

1 Wake EEG oscillation dynamics reflect both sleep pressure and 2 brain maturation across childhood and adolescence

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27 1 ABSTRACT

28 An objective measure of brain maturation is highly insightful for monitoring both typical

29 and atypical development. Slow wave activity, recorded in the sleep electroencephalogram (EEG),

30 reliably indexes age-related changes in sleep pressure as well as deficits

31 related to developmental disorders such as attention-deficit hyperactivity disorder

32 (ADHD). We aimed to determine whether wake EEG measured before and after sleep

33 could index the same developmental changes in sleep pressure, using data collected

34 from 163 participants 3-25 years old. We analyzed age- and sleep-dependent changes

35 in two measures of oscillatory activity, amplitudes and density, as well as two

36 measures of aperiodic activity, offsets and exponents. We then compared these wake

37 measures to sleep slow wave amplitudes and slopes. Finally, we compared wake EEG

38 in children with ADHD (N=58) to neurotypical controls. Of the four wake measures, only

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1 oscillation amplitudes consistently exhibited the same changes as sleep slow waves.
2 Wake amplitudes decreased with age, decreased after sleep, and this overnight de-
3 crease decreased with age. Furthermore, wake amplitudes were significantly related to
4 both sleep slow wave amplitudes and slopes. Wake oscillation densities decreased
5 overnight in children but increased overnight in adolescents and adults. Aperiodic off-
6 sets decreased linearly with age, decreased after sleep, and were significantly related
7 to sleep slow wave amplitudes. Aperiodic exponents also decreased with age, but in-
8 creased after sleep. No wake measure showed significant effects of ADHD. Overall, our
9 results indicate that wake oscillation amplitudes, and to some extent aperiodic offsets,
10 behave like sleep slow waves across sleep and development. At the same time, over-
11 night changes in oscillation densities independently reflect some yet-unknown shift in
12 neural activity around puberty.

13 Keywords: development, EEG, sleep, synaptic homeostasis hypothesis, aperiodic activ-
14 ity, oscillation bursts

15 **1 INTRODUCTION**

16 The EEG is one of few tools available to study the human developing brain already from
17 birth (Korotchikova et al., 2009). It is non-invasive, relatively cheap, and provides a real-
18 time readout of neuronal activity. It is an incredibly rich signal, with the potential as a
19 prognostic and diagnostic tool for both typical development and disease. Sleep EEG,
20 and in particular slow wave activity (0.5-4 Hz) during NREM sleep, has proven especially
21 sensitive to brain maturation (Campbell & Feinberg, 2009) and developmental disorders
22 such as ADHD (Furrer et al., 2019). This is because slow wave activity reflects the over-
23 all synchronicity of the brain, which decreases with age following decreasing synaptic
24 density across adolescence (Campbell & Feinberg, 2009; Huttenlocher, 1979; Jenni &
25 Carskadon, 2004), and may be lower in ADHD due to reduced cortical thickness (Shaw
26 et al., 2006). Furthermore, slow wave activity is greater in occipital regions in younger
27 children, and greater in frontal regions in adolescents and adults (Kurth et al., 2010),
28 possibly reflecting the slower maturation of higher order association areas (Shaw et al.,
29 2008).

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1 In addition to these developmental phenomena, sleep slow waves reflect the buildup
2 and dissipation of homeostatic sleep pressure, increasing following wake and decreas-
3 ing during sleep (Borbély, 1982). These hourly changes in sleep pressure detected
4 through slow wave activity are hypothesized to reflect synaptic *plasticity*: synaptic
5 strength increases with wake and daytime learning, and decreases during sleep (Tononi
6 & Cirelli, 2003, 2014). Generally, plasticity decreases with brain maturation across
7 childhood and adolescence and this is reflected in decreases in the overnight *changes*
8 in slow waves with age (Jaramillo et al., 2020). Thus, both absolute slow wave activity
9 and changes in slow wave activity are markers of brain development, reflecting average
10 synaptic density and synaptic plasticity respectively. While these predictions have been
11 repeatedly tested and validated in sleep, their consequences on the wake EEG have not
12 been as systematically investigated. Previous research has found correlations between
13 wake and sleep EEG power (Finelli et al., 2000), however, greater insights can be de-
14 rived from more specific analyses.

15 The EEG is made up of both *periodic* activity and *aperiodic* activity (Figure 1A, Do-
16 noghue et al., 2020). Periodic activity refers to oscillations, which appear as quasi-
17 gaussian bumps in the power spectrum at their corresponding frequency. Instead, ape-
18 rioidic activity is a form of background “noise,” producing the characteristic 1/f curve in
19 the EEG power spectrum. Aperiodic activity is defined by its *offset* (reflecting the overall
20 aperiodic power) and *exponent* (the steepness of the curve). Changes in exponent in
21 particular are hypothesized to reflect alterations in excitatory/inhibitory balance of neu-
22 ronal activity (Brake et al., 2024; Gao et al., 2017), and this could explain the multitude
23 of conditions which affect aperiodic signals.

24 Aperiodic exponents reflect levels of consciousness, becoming progressively steeper
25 with increasing sleep depth (Lendner et al., 2020, 2023; Schneider et al., 2022), anes-
26 thesis (Colombo et al., 2019), and disorders of consciousness (Colombo et al., 2023;
27 Maschke et al., 2023). Additionally, exponents become progressively shallower during
28 sleep, reflecting the dissipation of sleep pressure (Bódizs et al., 2024; Horváth et al.,
29 2022). Brain maturation also strongly affects exponents and offsets, becoming shal-
30 lower and lower from childhood to adulthood (Cellier et al., 2021; Hill et al., 2022; Trön-
31 dle et al., 2022), with age and sleep depth interacting (Favaro et al., 2023). Lastly, dif-

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1 ferences in aperiodic exponents and offsets have been found in children with ADHD
2 compared to neurotypical controls (Ostlund et al., 2021; Robertson et al., 2019).

3 Periodic activity, like aperiodic activity, can be estimated from spectral power by simply
4 subtracting the aperiodic activity from overall power, giving *periodic power* (Figure 1A).
5 However this misses two important independent changes that can happen to oscillations:
6 they can change in *amplitude*, and they can change in *density* (Figure 1B). The
7 amplitudes of oscillations reflect the synchronicity of the oscillating neuronal popula-
8 tion, and that synchronicity is determined both by the number of neurons in phase with
9 each other and the strength of their synaptic connections. Whether an oscillation oc-
10 curs at all (i.e., density) will instead depend on the activity of “pacemaker” interneurons
11 which entrain a population of neurons to the same rhythm (Le Bon-Jego & Yuste, 2007;
12 Perkel et al., 1964), and this pattern of rhythmic firing will be in service of some underly-
13 ing function that will come and go as needed. In short, amplitudes reflect synchronicity
14 and densities reflect activity.

15 Since oscillation amplitudes reflect synchronicity, this means they should reflect the
16 same information as slow waves measured during sleep. Sleep slow wave activity in-
17 creases along a saturating exponential function relative to the prior duration of wake-
18 fulness, increasing rapidly after only a few hours awake and increasing less and less
19 with further hours awake (Borbély, 1982; Dijk et al., 1987). We found that wake oscilla-
20 tion amplitudes follow the same trajectory across 24 hours awake (Snipes et al., 2023).
21 Instead, alpha oscillation densities (8-12 Hz) decreased, supporting the specificity of
22 the effect to amplitudes and masking the effect when measuring spectral power.

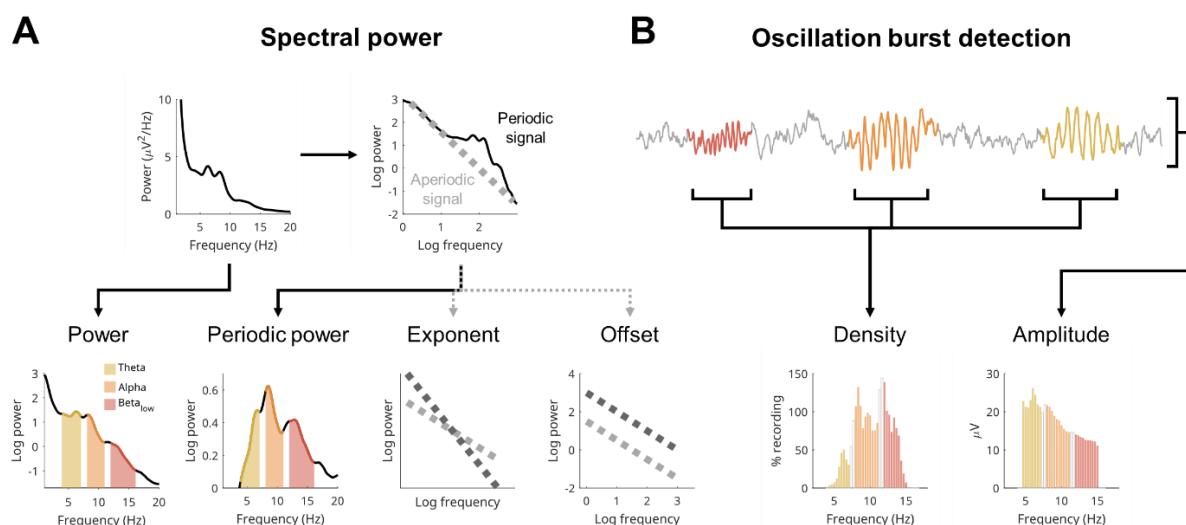
23 Given these results, we hypothesized that wake oscillation amplitudes should behave
24 like sleep slow waves also across development: absolute amplitudes should decrease
25 with age, and overnight changes in amplitude should decrease with age. Likewise,
26 changes in amplitude should also manifest an anterior-posterior gradient with age:
27 larger amplitudes in occipital regions in children and larger in frontal regions in adults.
28 Furthermore, given that children with ADHD have lower sleep slow wave activity, they
29 should likewise have lower wake amplitudes. In short, if both wake amplitudes and
30 sleep slow waves are supposed to reflect brain-wide synchronicity, then wake ampli-
31 tudes should also reflect sleep pressure, brain development, and the pathophysiology

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1 of ADHD. We further hypothesized that all these effects would be specific to amplitudes, with other wake EEG parameters changing independently with sleep and age.

3 Finally, we expected that wake amplitudes, more than any other wake EEG measure, 4 should directly correlate with specific sleep markers of synaptic plasticity: slow wave 5 amplitudes and slopes (the steepness of the waves) measured at the beginning and end 6 of the night (Esser et al., 2007; Jaramillo et al., 2020; Riedner et al., 2007; Vyazovskiy et 7 al., 2009). Slopes in particular are thought to reflect changes in synaptic strength. The 8 steepness of a slow wave is determined by the rate at which neuronal populations synchronize 9 and desynchronize firing, and increased synaptic strength will increase the speed of such synchronization (Vyazovskiy et al., 2009). Therefore, any wake measure 10 of sleep pressure and synaptic plasticity should correlate with both sleep slow wave 11 amplitudes and especially sleep slow wave slopes.

13 To answer these questions, we analyzed data collected from previous studies at the 14 University Children's Hospital of Zurich, with high-density wake EEG recordings measured 15 the evening before and morning after a night of sleep. The final dataset included 16 105 neurotypical participants from the ages of 3.5 to 25, and 58 children with ADHD. 17 From 44 neurotypical participants, the wake EEG was further compared to their sleep 18 EEG.



20 **Figure 1: Wake EEG measures. A:** Measures based on spectral power. Given the nature of power, it is traditionally 21 analyzed log-transformed to have more normally distributed values. These are then aggregated into bands. Here, we 22 focus on classical wake EEG bands: theta (4-7 Hz, yellow), alpha (8-11 Hz, orange) and low beta (12-16 Hz, red), with 23 gaps between bands to avoid overlapping information. The example comes from a 15-year-old male participant, 24 used for the entire figure. The EEG signal is composed of aperiodic "background activity" (gray signal in B) and oscillations.

