the linear relationship between the logarithm of EEG power and the logarithm of the frequency. The log-log scale of the NREM sleep EEG spectra was interpolated to equally spaced bins with an interpolation factor corresponding to the smallest frequency step, and then a linear fit was applied to the data to estimate the slope (linear decay rate of the log-log power) and the y-intercept of the spectrum. During fitting, to avoid non-random oscillation domains, frequency ranges below 2 and between 6–18 Hz were excluded. Peak detection was performed in a wide, 9–18 Hz frequency sigma range, by looking for local maxima. Specifically, we used the first and second derivatives to determine critical points and thus differentiate maxima and minima. A spectral peak was accepted if the first derivative was 0 and the second derivative was less than 0. We choose the largest peak among those detected and determined its frequency (location on the horizontal scale in Hz) and amplitude (deviation from the fitted linear).

### 2.6. Statistical analysis

Analyses were conducted on the data derived from adolescents with ADHD and adolescents not at-risk for ADHD.

To assess differences between groups, spectral components of sleep EEG were compared across each index, namely, SWA, spectral slope, spectral intercept, maximum peak amplitude and maximum peak frequency, computed on the average of the Fp1-F8 and F7-F8 electrodes.

In addition, permutation tests were conducted to compare on the power of each frequency bin (0.5 to 30 Hz with increments of 0.25 Hz), with 2000 iterations. The aim of these analyses was to cross-check the findings of the spectrum parameterization approach and provide parallel statistical tests without assuming linearity of the sleep EEG power in the log-log plane.

## 3. Results

Assumption checks were conducted (Table 1.), and accordingly, the adjusted *p*-values of peak amplitude and peak frequency are reported with the appropriate effect size measures.

Groups (adolescents with ADHD vs. adolescents not at-risk for ADHD) did not differ on the following indices: SWA, spectral slope, spectral intercept and maximum peak amplitude. Descriptive and inferential statistics are detailed in Table 2. Hypothesis-testing of the maximum spectral peak frequency, however, revealed significant differences ($W = 182$; $p = 0.049$; $r_{rb} = -0.339$). Former studies indicate age and sex related differences in terms of sleep spindle frequency (Bódizs et al., 2021; Zhang et al., 2021), therefore, we controlled these covariates in a general linear model as well. Results of age-controlled analysis indicate the significance of ADHD vs. not at-risk for ADHD difference in maximal spectral peak frequency ($F(1, 45) = 4.34$, $p = 0.042$). Figs. 1–5. depict the descriptives of all analyzed parameters.

**Table 1**
Assumption Checks (ADHD symptoms).

| *Normality test (Shapiro-Wilk)* | | | |
|---|---|---|---|
| | Group | *W* | *p* |
| SWA | ADHD | 0.918 | 0.106 |
| | not at-risk | 0.98 | 0.832 |
| slope | ADHD | 0.96 | 0.576 |
| | not at-risk | 0.964 | 0.404 |
| intercept | ADHD | 0.931 | 0.178 |
| | not at-risk | 0.97 | 0.563 |
| maxpeak freq | ADHD | 0.846 | 0.006* |
| | not at-risk | 0.976 | 0.739 |
| maxpeak amp | ADHD | 0.922 | 0.121 |
| | not at-risk | 0.976 | 0.73 |
| *Equality of variances test (Levene's)* | | | |
| | *F* | *df* | *p* |
| SWA | 0.118 | 1 | 0.733 |
| slope | 0.037 | 1 | 0.848 |
| intercept | 0.212 | 1 | 0.647 |
| maxpeak freq | 0.010 | 1 | 0.921 |
| maxpeak amp | 7.323 | 1 | 0.01* |

*Note.* *=$p < 0.05$.

**Table 2**
Descriptive and inferential statistics (ADHD risk groups).

| | | Mean | SD | test statistic | p | effect size |
|---|---|---|---|---|---|---|
| SWA | ADHD | 6.640 | 0.651 | $t(46) = 0.768$ | $p = 0.447$ | $d = 0.227$ |
| | not at-risk | 6.489 | 0.678 | | | |
| slope | ADHD | -2.203 | 0.221 | $t(46) = -0.390$ | $p = 0.698$ | $d = -0.115$ |
| | not at-risk | -2.178 | 0.214 | | | |
| intercept | ADHD | 4.804 | 0.850 | $t(46) = 0.949$ | $p = 0.348$ | $d = 0.280$ |
| | not at-risk | 4.579 | 0.769 | | | |
| maxpeak freq | ADHD | 11.264 | 0.610 | $W = 182$ | $p = 0.049$ | $r_{rb} = -0.339$ |
| | not at-risk | 11.617 | 0.474 | | | |
| maxpeak amp | ADHD | 1.057 | 0.710 | $t(46) = 1.361$ | $p = 0.180$ | $d = 0.402$ |
| | not at-risk | 0.834 | 0.429 | | | |