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1 latory activity (colored signal in B). When plotting the power spectrum on a log-log scale, a line can be fitted to the
2 aperiodic component of the signal, which can be subtracted from the whole spectrum, leaving behind only periodic
3 power. The aperiodic line can then be quantified by its offset (where it intersects 0 on the log-log scale, i.e. the log
4 power at 1 Hz), and its exponent (how tilted it is). Thus, the power spectrum provides four measures: log-transformed
5 power, periodic power, exponent, and offset of the aperiodic signal. **B:** Cycle-by-cycle analysis is used to detect
6 bursts of oscillations by identifying sections of the EEG signal that show periodic activity (colored), relative to the
7 aperiodic background activity (gray). Once bursts are detected, there are two main parameters to quantify them:
8 density (how much of the signal in time contains an oscillation) and amplitude (average peak-to-peak voltage of each
9 oscillation). Densities are expressed in percentage, and when pooling bursts detected in all channels, they can easily
10 exceed 100%, as the same burst will appear in multiple channels and multiple bursts can co-occur. Amplitudes are
11 in microvolts. The EEG trace was stitched together for illustrative purposes. N.B. Beta periodic power is lower than
12 alpha periodic power (in panel A), but their densities (in B) are roughly the same; this is because periodic power is
13 also influenced by the lower amplitudes of low beta compared to alpha. The densities of other participants are pro-
14 vided in Suppl. Figure 1-1.

15 2 METHODS

16 2.1 Datasets

17 The data for this manuscript was assembled from previous studies conducted between
18 2008 and 2021. The participant demographics of each dataset are in Table 1. In total,
19 we included 163 participants between the ages 3.5 and 24.7, 38% female, 7% left-
20 handed. Of these, 36% were diagnosed with ADHD at the department for Child and
21 Adolescent Psychiatry at the University of Zurich, the outpatient clinic of the Child De-
22 velopment Center, and at private children's clinics in Zurich Oerlikon. Patients were not
23 excluded based on medication status, and therefore were a mixture of medicated, pre-
24 viously medicated, and unmedicated (see Table 2, and (Furrer et al., 2019; Ringli et al.,
25 2013)). Otherwise, all participants were screened by telephone such that they all were
26 completely healthy, took no (other) medication, had no (other) comorbidities, and were
27 good sleepers. All participants were recruited from canton Zurich, Switzerland, and
28 recorded at the University Children's Hospital of Zurich, except for the children from 3.5
29 to 8 (Dataset2009, N=11), who were recruited in Providence, RI, USA, and recorded at
30 home. Sleep time was determined by their individual preferred sleep and wakeup time,
31 which they had to maintain the week prior to each measurement. Wake measurements
32 were done just before going to sleep, and ~30 minutes after waking up. 115 participants
33 had 2 sessions, spaced at least 1 week apart, both included in these analyses. Depend-
34 ing on the dataset, different paradigms were used involving different wake tasks (de-
35 scribed below) and there could be 1-4 tasks at each time point (see Table 1). Every set
36 of tasks for each dataset was repeated in the evening and in the morning. In total, 1243
37 recordings were included in these analyses lasting on average 4.2 min (task time + buff-

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1 er – artefacts), with a standard deviation of 2.5 min. The data (averaged across chan-
2 nels) for all participants is provided in Suppl. Data 1.

3 Informed consent was obtained from all adult participants, and from the legal guardi-
4 ans of all children below 14, as well as from adolescent participants 14-18. All studies
5 were approved by the local ethics committees and performed according to the declara-
6 tion of Helsinki.

	N	Female	Left-handed	ADHD	Age range	Mean age	Paradigm	Sessions	Recordings
	#	%	%	%	(years)	(years)		#	#
Dataset2008	38	32	0	0	8.7-23.4	14.1 (3.8)	Adaptation	2	7.3/8
Dataset2009	11	73	0	0	3.5-8.0	5.6 (1.5)	Oddball	1	1.8/2
Dataset2010	28	21	14	100	9.7-16.3	12.7 (1.9)	Adaptation	1	2.8/4
Dataset2016	18	44	0	0	18.4-24.7	21.6 (2.1)	Oddball	2	3.9/4
Dataset2017	42	43	17	36	8.1-17.6	12.2 (2.7)	Attention	2	12.7/16
Dataset2019	26	38	0	58	8.8-16.8	11.4 (2.0)	Attention	2	10.1/12
All	163	38	7	36	3.5-24.7	13.2 (4.4)			

7 **Table 1: All participants' demographics, split by dataset.** The year for each dataset indicates the beginning of data
8 collection. N indicates the number of participants. After the mean age of each dataset, the standard deviation is
9 provided in parentheses. Paradigm indicates which set of wake tasks were recorded. Sessions indicate the number
10 of sessions expected for each dataset, although in practice due to dropouts, some participants only completed 1.
11 Recordings indicate the average number of recordings per participant compared to the total number of recordings
12 expected by the experimental paradigm. Recordings were missing either because they contained too many artefacts
13 or were omitted entirely during data collection.

	N	Female	Left-handed	Age range	Mean age	Oddball
	#	%	%	(years)	(years)	%
Medicated in the past	6	17	0	9.7-14.8	12.5 (2.2)	33
Unmedicated	14	21	14	9.5-15.3	11.3 (1.6)	64
Medication the day before	22	23	14	8.7-16.3	12.3 (2.2)	45
Medication the day of	13	15	0	9.0-16.1	12.3 (2.0)	54
All patients	55*	20	9	8.7-16.3	12.1 (2.0)	51
Controls	105	47	6	3.5-24.7	13.8 (5.2)	58

14 **Table 2: ADHD demographics, split by patient status.** For each patient group, the Oddball column indicates how
15 many came from a dataset performing the oddball task (rather than the attention tasks). *For 3 patients, medication
16 status was missing, therefore the true total is 58.

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1 2.1.1 Oddball & motor adaptation paradigms

2 95 participants (Dataset2008, Dataset2009, Dataset2010, Dataset2016) performed an
3 auditory oddball task during their wake EEG. The task lasted 4 minutes and was per-
4 formed in the evening just before going to bed and in the morning ~30 minutes after
5 waking up. The task involved 300 tones at ~80 dB, with an interstimulus interval of 0.8 s.
6 A random 10% of stimuli were targets to which the participant had to push a button in
7 response. For the youngest children (Dataset2009), the 4-minute task was split into 2
8 segments, and for the adults (Dataset2016) the task was extended to 6 minutes.

9 66 of these participants (Dataset2008, Dataset2010) also performed a half-hour visuo-
10 motor adaptation task (Ghilardi et al., 2000) followed by a second oddball. One dataset
11 (Dataset2008) also included a second session with a control visuomotor task (no adap-
12 tation), counterbalanced with the motor adaptation task. The motor tasks were not in-
13 cluded in this analysis, because they further differed from evening to morning. For more
14 details on the adaptation task see Wilhelm et al. (2014). The youngest (Dataset2009)
15 and oldest (Dataset2016) participants only conducted one oddball and no motor task.
16 Dataset2016 also had two sessions, one night with phase-targeted auditory stimulation
17 during NREM sleep and the other sham. The experimental conditions did not affect the
18 wake EEG (data not shown).

19 The sleep data from these participants has been published (Buchmann et al., 2011;
20 Furrer et al., 2019, 2020; Jaramillo et al., 2020; Kurth et al., 2010; Ringli et al., 2013; Volk
21 et al., 2018; Wilhelm et al., 2014), as has a subset of the wake EEG data (Fattinger et al.,
22 2017).

23 2.1.2 Attention paradigm

24 68 participants (Dataset2017, Dataset2019) performed three tasks with a focus on at-
25 tention. These were studies investigating the relationship between slow waves, behav-
26 ior, and MR spectroscopy (Jaramillo et al., 2020; Volk et al., 2019). This included two
27 sessions to compare the effects of phase targeted auditory stimulation on slow waves
28 in sleep (sham and stimulation; data currently unpublished). The wake tasks were part
29 of the Test Battery for Attentional Performance (TAP) (Zimmermann & Fimm, 2012),
30 which included 2 minutes of a visual Go/No-Go task (respond to 1 stimulus, withhold

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1 response to another), 5.5 minutes of a visual and acoustic Alertness task, and then 2
2 1.5-minute Fixation recordings. For one dataset (Dataset2019) the Go/No-Go task was
3 adapted using cartoon images (extended to 12 minutes), but only 1 Fixation recording
4 was measured (lasting 2 minutes).

5 **2.2 EEG recordings and preprocessing**

6 All datasets were measured using 128 channel EGI Geodesic Sensor nets and EGI am-
7 plifiers (Electrical Geodesics Inc., Eugene, OR, USA). Wake recordings were done with
8 Cz reference, 1000 Hz sampling rate, and impedances kept below 50 kOhm. All anal-
9 yses were performed in MATLAB 2023b, with the EEGLAB toolbox v2023.1 (Delorme &
10 Makeig, 2004), the FOOOF/specparam toolbox v1.1.0 (Donoghue et al., 2020), and cus-
11 tom scripts.

12 EEG data was first mean-centered, then lowpass filtered at 40 Hz (EEGLAB's
13 *pop_eegfiltnew* function) and notch-filtered at either 50 or 60 Hz (Dataset2009) along
14 with subsequent harmonics. The data was downsampled to 250 Hz, then highpass-
15 filtered over 0.5 Hz (Kaiser filter, stopband = 0.25 Hz, stopband attenuation = 60, pass-
16 band ripple = 0.05).

17 Artifacts were removed with a fully automated procedure. Movement and other large
18 artifacts were detected in data filtered between 1 and 40 Hz, in 3 s segments. A seg-
19 ment was labeled a "major artifact" if it exceeded 500 μ V, or a "minor artifact" if the
20 correlation with neighboring channels was below .3. Major artifacts were always re-
21 moved, either by removing all data in all channels during those 3 s, or removing the en-
22 tire channel with such an artifact, depending on which (channel or segment) removed
23 the least amount of clean data. Minor artifacts were removed in a similar way, removing
24 iteratively either the channel with the most artifactual segments, or the segments with
25 the most artifactual channels, until all channels and all segments had at most 30% of
26 the data containing a minor artifact. Flat channels were removed using EEGLAB's
27 *clean_artifacts* function. The missing Cz channel was added (as a vector of zeros), then
28 the data was average referenced. Physiological artifacts (blinks, eye movements, mus-
29 cle tone, heartbeat) were removed with independent component analysis (ICA), with
30 components calculated separately as described in the next section. After these were

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1 removed, a second pass was conducted using EEGLAB's *clean_windows* function
2 (MaxBadChannels = .3, PowerTolerances = [-inf, 12]), then bad segments/channels still
3 containing amplitudes over 140 μ V were removed, and finally EEGLAB's
4 *clean_channels_nolocs* was applied (MinCorrelation = .5, IgnoredQuantile = .1, Max-
5 BrokenTime = .5). Recordings for which more than 25 channels were removed, or which
6 had less than 1 minute of data, were excluded from analysis. In a last step, EEG chan-
7 nels were interpolated, for a total of 123 channels, excluding the external electrodes
8 (49 56, 107, 113) and the face electrodes (126, 127).

9 **2.2.1 Automatic detection and removal of physiological artefacts using ICA**

10 For ICA, a separate copy of the EEG data was preprocessed as previously described,
11 however the data was filtered between 2.5 and 100 Hz, and downsampled to 500 Hz.
12 Automatically detected bad channels and bad time windows were removed, an empty
13 Cz channel added, and then the data was re-referenced to the average of all channels.
14 EEGLAB's *runica* function was run with principal component analysis (PCA) rank reduc-
15 tion. Then, components were automatically classified with EEGLAB's *iclabel*, as either
16 brain, muscle, eye, heart, line, channel noise, or other. This function provides a proba-
17 bility score for each label from 0 to 1, so the label with the largest score for each com-
18 ponent was taken. Components classified as muscle, eye, or heart were removed. Of
19 the remaining noise classifications (line, channel, other), due to poor classification ac-
20 curacy, an additional step was implemented. Spectral power was calculated for each
21 component (*pwelch*, 4 s Hanning windows, 50% overlap), then smoothed over 5 Hz
22 (*lowess*) to facilitate model fitting. Unlike for the main analyses, the periodic signal was
23 not of interest, therefore stronger smoothing was possible. The *specparam* algorithm
24 (Donoghue et al., 2020; Ostlund et al., 2022) was applied to the power spectrum be-
25 tween 8 and 30 Hz to extract aperiodic exponents. Components for which the spectral
26 exponent was shallower than 0.5 (so almost flat or even tilted positive), were consid-
27 ered noise and therefore excluded, as they reflected either muscle activity or other non-
28 physiological signals. Using the manually labeled components in an independent adult
29 dataset (Snipes et al., 2022), we confirmed that this procedure was sufficiently compa-
30 rable to human detection of artifactual components. We further confirmed that the
31 outcome matched human component classification in a small subset of the children's

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1 data. However, considering the trend towards 0.5 exponents observed in Figure 2, for
2 future datasets with older participants we would recommend a lower threshold.

3 For the Dataset2009 cohort of <8-year-olds, given how little data there was and how
4 many more movement artifacts, we chose to apply to same manual artifact rejection as
5 in Snipes et al. (2022) to preserve as much data as possible.

6 **2.3 Burst detection**

7 Bursts of oscillations were detected using cycle-by-cycle analysis (Cole & Voytek, 2019)
8 implemented in MATLAB (Snipes et al., 2023). Bursts were detected with the same
9 thresholds as in Snipes et al. (2024). Briefly, EEG was narrow-band-pass filtered in over-
10 lapping ranges (2-6 Hz, 4-8 Hz...), from which zero-crossings were detected. Then, in
11 the broadband filtered data (0.5-40 Hz), peaks were identified between the zero-
12 crossings, and a cycle was considered an oscillation from positive-to-positive peak. A
13 minimum number of consecutive cycles must meet a set of criteria (monotonicity, peri-
14 od consistency, amplitude consistency, shape consistency, etc.) for this to be consid-
15 ered a burst. Importantly, amplitude itself is never used as a threshold, as this would
16 create a greater dependency between amplitude and density (such that a decrease in
17 an amplitude threshold would result in an automatic increase in density).

18 Three sets of criteria were used. The first aimed to detect bursts relying on many low-
19 threshold criteria (frequency in range of narrowband filter; PeriodConsistency = .5; Am-
20 plitudeConsistency = .4; FlankConsistency = .5; ShapeConsistency = .2; Monotonic-
21 tyInTime = .4; MonotonicityInAmplitude = .4; ReversalRatio = .6; MinCycles = 4). The
22 second had fewer criteria with intermediate thresholds but a higher minimum number
23 of cycles (PeriodConsistency = .6; AmplitudeConsistency = .6; MonotonicityInAmplitude
24 = .6; FlankConsistency = .6; MinCycles = 5). The third set had fewer criteria but stricter
25 monotonicity thresholds (frequency in range of narrowband filter; PeriodConsistency =
26 .7; FlankConsistency = .3; MonotonicityInAmplitude = .9; MinCycles = 3). These criteria
27 were chosen a-priori based on manual tuning of the burst detection on an independent
28 dataset of wake EEG in adults during sleep deprivation.

29 After bursts were detected in each channel separately, they were grouped into clusters
30 when they occurred simultaneously in multiple channels with roughly the same fre-

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1 quency. The frequency of bursts was calculated as the inverse of the average distance
2 between negative peaks (1/period). Bursts for which the shorter one overlapped at least
3 50% of the longer one, and were within 1 Hz of each other, were considered part of the
4 same burst cluster. Bursts identified separately in each channel were used for all the
5 topographies, otherwise burst clusters were used to reduce the effect of burst globality
6 (spread across the scalp) on measures of density.

7 **2.3.1 Oscillation measures**

8 *Oscillation amplitudes* were calculated as the average negative-to-positive peak volt-
9 age difference for all cycles involved in all bursts, with units in microvolts (μ V). *Oscilla-*
10 *tion densities* were calculated as the percentage of the recording occupied by bursts
11 (sum of all the bursts' durations divided by the duration of the recording). This accounts
12 for both the duration and overall quantity of bursts in the recording. The average dura-
13 tion and number of bursts can be affected by the background aperiodic signal which
14 can break up sustained oscillations into smaller bursts (Tal et al., 2020), therefore burst
15 density was preferable. When calculating across multiple channels (e.g. Figure 2), os-
16 cillation density could easily exceed 100%, as burst clusters in different frequency
17 ranges often co-occur. When combining densities across multiple frequency bands,
18 bursts were pooled rather than averaged (sum of the durations of all the bursts of any
19 frequency, divided by the duration of the recording). In our previous publication (Snipes
20 et al., 2023), we referred to oscillation densities as “quantities”, however this term did
21 not properly account for the normalization in time.

22 **2.3.2 The choice of frequency bands**

23 Only bursts between 2 and 16 Hz were detected. Below 4 Hz very few bursts could be
24 identified, therefore only bursts above 4 Hz were included in the analysis. Bursts over
25 16 Hz could be detected, but with higher false-positive rates, as determined by visual
26 inspection. The choice of cutoff at 16 Hz was done arbitrarily a-priori to capture alpha
27 (8-12 Hz) with generous padding. The division of bands for Figure 6 and Figure 7 was
28 done using conventional bands (Pernet et al., 2020) with 1 Hz gaps to reduce overlap-
29 ping information due to the drift in peak frequencies across individuals and ages: theta
30 (4-7 Hz), alpha (8-11 Hz) and low beta (12-16 Hz). The inclusion of low beta was done a-
31 posteriori based on results observed in Figure 5B.

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1 Many researchers advocate for the use of an individual alpha frequency (IAF) to define
2 frequency ranges, especially when analyzing data across development (Bazanova &
3 Vernon, 2014; Klimesch, 1999; Ostlund et al., 2022; Tröndle et al., 2022). This is done by
4 selecting the peak alpha frequency separately for each individual and defining the band
5 around this peak. The shift in IAF with age makes a strong case for such an approach
6 (Freschl et al., 2022). The main problem with using IAF is that it assumes the largest
7 oscillation will be functionally the same for all participants. Given that our participants
8 displayed large heterogeneity in the number and amplitude of frequency peaks within
9 the 2-16 Hz range (Suppl. Figure 1-1), and the vast majority of our participants were old
10 enough that the peak alpha frequency was larger than 8 Hz (Freschl et al., 2022), we
11 preferred to use fixed bands with gaps. The completely distinct topographies across the
12 three bands for all age groups support this decision for this dataset (Figure 6, Figure 7).

13 **2.4 Spectral power analysis**

14 Spectral power was calculated using MATLAB's *pwelch* function, with 4 s Hanning win-
15 dows and 50% overlap. When average power across channels was calculated, edge
16 channels were excluded (total count: 98). To dissociate periodic and aperiodic spectral
17 power, we used the MATLAB extension of *specparam* (formerly known as FOOOF (Do-
18 noghue et al., 2020)). Spectra were smoothed over 2 Hz, and the aperiodic signal was
19 fitted between 2 and 35 Hz (frequencies sufficiently separated from the 0.5-40 Hz filter
20 range of the preprocessed data). Otherwise, the default settings were used (peak width:
21 [0.5 12], max number of peaks: inf; minimum peak height: 0; peak threshold: 2; aperi-
22 odic mode: fixed).

23 **2.4.1 Spectral and aperiodic measures**

24 Power was calculated by averaging the log-transformed power values between 4 and 16
25 Hz. Aperiodic offsets were provided by *specparam* as the log power value at 1 Hz of the
26 aperiodic signal, and exponents as the x value in the $1/f^x$ model that describes the
27 steepness of the aperiodic signal. The values are such that positive exponents refer to a
28 downward descending aperiodic signal, and the larger the value the steeper the de-
29 scence. Periodic power was calculated as the log-transformed power from which the
30 aperiodic signal was subtracted. Fitting model parameters *r-squared* and *mean abso-*

Methods

1 *lute error* (MAE) were similarly analyzed to evaluate whether the model fit could ac-
2 count for the results (Ostlund et al., 2022), with the results provided in Suppl. Figure 2-
3 1. Overall, the specparam model fit was very good, with average MAE of 0.028 (inter-
4 quartile range: 0.019, 0.035) and r-squared values of 0.998 (0.997, 0.999).

5 **2.5 Sleep EEG**

6 A subset of wake data was compared to previously analyzed sleep data (Jaramillo et al.,
7 2020), comprising the *sleep-wake dataset*. This included only neurotypical participants
8 from Dataset2016 and Dataset2017, resulting in 44 participants, 50% female, with
9 mean age of 16.3 (8.1-24.8). Only one night per participant (experimental baseline) was
10 included. The corresponding wake data was the auditory oddball for Dataset2016 (>18-
11 year-olds), and the TAP Alertness task for Dataset2017 (<18-year-olds). The channel-
12 averaged data is provided in Suppl. Data 2.

13 EEG sleep data was recorded at 500 Hz with Cz reference. Sleep stages were scored
14 according to standard AASM scoring guidelines (Iber, 2007). Epochs containing arte-
15 facts were rejected after visual inspection using a semi-automatic approach (Huber et
16 al., 2000). Channels with poor signal quality were removed and interpolated, and outer
17 edge channels were removed (total count: 98).

18 Traditionally, sleep pressure is quantified with average spectral power (“slow wave ac-
19 tivity” is power between 0.5-4 Hz). However, directly measuring individual slow wave
20 parameters is considered a more precise marker of sleep pressure and synaptic plas-
21 ticity (Esser et al., 2007; Riedner et al., 2007; Vyazovskiy et al., 2009). This is why both
22 here and in our previous study (Jaramillo et al., 2020) we analyze sleep data focusing on
23 slow wave amplitudes and especially slopes.

24 Slow wave detection was performed similarly to Riedner et al. (2007). The EEG was
25 bandpass filtered from 0.5 to 4 Hz (stopband 0.1 and 10 Hz, Chebyshev Type II filter)
26 and rereferenced to the average. Negative deflections between zero-crossings were
27 identified as slow waves if they were separated by 0.25-1 s. *Slow wave amplitude* con-
28 sisted of the most negative amplitude between zero-crossings of the signal. The de-
29 scending *slow wave slope* was the amplitude of the negative peak divided by the time
30 from the positive-to-negative zero-crossing to the negative peak. The negative peak was

Methods

1 used because this corresponded to silent periods of neuronal spiking, and the de-
2 scending slope is thought to reflect the synchronization of this silent period across neu-
3 rons (Nir et al., 2011; Vyazovskiy et al., 2009).

4 Slow waves were detected in the first and last hour of artifact-free NREM sleep. Given
5 that slopes are directly calculated from amplitudes, slow waves from the first to last
6 hour were first matched by amplitude (see Jaramillo et al. (2020) for details), and only
7 the slopes of matched slow waves were included in the analysis (~60% of total waves).
8 Thus, the decrease in slow wave slopes across the night is independent from the de-
9 crease in amplitudes.

10 2.6 Statistics

11 Statistics were performed using the MATLAB Statistics and Machine Learning Toolbox.
12 For all analyses, statistical significance was determined for p-values < .05. Given the
13 heterogeneous datasets pooled together for this analysis, we chose to conduct linear
14 mixed effects models to model the relationship between age, sleep, ADHD, and EEG
15 measures. This was done with the function *fitlme*.

16 For each fixed factor of the model, β estimates, t-values, p-values, and degrees of free-
17 dom (df) are reported in the text. β estimates of continuous variables (e.g., age) indicate
18 by how much the EEG measure (e.g. density) changes for each unit of the continuous
19 variable (e.g., 1 year) when all other variables are 0. Similarly, the β estimates of cate-
20 gorical variables (e.g. group) indicate how much the EEG measure changes from that
21 category (e.g., ADHD) to the baseline category (e.g., controls), for all other factors set to
22 0. Lastly, t-values allow a comparison of the magnitude of the effect of each factor
23 when comparing models with different measuring units. To determine the topography of
24 the effects, we plotted β estimates and their associated statistical significance, cor-
25 rected for multiple comparisons across channels using false discovery rates (FDR;
26 (Benjamini & Yekutieli, 2001)).