*Note.* asd

In addition, permutation test (Fig. 6) of power values at frequency bins indicated increased spectral power in NREM sleep EEG of adolescents with ADHD as compared to adolescents not at-risk for ADHD in frequencies covering roughly the slower spindle ranges: 10 Hz ($p = 0.041$), 10.25 Hz ($p = 0.029$), 10.5 Hz ($p = 0.023$), 10.75 Hz ($p = 0.020$), 11 Hz ($p = 0.035$) and 11.25 Hz ($p = 0.043$); as well as in the beta frequency ranges: 15.5 Hz ($p = 0.046$), 15.75 Hz ($p = 0.043$), 16 Hz ($p = 0.047$), 17 Hz ($p = 0.046$), 17.25 Hz ($p = 0.045$), 17.5 Hz ($p = 0.045$), 17.75 Hz ($p = 0.044$), 18 Hz ($p = 0.030$), 18.25 Hz ($p = 0.047$). In addition, groups marginally differed at the lower and upper borders of these ranges [9.75 Hz ($p = 0.065$) and 11.5 Hz ($p = 0.051$) 14.75 Hz ($p = 0.060$), 15.25 Hz ($p = 0.059$), 16.25 Hz ($p = 0.053$) 16.5 Hz ($p = 0.053$), 16.75 Hz ($p = 0.059$), 18.5 Hz ($p = 0.058$)].

## 4. Discussion

We examined indirect evidence of the delayed cortical maturation hypothesis of ADHD. Typical brain maturation is characterized by drastic changes in the sleep EEG (Graven, 2006; Tarokh & Carskadon, 2010); whereas lack of changes are indicative of atypical maturation. Accordingly, previous studies reported ADHD-related differences in various neurophysiological markers of sleep (Ruiz-Herrera et al., 2021; Biancardi et al., 2021; Scarpelli et al., 2019). Slow-wave sleep has been described to display a decreasing trend as a function of age (Ricci et al., 2021; Ohayon et al., 2004; Gaudreau et al., 2001), that is to say, a developmental reduction of sleep pressure is apparent. However, in contrast to frequently reported sleep disturbances in ADHD and thus our hypotheses, the current findings are not indicative of differences in SWA between adolescents with and not at-risk for ADHD. More precisely, neither spectral slope, indicating the ratio of slow to fast oscillations, nor total power in the slow wave range revealed atypical sleep pressure in ADHD. Studies investigating the aperiodic component of the power spectrum in resting and task-related states have generally reported a link between the spectral exponent and ADHD (Robertson et al., 2019; Karalunas et al., 2022; Ostlund et al., 2021). The difference between our results and the findings of other studies may arise from our different data recording approach, namely, mobile polysomnography, resulting in somewhat more artefact-contaminated data, but higher ecological validity, namely better representation of habitual sleep. The bipolarly derived and reduced montage setup of our devices may also contribute to contrasting results. Furthermore, we only involved medication-free participants in our current study, which could result in subtle differences in sleep EEG findings compared to previous reports on various groups of drug-naïve or pharmacologically treated adolescents with ADHD. Studies investigating the effects of ADHD-related medical treatment on sleep have yielded conflicting results (Biancardi et al., 2021; Furrer et al., 2019), with some data indicating that ADHD pharmacotherapy might result in long-term effects, well beyond 48 h (Schlochtermeier