27 We ran models with fixed factors Task, Time, Age, Group, and Sex, the interaction be-
28 tween Time and Age, and nested mixed factors Participant and Session. *Task* compared
29 the levels *Oddball* vs. *go/no-go*, *alertness*, and *fixation*. *Time* compared the time of re-
30 cording; evening vs. morning for wake, and first vs. last hour of NREM sleep. *Group*

Results

1 compared neurotypical participants vs. those with ADHD. Sex compared females vs.
2 males. Depending on the analysis and subset of the data, different factors were includ-
3 ed or not, and so the exact model is specified before each analysis and in each figure
4 caption.

5 As a simpler analysis and sanity check, we conducted Pearson's correlations between
6 age and each wake measure, including only auditory oddball recordings, averaging ses-
7 sions (Figure 2). In each figure, r values are provided as effect sizes. Pearson's corre-
8 lations were also conducted between all measures in the sleep-wake dataset (Suppl.
9 Figure 2-2).

10 3 RESULTS

11 3.1 Effects of age, sleep, sex, and ADHD on wake EEG measures

12 The following linear mixed effects model was applied to each wake EEG measure, aver-
13 aged across channels: Measure ~ Task + Time * Age + Group + Sex +
14 (1|Participant) + (1|Participant: Session). The full outputs of the model are provided
15 in Suppl. Data 3.

16 Oscillation amplitudes significantly decreased with age ($\beta = -0.783$, $t = -11.33$, $p < .001$,
17 $df = 1234$), significantly decreased the morning following sleep ($\beta = -3.977$, $t = -15.90$, p
18 $< .001$, $df = 1234$), with a significant positive interaction ($\beta = 0.143$, $t = 7.74$, $p < .001$, $df =$
19 1234), such that amplitudes decreased less overnight with increasing age (Figure 2,
20 bottom row). Amplitudes were significantly lower in males than females ($\beta = -1.476$, $t = -$
21 2.38 , $p = .017$, $df = 1234$), and were not significantly different in participants with ADHD
22 ($\beta = -0.574$, $t = -0.89$, $p = .373$, $df = 1234$). As can be seen in Figure 2, the relationship
23 between age and amplitudes was quite robust, both as absolute values ($r_{eve} = -.66$, $r_{mor} =$
24 $-.63$) and overnight changes ($r = .56$). Overall, amplitudes changed in the expected di-
25 rections for both development and sleep pressure, except for the lack of an effect of
26 ADHD.

27 Oscillation densities significantly decreased with age ($\beta = -3.540$, $t = -2.11$, $p = .035$, $df =$
28 1234), and the morning after sleep ($\beta = -77.506$, $t = -10.39$, $p < .001$, $df = 1234$), with a
29 significant positive interaction between age and time of recording ($\beta = 4.878$, $t = 8.85$, p

Results

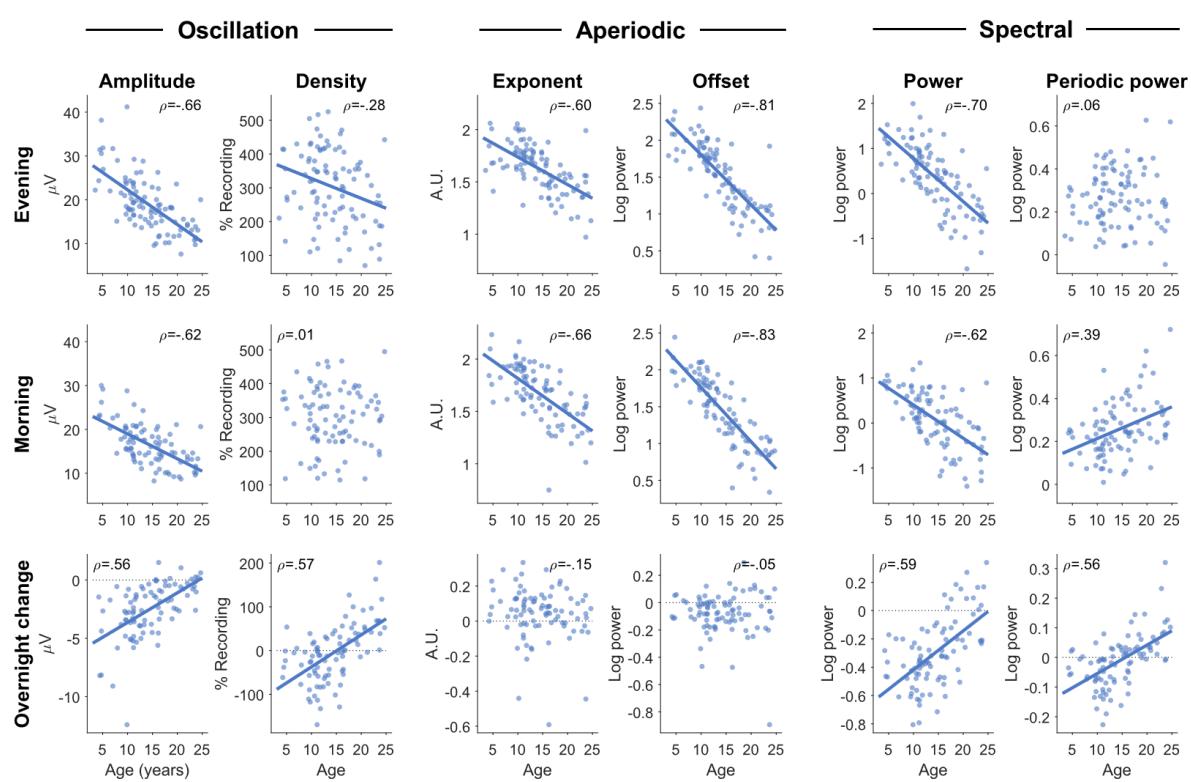
1 $< .001$, $df = 1234$). Unlike amplitudes, the correlation between age and density was
2 weak ($r_{eve} = -.28$, $r_{mor} = .01$). Instead, the correlation between age and overnight change
3 in density was quite strong ($r = .57$), such that oscillation densities decreased overnight
4 in children under 15 and increased in young adults (Figure 2). There was no effect of
5 ADHD ($\eta^2 = 0.154$, $t = -0.01$, $p = .992$, $df = 1234$) or sex ($\beta = -16.446$, $t = -1.10$, $p = .271$,
6 $df = 1234$). Overall, oscillation densities behaved independently from amplitudes, es-
7 pecially in the direction of overnight changes in adolescents and adults.

8 Aperiodic exponents became significantly shallower with age ($\beta = -0.027$, $t = -9.23$, $p <$
9 $.001$, $df = 1234$) but significantly steeper overnight ($\beta = 0.099$, $t = 5.51$, $p < .001$, $df =$
10 1234), with a trending negative interaction between time of recording and age ($\beta = -$
11 0.003 , $t = -1.91$, $p = .056$, $df = 1234$). The increased overnight steepness was driven pri-
12 marily by a decrease in higher frequency power (Suppl. Figure 5-1), whereas decreasing
13 steepness with age is driven by decreases in low frequency power (Favaro et al., 2023).
14 The correlations between exponents and age were as robust as for oscillation ampli-
15 tudes ($r_{eve} = -.60$, $r_{mor} = -.66$), but the correlation with overnight change was weak ($r = -$
16 $.15$, not significant). There was no significant effect of ADHD ($\beta = 0.028$, $t = 1.07$, $p =$
17 $.284$, $df = 1234$) or sex ($\beta = 0.041$, $t = 1.63$, $p = .102$, $df = 1234$). This means that aperiodic
18 exponents also change independently from oscillation amplitudes, and do not reflect
19 the direction of changes expected for sleep pressure.

20 Aperiodic offsets also significantly decreased with age ($\beta = -0.067$, $t = -16.59$, $p < .001$,
21 $df = 1234$), but with no significant effect of time ($\beta = 0.029$, $t = 1.65$, $p = .100$, $df = 1234$),
22 and a significant negative interaction ($\beta = -0.004$, $t = -3.11$, $p = .002$, $df = 1234$), such
23 that offsets decreased more overnight with age. The correlations between age and off-
24 sets were the strongest of all measures ($r_{eve} = -.81$, $r_{mor} = -.83$), however, the correlation
25 between age and overnight change in offset was negligible ($r = -.05$). Again, there was no
26 effect of ADHD ($\beta = 0.021$, $t = 0.55$, $p = .579$, $df = 1234$) or sex ($\beta = -0.003$, $t = -0.07$, $p =$
27 $.944$, $df = 1234$). Overall, offsets correlated with age in the same direction as ampli-
28 tudes, changed overnight in the same direction, but the overnight change was not larger
29 in children compared with adults, counter to what would be expected for sleep pres-
30 sure.

Results

1 Curiously, fitting parameters of the specparam algorithm showed both significant age
2 and time of recording effects, with the model fit improving in the morning and worsen-
3 ing with age (Suppl. Figure 2-1). Therefore, differences in fit quality may partially con-
4 tribute to the effects observed for aperiodic measures. However, the changes in model
5 fitting go in the opposite direction one would expect of data quality (i.e. worse in the
6 morning and better in adults). This suggests that some other aspect of the EEG signal
7 that changes with age and time can affect the fit quality of the specparam algorithm.



8
9 **Figure 2: Wake EEG measures correlated with age.** Only auditory oddball recordings were included, pooling both
10 neurotypical and ADHD participants. Each dot represents the value for a single participant. For participants with
11 multiple sessions, values across sessions were first averaged. Pearson's correlations were done for each figure, with
12 rho values provided in the corner. If the p-value was less than .05, a correlation line was drawn (without correcting for
13 multiple comparisons). Amplitudes and densities of oscillations were obtained from burst clusters, pooling all fre-
14 quencies (4-16 Hz). Exponent values are such that larger values indicate a steeper aperiodic signal; units are "arbitr-
15 ary" (a.u.), in that exponents are scale-free measures. Power spectra were calculated for each channel, then aver-
16 aged across all channels, excluding the outermost ring of channels. Power and periodic power were then calculated
17 by averaging values from 4 to 16 Hz. The same figure for the specparam fitting estimates is provided as Suppl. Figure
18 2-1. The correlations between each measure with the other is provided in Suppl. Figure 2-2.

19 In summary, of the four EEG measures, only amplitudes followed the same trajectories
20 expected for both development and sleep pressure. The absolute values of all four
21 measures had a negative correlation with age, and differed primarily in the overnight
22 response and the relationship between age and overnight response. Oscillation densi-

Results

ties in particular showed a strong effect of age on overnight changes, reversing direction between childhood and adolescence. No measure showed any relationship with ADHD, and only amplitudes were affected by sex. Therefore, in later analyses we did not include these factors, and pooled patients and controls for greater statistical power.

3.1.1 Comparison of oscillation and aperiodic measures to spectral measures

To quantify the extent to which wake spectral measures were influenced by oscillation and aperiodic measures, we directly correlated each measure to the other in the sleep-wake dataset (Suppl. Figure 2-2), and then controlled for time of the recording, task, and age using linear mixed effects models in the complete wake dataset: Measure1 ~ Measure2 + Time * Age + Task + (1|Participant) + (1|Participant: Session) (Suppl. Table 2-1).

Without correcting for anything, all measures significantly correlated with each other, except periodic power which did not correlate with either aperiodic exponents or offsets. Power was most related to amplitudes, also in the mixed effects model (correlation $r = .93$, mixed effects t -value = 39.3). Power was also significantly related to densities ($r = .82$, $t = 28.2$) and offsets ($r = .80$, $t = 17.5$), but less so with exponents ($r = .54$, $t = 4.7$). Periodic power was most correlated with densities ($r = .80$, $t = 46.9$) and somewhat with amplitudes ($r = .53$, $t = 20.6$).

3.1.2 Comparisons between wake and sleep EEG measures

Using the sleep-wake dataset, we directly compared wake measures with sleep slow wave amplitudes and slopes, first through simple correlations (Suppl. Figure 2-2) and then with the linear mixed effects model: SleepMeasure ~ WakeMeasure + Time * Age + Task + (1|Participant). The full models are provided in Suppl. Data 4.

All wake measures were significantly correlated with both slow wave amplitudes (wake amplitude $r = .61$; density $r = .27$; exponent $r = .46$; offset $r = .66$) and slopes (amplitude $r = .70$; density $r = .38$; exponent $r = .60$; offset $r = .80$). When controlling for the time of the recording, task, and age, wake amplitudes were significantly related to both slow wave slopes ($\beta = 4.21$, $t = 2.8$, $p = .006$, $df = 82$) as well as slow wave amplitudes ($\beta = 0.78$, $t = 2.7$, $p = .007$, $df = 82$). The same was true for wake power (slow wave slopes: $\beta = 26.36$, $t = 2.3$, $p = .023$, $df = 82$; slow wave amplitudes: 5.84 , $t = 2.6$, $p = .011$, $df = 82$).

Results

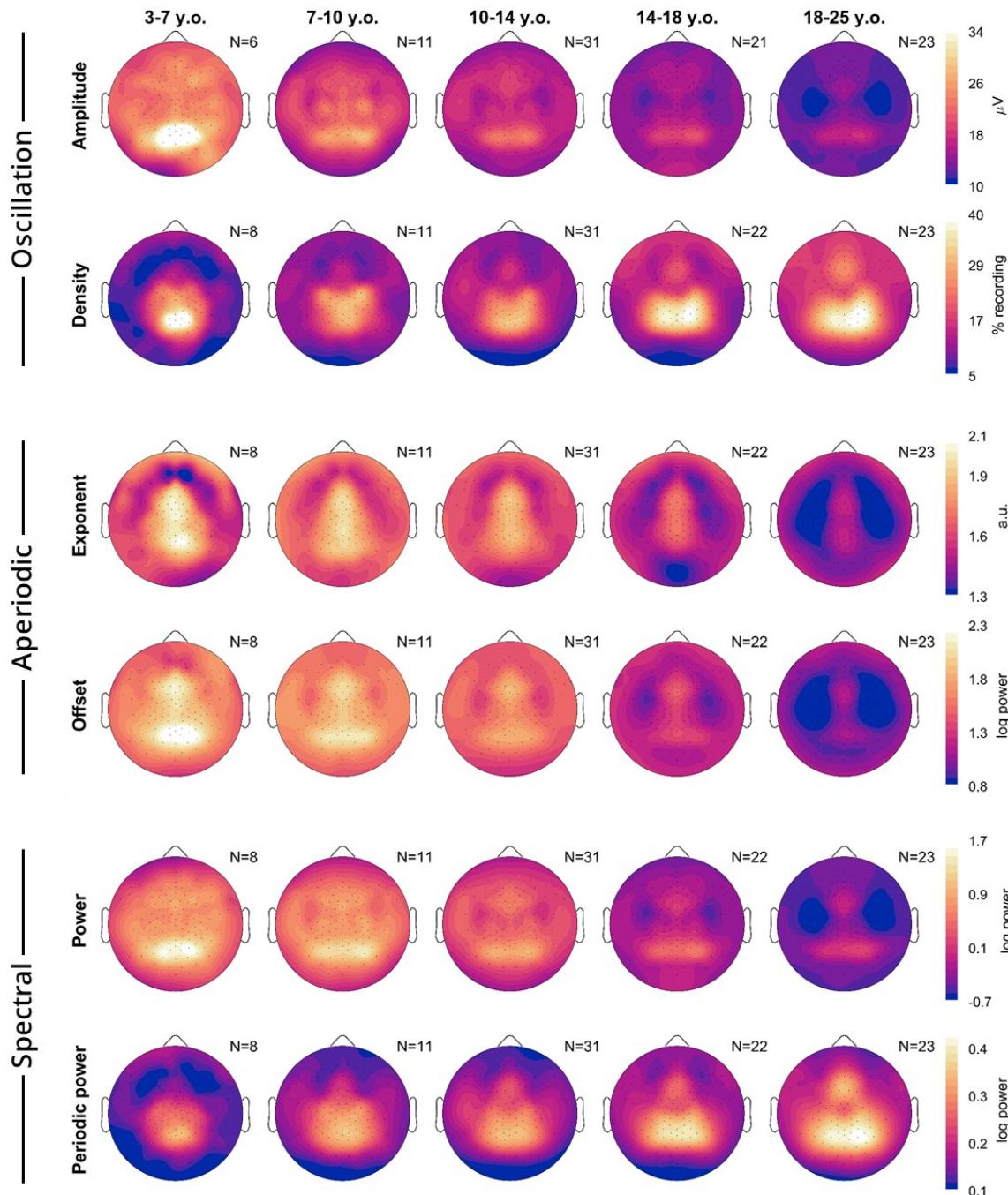
1 While aperiodic offsets had shown the strongest correlations to sleep measures, with
2 the mixed effects model they were only significantly related to amplitudes ($\beta = 14.69$, $t =$
3 2.8 , $p = .006$, $df = 82$) but not slopes ($\beta = 40.26$, $t = 1.6$, $p = .111$, $df = 82$). No other wake
4 measure was related to either slopes (density: $\beta = 0.05$, $t = 1.1$, $p = .289$, $df = 82$; expo-
5 nent: $\beta = 7.78$, $t = 0.3$, $p = .785$, $df = 82$; periodic power: $\beta = 44.92$, $t = 1.2$, $p = .247$, $df =$
6 82) or amplitudes (density: $\beta = 0.02$, $t = 1.2$, $p = .235$, $df = 82$; exponent: $\beta = 5.99$, $t = 0.8$,
7 $p = .453$, $df = 82$; periodic power: $\beta = 7.69$, $t = 0.8$, $p = .412$, $df = 82$).

8 Overall, wake amplitudes were most related to sleep slow waves, with significant rela-
9 tionships to both amplitudes and slopes. This association was reflected in wake power.
10 Wake offsets were also significantly correlated to sleep measures, however the associa-
11 tion between offsets and sleep slopes did not survive the correction for age and time
12 in the mixed effects model.

13 **3.2 Topography of wake EEG measures by age**

14 Figure 3 provides the average topographical maps of each measure for five age bins,
15 averaging (or pooling for densities) all frequencies from 4-16 Hz, from the oddball task.
16 Amplitudes, densities, exponents, and offsets all showed unique topographies from
17 each other. Across ages, for each measure there were primarily changes in magnitude
18 more so than major regional differences. However, oscillation amplitudes in the young-
19 est cohort began as a single midline occipital spot, which spread bilaterally in the 7–10-
20 year-olds. Prominent central bilateral peaks also appeared in the 7–10-year-olds. Oscil-
21 lation densities similarly started as a single midline occipital spot, but these became
22 more lateral-parietal in the 14–18-year-olds. Like amplitudes, two small bilateral cen-
23 tral peaks emerged in the densities of 7–10-year-olds, which merged with the primary
24 occipital-parietal cluster in the 14–18-year-olds. Furthermore, a frontal peak gradually
25 emerged with age. Exponents were steepest in midline channels, whereas offsets
26 showed both a frontal midline and occipital peak. As with the correlations between
27 measures, power topographies most resemble amplitudes, and periodic power resem-
28 bles densities.

Results



1
2 **Figure 3: Topography averages of wake EEG measures.** Each plot is a schematic of the EEG viewed from above,
3 with the nose on top. Lighter colors indicate greater magnitude over a given location for that measure (rows). Only
4 neurotypical participants and oddball recordings were included, and participants were grouped into age bins (col-
5 umns). Multiple oddball recordings from different sessions and times of day were first averaged for each participant.
6 The number of participants included is indicated in the top right corner of each plot. Acronyms: y.o., years-old; a.u.,
7 arbitrary units.

8 To determine the topography of overnight changes in EEG measures, we performed lin-
9 ear mixed effects models for each channel, with the model $\text{Measure} \sim \text{Time} + \text{Task} +$
10 $(1|\text{Participant}) + (1|\text{Participant}:\text{Session})$, dividing participants into 4 age bins (the

Results

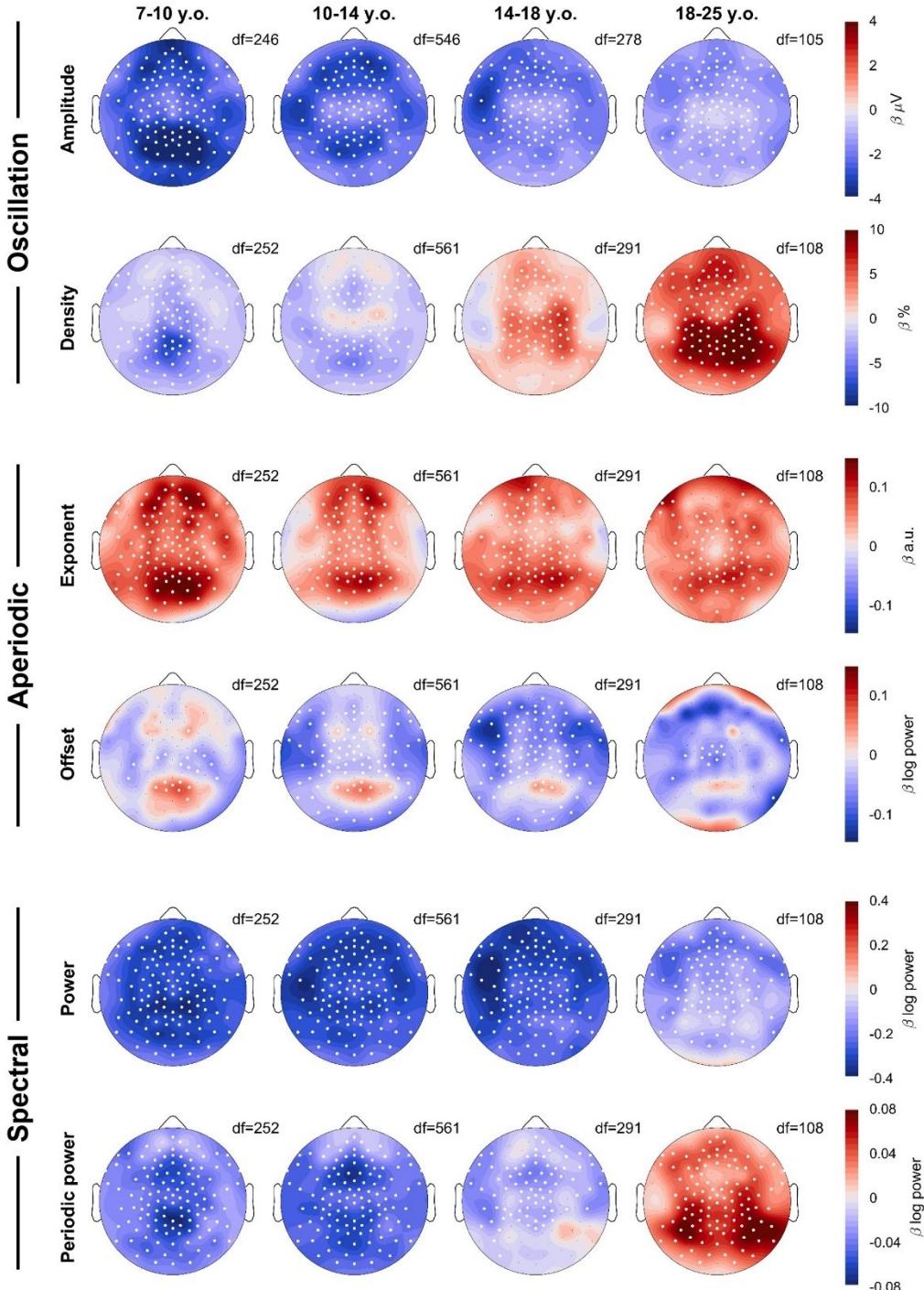
1 youngest 3-7 were excluded as they were too few, with too few recordings). Figure 4
2 plots the β estimates for the effect of Time.