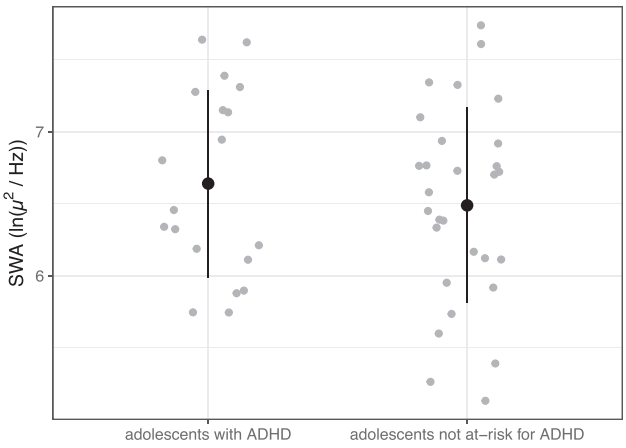


**Fig. 1.** NREM sleep EEG SWA as a function of ADHD.

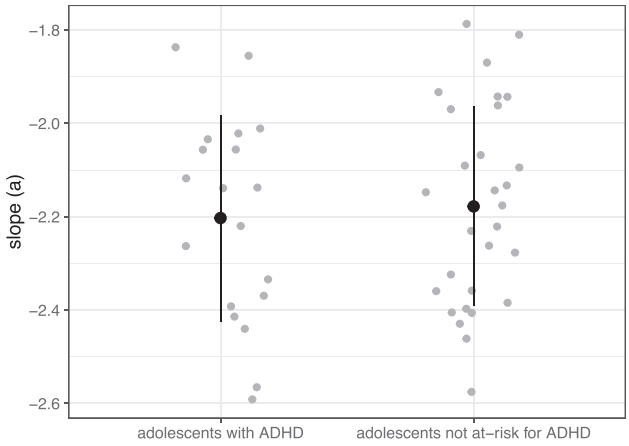


**Fig. 2.** NREM sleep EEG spectral slope as a function of ADHD.

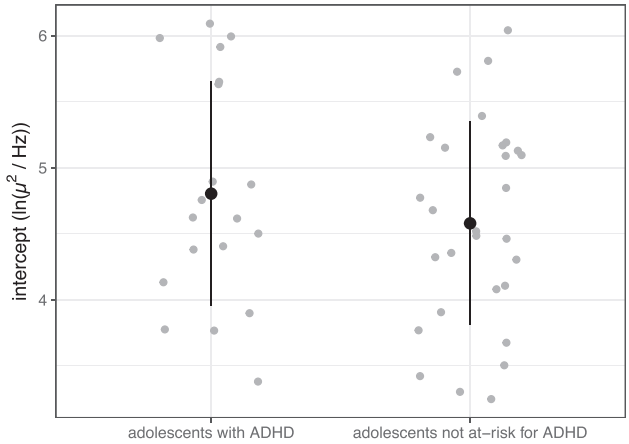


**Fig. 3.** NREM sleep EEG spectral intercept as a function of ADHD.

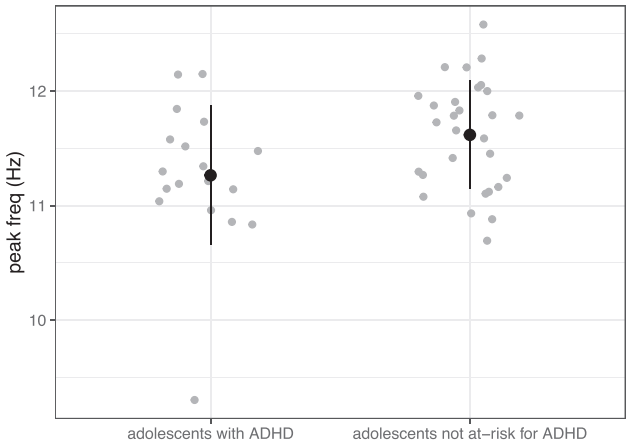


**Fig. 4.** NREM sleep EEG 9–18 Hz spectral peak frequency as a function of ADHD.

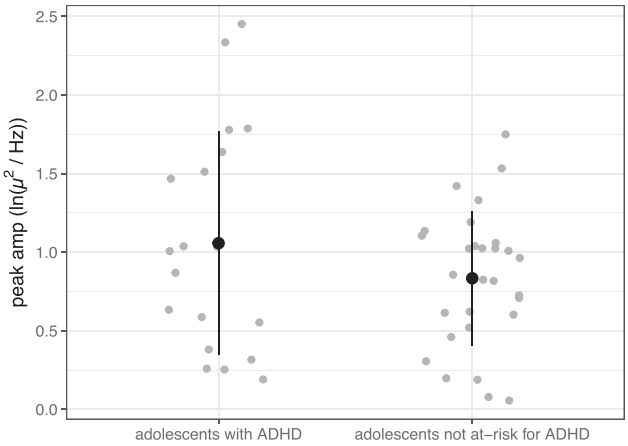


**Fig. 5.** NREM sleep EEG 9–18 Hz spectral peak amplitude as a function of ADHD.

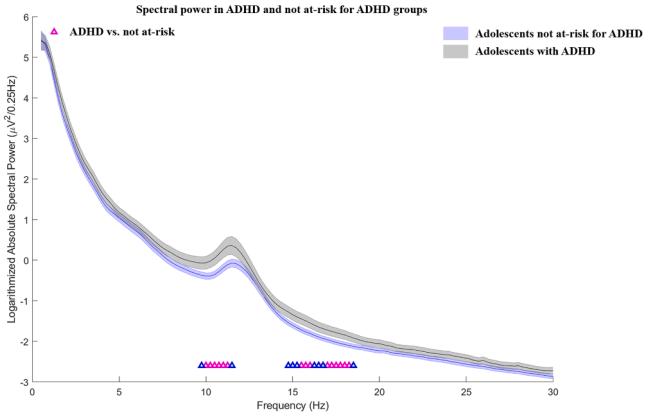


**Fig. 6.** Permutation test of frequency bins contrasting adolescents with ADHD and adolescents not at-risk for ADHD. *Note*: pink triangle indicates statistical significance at $p < 0.05$, blue triangle indicates marginal significance at $0.07 > p > 0.05$.

et al., 2011). It is thus reasonable to assume that medication may be a confounding variable, therefore, it is imperative to account for medication status in analyses of ADHD and sleep.