3 Amplitudes showed widespread overnight decreases across all age groups, however
4 the decrease was largest in occipital channels for the youngest group, and slightly more
5 fronto-temporal in young adults. The overnight density topographies resembled the av-
6 erage density topographies from Figure 3, in terms of location of the effects. The young-
7 est group showed the largest overnight decrease in the same midline-occipital spot
8 where there were the most oscillations (Figure 3), and adults showed the largest in-
9 crease in the same bilateral occipital-parietal areas where they had the largest densi-
10 ties.

11 The overnight increase in the steepness of exponents was widespread, but peaked in an
12 occipital spot in all age groups, with additional bilateral frontal spots in <18-year-olds.
13 These topographies do not correspond to the average topography of exponents from
14 Figure 3. Offsets revealed widespread decreases, with localized increases in the same
15 occipital locations for which exponents increased the most. This suggests that aperiod-
16 ic offsets generally decrease, although the increase in exponent steepness contrasts
17 this effect.

18 Power and periodic power again showed similarities to amplitudes and densities re-
19 spectively, however while densities increased in the 14–18-year-olds, periodic power
20 decreased. Likewise, the overnight increase in periodic power for >18-year-olds was
21 more occipital and lateral than the increase in densities, and the larger decrease in
22 amplitudes in occipital regions of 7–10-year-olds was less evident in power than for
23 amplitudes.

Results



1

2 **Figure 4: Topographies of wake EEG overnight changes.** A linear mixed effects model was run for each measure,
3 each age group, and each channel: Measure ~ Time + Task + (1|Participant) + (1|Participant:Session). Color reflects β
4 estimates for the fixed effect Time, such that red indicates an overnight increase in that measure. The factor Task
5 was not included for the 18-25 y.o. group, as these participants only performed oddballs. White dots indicate
6 channels for which the β estimate was statistically significant, corrected for multiple comparisons with FDR (false discov-
7 ery rate). Black dots indicate remaining channels. Data includes both patients and neurotypical controls. Degrees of
8 freedom (df) are provided for each plot. For each column, the sample sizes were N = 29, 67, 36, & 23.

Results

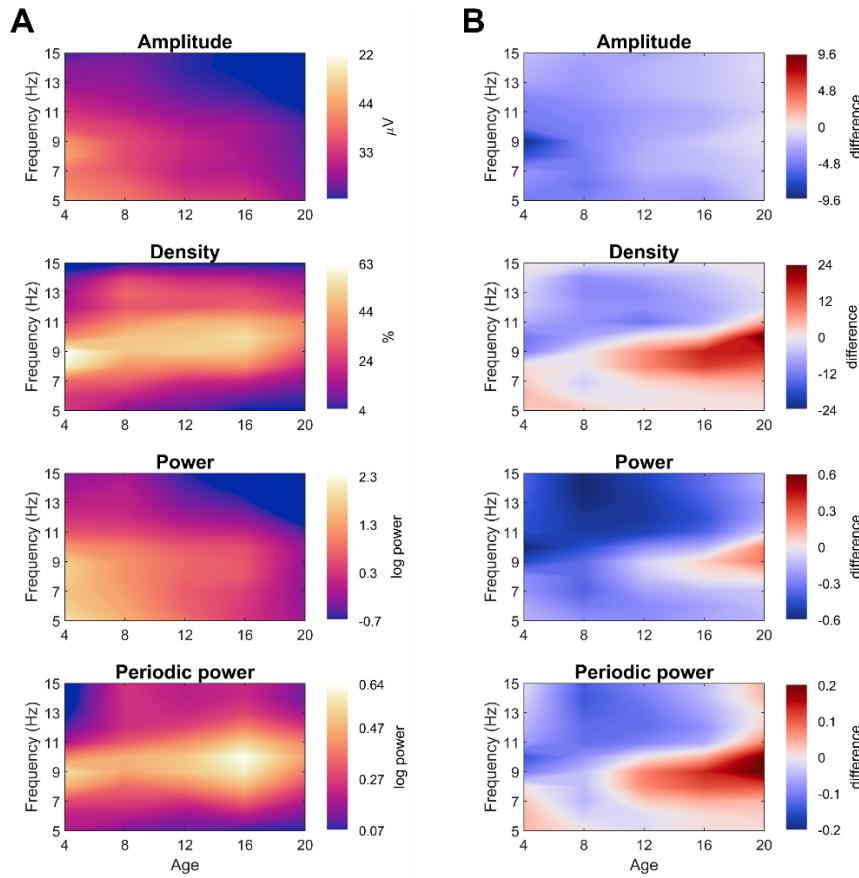
1 **3.3 Frequency-specific wake EEG changes with age and sleep pres-** 2 **sure**

3 In the previous analyses, we had pooled all frequencies between 4 and 16 Hz. Subse-
4 quently, we explored how oscillation and spectral EEG measures changed for each fre-
5 quency, this time averaging channels. Average evening values for each age and fre-
6 quency are plotted in Figure 5A, and the overnight differences are plotted in Figure 5B.

7 Average oscillation amplitudes and spectral power showed gradual gradients, with
8 highest values in the youngest participants and lowest frequencies, and lowest values
9 in the oldest participants and highest frequencies. Like average amplitudes, overnight
10 changes in amplitude showed largely gradual decreases with age and frequency.

11 Average density and periodic power instead had distinct peak values between 8 and 11
12 Hz, with the peak shifting upwards with age, a well-known property of alpha oscillations
13 during development (Freschl et al., 2022; Smith, 1938; Tröndle et al., 2022). Overnight
14 densities (and periodic power) showed decreases in higher frequencies (>11 Hz, i.e. low
15 beta) and increases in alpha. These increases only began between 8-10 years of age,
16 they were strongest in adults, and the range shifted to higher frequencies with age.

Results



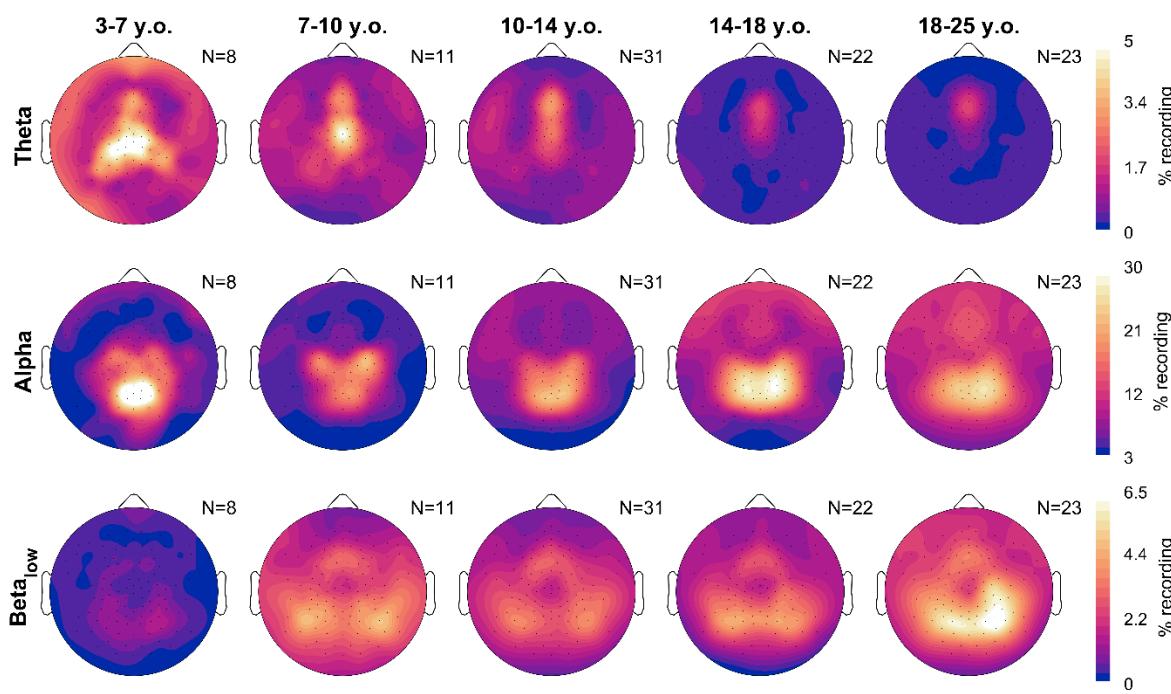
1

2 **Figure 5: Average spectrograms across ages.** From the oddball task, pooling controls and ADHD participants.
3 Average spectra from the other tasks are provided in Suppl. Figure 5-1. **A:** Average values, such that lighter colors
4 indicate greater magnitude for a given frequency and age. For spectra extending to older ages and sleep stages, see
5 Sun et al. (2023). **B:** Difference values between morning and evening recordings, such that red indicate a greater
6 magnitude in the morning. The measurement unit of each figure is the same as that of A.

7 Given the dissociation between alpha and beta for density, we explored the topographic
8 changes in density, split by both age and frequency. In Figure 6, we plot average values.
9 Theta oscillations were the overall rarest. They were most prevalent in the youngest
10 children, and with age, the peak in theta density gradually shifted upward and de-
11 creased in magnitude. Alpha density instead started as a midline occipital cluster in the
12 3–7-year-olds. Alpha densities in the occipital spot decreased in the 7–10 cohort, with
13 bilateral central spots instead becoming more pronounced. With age, these three
14 peaks morphed into a continuous occipital-parietal cluster, while in the remaining
15 channels overall density of alpha increased. Lastly, low beta oscillations showed yet
16 another topography. They were almost completely absent in the youngest group, and
17 started to appear in the 7–10-year-olds as lateral occipital peaks and a frontal midline

Results

1 spot. Gradually, the lateral peaks converge towards the midline, and in adults became
2 partially overlapping with alpha, on average right-lateralized.



3
4 **Figure 6: Average topographies of oscillation densities, split by frequency band.** Recordings are the same as in
5 Figure 3. Theta is 4-7 Hz, alpha is 8-11 Hz, and low beta is 12-16 Hz. Each band has a different color scale range.

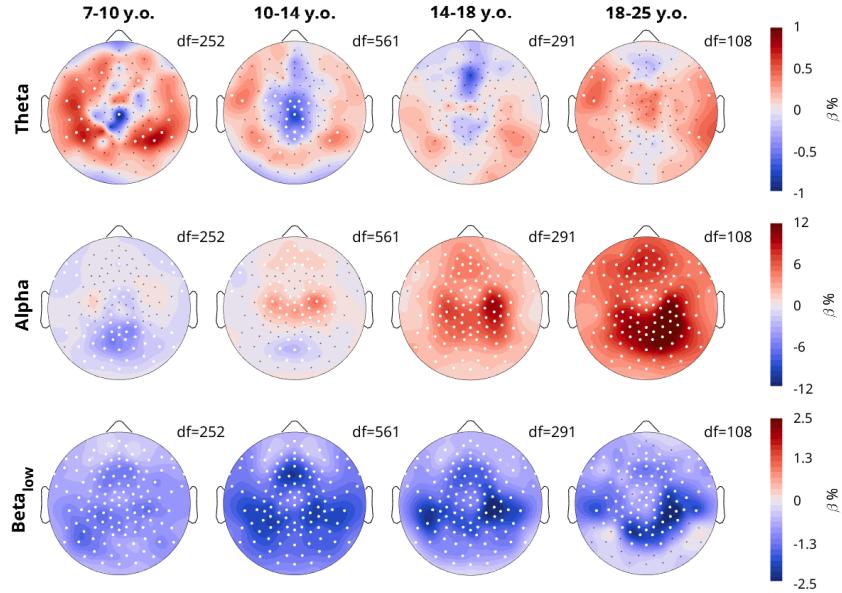
6 The overnight changes in density split by frequency are in Figure 7, based on the model
7 Density ~ Time + Task + (1|Participant) + (1|Participant: Session). Like in Figure 4,
8 these reflect the β estimates for the fixed effect of Time.

9 The overnight changes in theta density were small (around 1%), however quite variable
10 by region and age. In the 7–10-year-olds, theta density generally increased overnight
11 except in a central spot, exactly where the largest theta densities were seen in Figure 6,
12 which instead decreased. This continued for the 10–14-year-olds. In the 14–18-year-
13 olds, there were no significant effects, however the midline theta spot, now more
14 frontal, showed a decrease. In adults, only some scattered theta increases were ob-
15 served.

16 For alpha, the main occipital spot in the 7–10-year-olds decreased overnight. Already in
17 the 10–14-year-olds a bilateral central alpha rhythm started to increase overnight, with
18 still some slight decreases in the occipital spot. The overnight increases spread to the
19 entire scalp in adolescents and adults, peaking in occipital parietal areas, especially

Results

1 right lateralized. For low beta, across all ages there were decreases, with the peak shift-
2 ing across age bins.



3

4 **Figure 7: Topographies of overnight density changes, split by frequency band.** Participants and plot information
5 are the same as in Figure 4, with color indicating the β estimates for the linear mixed effects model, such that red
6 indicates an overnight increase in density. The model was $\text{Density} \sim \text{Time} + \text{Task} + (1|\text{Participant}) +$
7 $(1|\text{Participant}:\text{Session})$.

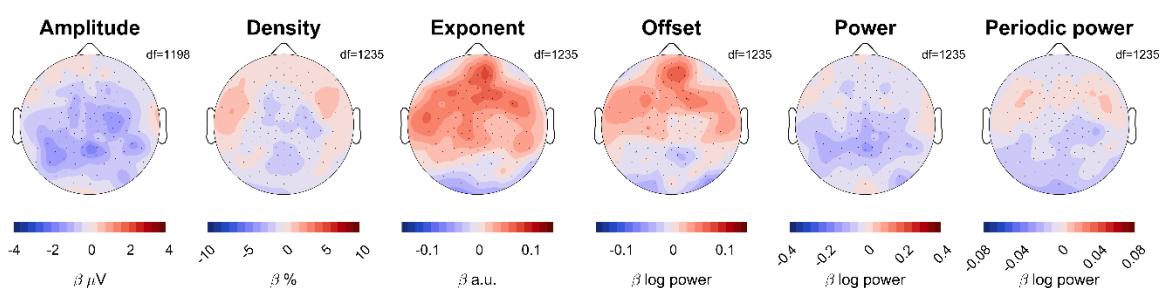
8 **3.4 Effect of ADHD on the wake EEG topography**

9 In our initial mixed effects models pooling channels and frequencies, we found no sig-
10 nificant effects for ADHD, which is why in subsequent analyses and figures we no long-
11 er included a Group effect. However, we nevertheless conducted mixed effects models
12 to determine the effect of ADHD for each channel: $\text{Measure} \sim \text{Task} + \text{Time} * \text{Age} +$
13 $\text{Group} + (1|\text{Participant}) + (1|\text{Participant}:\text{Session})$. We found no significant effects
14 when correcting for multiple comparisons (Figure 8). However, amplitudes were on av-
15 erage lower in participants with ADHD and frontal exponents were steeper compared to
16 controls.

17

18

Discussion



1

2 **Figure 8: Effects of ADHD on EEG measures.** Red indicates larger values in patients compared to controls. The
3 scale for each topography is the same as for Figure 4. The model was Measure ~ Time*Age + Task + Group +
4 (1|Participant) + (1|Participant:Session). White dots would have indicated statistically significant channels, following
5 FDR correction for multiple comparisons.

6 4 DISCUSSION

7 In this study, we compared four distinct wake EEG measures and their relationship to
8 development, sleep pressure, and ADHD. Specifically, we hypothesized that oscillation
9 amplitudes would behave like sleep slow waves, because both measures likely reflect
10 neuronal synchronization due to synaptic density and plasticity. Our predictions were
11 met on all accounts except for the sensitivity of wake amplitudes to ADHD. Of the four
12 measures, only amplitudes decreased overnight in all ages and the overnight decrease
13 was largest in younger children (Figure 2). Wake amplitudes were significantly correlat-
14 ed to sleep slow wave measures, also when correcting for age and participant. Aperiod-
15 ic offsets showed similar development and overnight changes, as well as strong corre-
16 lations to sleep measures, but they did not display larger overnight decreases in young-
17 er children, a key indicator of higher neural plasticity.

18 While oscillation amplitudes were the only wake measure that followed all the patterns
19 expected for sleep pressure, every EEG measure reflected development. Average am-
20 plitudes, exponents, and offsets were all strongly anticorrelated with age, with offsets
21 showing the strongest relationship (Figure 2). The correlation between average densi-
22 ties and age was weak, but we found distinctive regional patterns across age groups
23 (Figure 6). Overnight changes in both oscillation amplitudes and density were robustly
24 correlated with age (Figure 2, Figure 7), whereas overnight changes in exponents and
25 offsets were not as affected by age, as evidenced by the low correlations in Figure 2.

Discussion

1 These results are summarized in Figure 9. Finally, no measure showed significant ef-
2 fects of ADHD (Figure 8).

		Sleep pressure	Development
Oscillation	Amplitude	n.a.	✓
	Overnight amplitude	✓	✓
	Density	n.a.	✓
	Overnight density	✗	✓
Aperiodic	Exponent	n.a.	✓
	Overnight exponent	✗	n.s.
	Offset	n.a.	✓
	Overnight offset	mixed	mixed

3

4 **Figure 9: Summary of which measure reflects sleep pressure or development.** Sleep pressure was determined by
5 whether a measure decreased overnight and whether the decrease was larger in younger children. Development was
6 determined by whether there were strong effects of age. Overnight offsets had “mixed” results across analyses.
7 Acronyms: n.a., not applicable; n.s., not significant.

8 4.1 Oscillation amplitudes

9 Like for slow waves during sleep, the decrease in wake oscillation amplitudes with age
10 could be explained by decreasing synaptic density in the cortex across adolescence
11 (Huttenlocher, 1979). The overnight decrease in wake amplitudes could be explained by
12 sleep’s role in reducing net synaptic strength (Cirelli & Tononi, 2022; Tononi & Cirelli,
13 2014), and such plastic changes are more pronounced in children than adults (Jaramil-
14 lo et al., 2020). The larger decrease in occipital regions in children compared to adults
15 may be because primary sensory and motor areas obtain peak cortical thickness earlier
16 in children, followed by adjacent secondary areas and finally frontal association areas
17 (Shaw et al., 2008), resulting in an overall posterior-anterior maturation trajectory (Kurth
18 et al., 2010). Therefore, younger children show larger overnight decreases in amplitudes
19 in occipital areas because these areas undergo higher plastic changes at that matura-
20 tional stage. Finally, even the significant sex difference was comparable to sleep slow
21 wave activity (Mourtazaev et al., 1995), with wake oscillation amplitudes higher in fe-
22 males than males (although the effect was relatively small).

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1 Higher slow wave activity in females has been hypothesized to reflect their smaller
2 heads and thinner skulls (Dijk et al., 1989). It is then also possible that head and skull
3 size affect the age-related changes we observed for wake amplitudes. However, Scha-
4 woronkow and Voytek (2021) did not find any changes in wake oscillation amplitudes
5 from 1-7 month-old infants, despite this period corresponding to the fastest growth in
6 head circumference (Roche et al., 1986). Therefore, it is unlikely that the more modest
7 increases in head size from childhood to adolescence can entirely explain the large de-
8 crease in amplitudes we observed here.

9 While wake oscillation amplitudes behave largely like sleep slow waves, the effects are
10 reduced. In adults the overnight decrease in wake amplitudes was near 0 μ V (Figure 2),
11 which is not the case for either slow wave slopes or amplitudes during sleep (Jaramillo
12 et al., 2020; Riedner et al., 2007). One possible explanation for this is that the evening
13 wake recordings fell within the wake maintenance zone. This is a circadian time window
14 just before bedtime characterized by increased alertness (Shekleton et al., 2013;
15 Strogatz et al., 1987). We had found in our previous study that oscillation amplitudes
16 are significantly reduced in this window, counteracting the otherwise monotonic
17 buildup in amplitudes that occurred throughout the day (Snipes et al., 2023). In adults
18 the contrasting effect of the wake maintenance zone may be sufficient to equalize
19 evening and morning amplitudes. Overall, wake oscillation amplitudes may reflect the
20 same information as sleep slow waves but may be more affected by circadian or other
21 factors. Therefore, wake amplitudes may be affected by the same underlying neuronal
22 changes as sleep slow wave activity, but they are not sufficiently specific nor sensitive
23 to replace this gold standard measure of sleep pressure.

24 **4.2 Oscillation densities**

25 As could be expected, the densities of oscillations were the most complex EEG meas-
26 ure, with effects differing depending on age, time, topography, and oscillation frequen-
27 cy.

28 Theta oscillations were most prevalent in early childhood, and decreased progressively
29 with age, supporting previous results measuring relative theta power (Somsen et al.,
30 1997). We further found that the peak in theta densities steadily drifted more frontally

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1 across childhood and adolescence. This frontal theta in adults is known to originate
2 from the midline prefrontal cortex, and to be anti-correlated to the default mode net-
3 work (Ishii et al., 2014; Michels et al., 2010; Scheeringa et al., 2008). Therefore, this drift
4 in theta may reflect the steady maturation of both frontal cortices and the default mode
5 network (Fan et al., 2021).

6 There is still unresolved contradictory evidence on the role of theta in adults (Snipes et
7 al., 2022), without including the question of theta during development. On the one
8 hand, theta is often associated with cognitive effort (Buzsáki, 2005; Cavanagh & Frank,
9 2014; Meyer et al., 2019; Mitchell et al., 2008). On the other, it is also associated with
10 sleepiness (Finelli et al., 2000; Smith, 1938; Snipes et al., 2022) and fatigue (Arnaud et
11 al., 2021; Tran et al., 2020; Wascher et al., 2014). A possible resolution to this paradox
12 is that there are distinct oscillations that originate from different circuits with different
13 functions and just happen to occur at the same frequency. Our results in Figure 7 would
14 support this, as the peak source of theta shows overnight decreases, whereas theta
15 from the rest of the cortex instead shows overnight increases, even as the theta peak
16 drifts more frontally. Alternatively, theta could reflect a general form of “idling rhythm”
17 (Snipes et al., 2022, 2024), originating from disengaged cortical areas, and what chang-
18 es from evening to morning is which circuits tend to idle. Theta as an idling rhythm is
19 supported by simultaneous EEG-fMRI studies that find theta activity anti-correlating
20 with brain metabolism (Scheeringa et al., 2008). This would make theta functionally
21 comparable to alpha (Laufs et al., 2003), differing only by source and frequency.

22 Alpha begins in young childhood as a midline spot (Figure 6). Two lateral central peaks
23 become more defined at 7-10 years of age. These likely reflect sensorimotor mu
24 rhythms which appear when motor activity is absent or even suppressed (Pfurtscheller
25 et al., 2006; Pineda, 2005), and is already present in infants (Berchicci et al., 2011). We
26 find that with age, they become topographically indistinguishable from occipital alpha,
27 at least when recorded during an oddball task. These lateral central peaks are the first
28 to show overnight increases in 10-14 year-olds, while the overnight decrease in the oc-
29 cipital midline spot becomes less prominent. The overnight increase in alpha densities
30 then spreads over bilateral parietal and occipital areas across adolescence and adult-
31 hood. This dissociation between overnight decreases in childhood and increases in

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1 adulthood, as well as the slight differences in topography, could suggest that occipital
2 alpha is in fact qualitatively distinct in children and adults. However, these rhythms
3 were previously considered functionally equivalent because also in infants alpha power
4 increases with eyes closed compared to eyes open (Stroganova et al., 1999). More re-
5 search is needed on the sources of these oscillations.