A further, relevant point of consideration is that ADHD medication might have long-term behavioral and/ or physiological effects apparent beyond 48 h (Schlochtermeier et al., 2011). Such pharmacological effects may be reflected in wake state EEG, which in turn could influence sleep through an interaction between wakefulness and sleep as well as the known sensitivity of NREM SWA to sleep pressure. We analyzed home-recorded (mobile polysomnography) whole-night EEG data enhancing ecological validity. Yet, as participants exhibited variability in the amount of artefact-free NREM episodes, mobile polysomnography may reduce data quality.

The peak frequency in the sigma band is a measure of the oscillatory wavelength of sleep spindles, which are postulated to be involved in sleep maintenance (De Dea et al., 2018). Consistent with hypotheses, whitened peak frequency revealed significant differences between adolescents with and not at-risk for ADHD, such that former group exhibited lower peak frequencies. This finding was partially supported by binwise spectral analyses of NREM sleep EEG: whereas for those with ADHD, power in the low sleep spindle ranges exceeded the respective values of those not at-risk for ADHD, in the upper sleep spindle ranges, no differences were detected. As sleep spindles display an age-dependent frequency increase (Bocskai et al., 2022; Zhang et al., 2021), these results evince ADHD is characterized by atypical brain maturation and might confirm hypotheses of delayed maturation.

The mobile headband may prove useful for assessing sleep in neurodevelopmental and other psychiatric disorders beyond ADHD, because atypical sleep is also characteristic of these diagnoses and thus relevant to case conceptualization and planning of treatment. However, healthcare professionals and researchers often encounter difficulties. Mobile technology, by allowing for measurement of sleep to take place in the home of the patient, provides results that better reflect the typical sleep patterns of the patient and may also represent a new supplementary method for sleep studies.

Related, it will be an important next step in this line of research to determine the extent to which the between-groups differences observed herein are driven by ADHD and/ or presence of comorbid neurodevelopmental or other psychiatric disorders or symptoms for which both atypical sleep and ADHD comorbidity are relevant.

In childhood, sleep is directly linked to development and indirectly to learning. However, measuring this complex process in a laboratory setting is challenging, as participants are difficult to motivate to participate in measurements (often multiple). Use of mobile headband technology for assessments conducted in home settings may offer a solution to these laboratory challenges.

## 5. Conclusion

The present data-driven method of EEG spectrum parametrization is ideal to deal with the high inter-individual variability of neural signals (Bódizs et al., 2021). More precisely, the whitened measure of the peak frequency in the sigma range is adjusted by the aperiodic background activity following a 1/f power law; making this approach more sensitive to individual differences and thus allowing better assessment of sleep in diagnostic practice. In addition, the spectral slope, indicating the ratio of low to high frequencies, is not biased by arbitrarily choosing the frequency ranges of band-limited power analyses, including the widely used theta-beta ratio-type measure assessed during resting state conditions of ADHD participants (Saad et al., 2018). By relying on this computational approach, as well as on home recordings providing high ecological validity we found no evidence for an altered sleep intensity as revealed by steeper NREM sleep EEG spectral slopes in 14–19 years old participants with ADHD. Indeed, we found lower oscillatory spectral peak frequency in the sleep spindle range, which was found to be a characteristic feature of younger age groups in several former studies. The lack of a difference in sleep intensity and the decelerated sleep spindle frequency were partially replicated by band-limited spectral analysis of SWA and by binwise power differences according to permutation test statistics, respectively. Together these results indicate the relevance of the appropriate use of sleep-wake-specific neurophysiological measures in biomarker studies of ADHD, as well as the need to consider factors like, ecological validity, medication effects, and the potential age-specificity of several developmental markers of sleep. Last, but not least, the need to consider ADHD symptoms seems to be a crucial factor in later sleep studies in the field of developmental disorders.

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## CRediT authorship contribution statement

**Magyar Tárek Zoltán:** Writing – original draft. **Blanka Vojnits:** Writing – original draft. **Bódizs Róbert:** Writing – original draft. **Takács Mária:** Writing – original draft. **Reicher Vivien:** Writing – original draft. **Szalárdy Orsolya:** Writing – original draft. **Bunford Nóra:** Writing – original draft.

## Conflicts of Interest

None.

## Data availability

Data will be made available on request.

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