6 It is also possible that this dissociation in overnight density changes is driven by some
7 other difference with age, such as a longer window of sleep inertia in young children,
8 longer sleep duration, or a shifted circadian rhythm compared to adults. Melatonin in
9 the morning is elevated in children under 10, whereas older children and adolescents
10 have morning melatonin levels comparable with the rest of the day (Attanasio et al.,
11 1985). In adults, alpha power fluctuates with circadian rhythm and is therefore syn-
12 chronized to melatonin levels (Cajochen et al., 2002). Therefore, it is possible that the
13 dissociation of decreasing/increasing alpha densities originates from children and
14 adults being at different phases of their alpha circadian rhythm in the morning. More
15 research is needed into the circadian effects on the EEG across development.

16 Finally, we found overnight decreases in low beta densities across ages. There is ample
17 literature on “beta bursts” from sensorimotor areas (Feingold et al., 2015; Jones et al.,
18 2009; Little et al., 2019; Lundqvist et al., 2024; Shin et al., 2017; Wessel, 2020), however
19 these are likely qualitatively distinct from the beta oscillations we observed. First, the
20 topography of sensorimotor beta rhythms was found to be either bilateral-central in
21 infants or frontal in adults (Rayson et al., 2023), unlike the bilateral occipital topogra-
22 phies in Figure 6. Second, sensorimotor beta was found to occur in “bursts” of power
23 less than 150 ms long (Sherman et al., 2016) and could thus correspond to only 1-3 cy-
24 cles of beta (Jones, 2016; van Ede et al., 2018). Supporting this, lagged coherence de-
25 creases rapidly after 2 cycles (Rayson et al., 2023). We detected bursts that were *at*
26 *least* 3 cycles long, only up to 16 Hz, and therefore always longer than 187 ms. There-
27 fore, the low-beta bursts we captured with cycle-by-cycle analysis are likely distinct
28 from the previously investigated sensorimotor beta activity. It remains an open ques-
29 tion what is the functional role of these low-beta oscillations, and why they may be less
30 common the morning after sleep.

Discussion

1 **4.3 Aperiodic offsets and exponents**

2 Our results on offsets and exponents replicate previous findings: they decrease linearly
3 with age (Cellier et al., 2021; Hill et al., 2022; Tröndle et al., 2022) and originate from
4 broad, primarily midline sources (Favaro et al., 2023). As in Lendner et al. (2023), we
5 found exponents becoming increasingly steeper after sleep, extending this finding
6 across ages. We further found, unusually, that exponents and offsets change in oppo-
7 site directions, with offsets decreasing after a night of sleep. For aperiodic activity
8 measured *during* sleep, both exponents and offsets decrease across the night, and the
9 decrease decreases with age (Horváth et al., 2022). This indicates that changes in ape-
10 riadic activity related to sleep-wake history do not reflect the same information when
11 measured during wake or during sleep. It is possible that during sleep the decrease in
12 exponents reflects the decrease in sleep pressure, whereas during wake the increase in
13 exponents could reflect something like lingering sleep inertia in the morning. Notably,
14 the change in aperiodic exponents with age and sleep depth affect primarily lower fre-
15 quencies (Favaro et al., 2023), whereas the overnight wake EEG changes affect primari-
16 ly higher frequencies (Suppl. Figure 5-1). This means that the pivot point (the frequency
17 at which the aperiodic signal rotates) of the exponent differs, and this likely has biologi-
18 cal significance. Future studies should, as in Bódizs et al. (2021), specifically identify
19 such pivot points, also to better dissociate changes in offset and exponent.

20 Second to oscillation amplitudes, aperiodic offsets showed the closest resemblance to
21 sleep slow wave activity, decreasing overnight and showing strong correlations with
22 slow wave measures. While aperiodic offsets were not significantly related to slow
23 wave slopes when controlling for age and participant, this does not imply a significant
24 difference from wake oscillation amplitudes. However, offsets did not show the charac-
25 teristic decrease in overnight changes with age that was observed for slow waves and
26 wake amplitudes (Figure 2). This could suggest that offsets also reflect changes in syn-
27 aptic strength and plasticity, but may be less sensitive or specific than wake ampli-
28 tudes, especially to overnight changes.

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1 4.4 Spectral power vs oscillation burst detection

2 As explained by previous papers, changes in spectral power do not differentiate be-
3 tween changes in oscillation amplitudes or densities (Donoghue et al., 2022; Quinn et
4 al., 2019; Snipes et al., 2023; Tal et al., 2020; Zich et al., 2020). With this study, we again
5 demonstrate that amplitudes and densities meaningfully change independently, both
6 across sleep and development. However, we also found that the changes in amplitude
7 were largely captured by changes in average power, whereas changes in density were
8 largely captured by changes in periodic power. This suggests that power and periodic
9 power can be used as proxies for amplitudes and densities. Previous studies of devel-
10 opment have likewise found differences between power and *relative power* (similar to
11 periodic power) across development (Somsen et al., 1997). These results may be re-
12 interpreted as the dissociation between changes in amplitude and density.

13 Arguing against this approach, however, is the topography of overnight changes in 14–
14 18-year-olds: oscillation densities increased overnight, whereas periodic power de-
15 creased, likely reflecting the greater influence of the overnight decrease in amplitudes.
16 Similarly, the overnight increase in densities is more central than the overnight increase
17 in periodic power. Therefore, differences between power and periodic power may sug-
18 gest different influences of oscillation amplitudes and densities, but to know for cer-
19 tain, oscillations should be measured directly.

20

21 4.5 ADHD

22 Despite a relatively large sample size ($N = 58$), we did not observe any significant effects
23 of ADHD on our EEG measures. We did find exponents to be steeper on average in pa-
24 tients, supporting the results of Robertson et al. (2019), and contrasting those of Ost-
25 lund et al. (2021). One explanation could be that our participants were performing tasks
26 for most recordings, and the differences between patients and controls may mostly
27 emerge in resting EEG; patients may have developed compensation mechanisms
28 masking potential differences during the tasks. It's also possible our analysis did not
29 reach significance because our participants were a combination of both medicated and
30 unmedicated patients, and it is known that medication will reduce the effects of ADHD

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1 on the EEG (Furrer et al., 2019; Karalunas et al., 2022). Additionally, our participants
2 were screened for good sleep quality (to have a chance of falling asleep in the laborato-
3 ry with an EEG net, and to have similar levels of sleep pressure as controls). However,
4 around 40-55% of children with ADHD report sleep deficits (Becker et al., 2019; Corkum
5 et al., 1998; Holmberg & Hjern, 2006; Konofal et al., 2010), so it is possible that poor
6 sleep quality in patients contributes to differences in the wake EEG observed in prior
7 studies (Clarke et al., 2020). Finally, it's possible that only subtypes of patients with
8 ADHD have a specific relationship to any of the measured EEG markers; ADHD is highly
9 heterogeneous with varying symptoms among individuals.

10 Regardless of the reason, given that we do not see any systematic differences between
11 patients and controls, none of the wake EEG measures we tested make for a reliable
12 intrinsic marker of ADHD which could potentially be used to aid diagnosis. Instead, re-
13 search investigating such markers in these and other patient populations should take
14 special care to control for sleep/wake history and sleep quality, as these may have a
15 greater impact on the EEG. Instead, within the same participants we observed signifi-
16 cant differences in sleep slow wave activity from controls (Furrer et al., 2019), suggest-
17 ing sleep is more sensitive to developmental deficits.

18 **4.6 Limitations**

19 An important limitation of this study is the scarcity of datasets under 8. It is known that
20 in young children there is a switch from primarily synaptic growth to primarily synaptic
21 pruning, peaking in different cell populations and regions at different ages (Cao et al.,
22 2020; Petanjek et al., 2011; Shaw et al., 2008), which is also reflected in sleep slow
23 wave activity peaking around this age (Feinberg & Campbell, 2013). Wilkinson et al.
24 (2024) found an increase with age in both offsets and exponents across infants 0-3
25 years old, and McSweeney et al. (2023) found a quadratic relationship between age and
26 offsets/exponents, such that they peaked at 5-7 years old. This would suggest more
27 complex relationships between age and EEG measures than the linear trends observed
28 here.

29 A further limitation of the data is its lack of uniformity, pooling multiple experimental
30 paradigms and tasks, and medicated and unmedicated patients. Our primary findings

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1 remain significant also for more uniform subsets of the data (e.g. Figure 2 is only odd-
2 ball, Suppl. Figure 2-2 only neurotypical controls), but it's possible that different tasks
3 would generate different topographies, and significant effects of ADHD would have
4 emerged from entirely unmedicated patients.

5 This paper poses two potential weaknesses in interpretability. First, if one assumes that
6 aperiodic and oscillatory signals are a linear sum of each other in the time-domain of
7 the EEG signal, it is possible that the aperiodic changes explain part of the changes in
8 oscillation amplitudes. A better understanding of the relationship between oscillations
9 and the aperiodic signal are needed to validate such an assumption, and then precise
10 simulations would be needed to determine the magnitude of this effect. However, in
11 practice we have demonstrated that oscillation amplitudes can behave differently from
12 aperiodic measures, and therefore it is at least useful to measure them independently.
13 Second, overnight changes do not dissociate between homeostatic (related to sleep
14 pressure) and circadian (related to clock time) effects. To do so would require substan-
15 tially more intensive protocols, such as sleep deprivation, sleep restriction, or shifting
16 sleep windows over several days. However, collecting more wake recordings through-
17 out the day would already provide an indication as to when an effect is circadian or ho-
18 meostatic.

19 Finally, our data is limited to EEG. Future studies and analyses would greatly benefit by
20 comparing these measures to structural and functional brain changes observable with
21 MRI, as well as cognitive and behavioral outcome measures related to development.
22 This would bridge the gap between a purely basic research finding to practical applica-
23 tions.

24 **4.7 Conclusions**

25 We have found that overnight changes in wake oscillations provide distinct markers of
26 brain maturation. Both absolute amplitudes and overnight changes in amplitudes de-
27 crease linearly with age, the effect more occipital in younger children. Overnight chang-
28 es in oscillation density, especially of alpha oscillations, dissociate children from ado-
29 lescents and adults by switching from an overnight decrease to an increase in density.
30 Understanding the reason behind this effect would likely provide important information

Data and code availability

1 on brain development around puberty and adolescence. More generally, we have
2 shown that there are a multitude of changes in the EEG with development that go be-
3 yond simple spectral power, each with their own functional significance. Moving for-
4 ward, researchers should analyze independently oscillatory amplitudes and densities
5 as well as aperiodic exponents and offsets, as they offer different insights into neuronal
6 activity and structure.

7 **5 DATA AND CODE AVAILABILITY**

8 The data tables used for the linear mixed effects models are provided as supplementary
9 material (Suppl. Data 1, Suppl. Data 2), and the output of the linear models for wake
10 and sleep data are likewise provided (Suppl. Data 3, 4). The raw and preprocessed EEG
11 is available upon request, requiring permission from the local ethics committee.

12 The burst detection can be conducted either with the original python toolbox bycycle
13 (<https://github.com/bycycle-tools/bycycle>), or with our MATLAB implementation
14 (<https://github.com/HuberSleepLab/Matcycle>). The preprocessing and analysis code is
15 likewise open source (<https://github.com/snipeso/children-wake/>).

16 **6 AUTHOR CONTRIBUTIONS**

17 Methodology, SS, EK, VJ, CV, ML, SK, RH; Software, SS; Formal Analysis, SS, VJ; Investi-
18 gation, EK, VJ, CV, MF, MS, ML, SK, RH; Resources, ML, OGJ, RH; Writing—Original draft,
19 SS; Writing-Review & Editing, SS, EK, VJ, MF, MS, SK, OGJ, RH; Supervision, RH; Funding
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21 **7 DECLARATION OF COMPETING INTERESTS**

22 None of the authors have competing interests.

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10

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