

Changes in sleep architecture and topography of sleep EEG slow wave activity following acute wake extension in early adolescence.

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INTRODUCTION AND AIMS

Peaks in sleep slow-wave activity (SWA; EEG power in the 0.5-4Hz band) shift to frontal areas from childhood through adolescence^{1,2}.

SWA also increases acutely during recovery sleep (RS) after wake extension (i.e., staying awake longer than typical), while **sigma** power (11-15Hz sleep spindle frequency)³ decreases.

Prior studies in childhood (5-12 yr) indicate that RS SWA may be localized to specific scalp areas⁴, but unclear is whether peak SWA in early adolescence (10-13yr) is expressed in a similar scalp topography between RS and typical sleep.

Also unknown is whether child traits such as attention-deficit hyperactivity (ADHD) features influence the effects of wake extension on subsequent sleep.

CURRENT STUDY AIMS: 1) Characterize sleep architecture, spectra, and SWA topography in young adolescents after acute wake extension; 2) Explore if ADHD traits predict different sleep responses to wake extension.

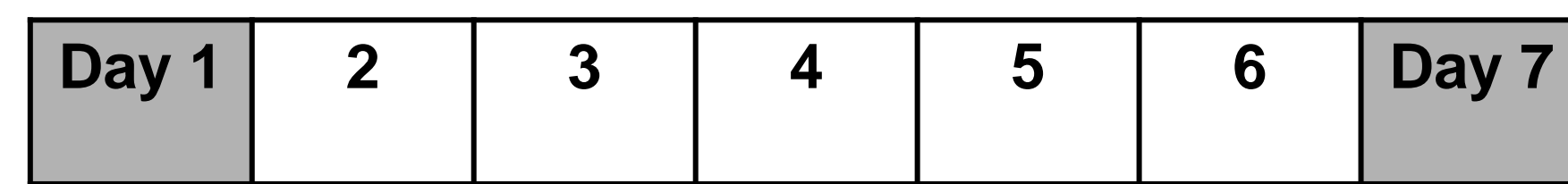
METHODS AND PROCEDURE

Participants: Young adolescents (N=22, 12F 10M); ages 10-13 years (M±SD: 12.03±1.14) with and without attention-deficit hyperactivity disorder (ADHD) diagnosis, free of mood disorders and psychosis.

Procedure:

BEFORE STUDY: Demographic characterization & at-home sleep stabilization:

Consent, KSADS, cognitive function, Conners ADHD Scales

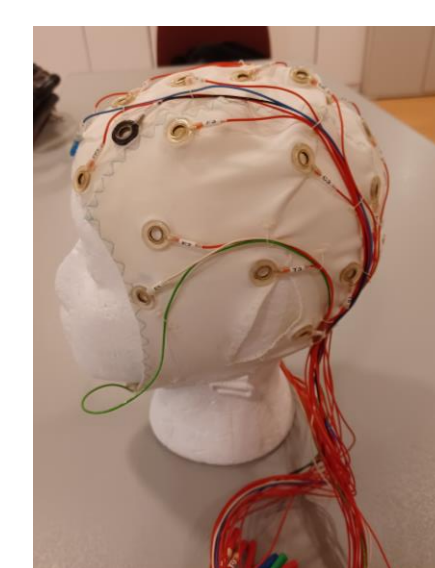
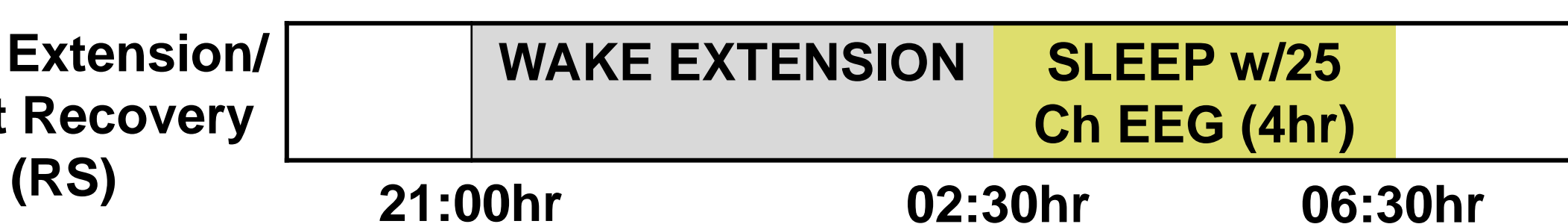


Actigraph + sleep diary (Sleep Stabilization: 9.5hr time in bed per night)



[Actigraph: Micro Motion Logger (AMI)]

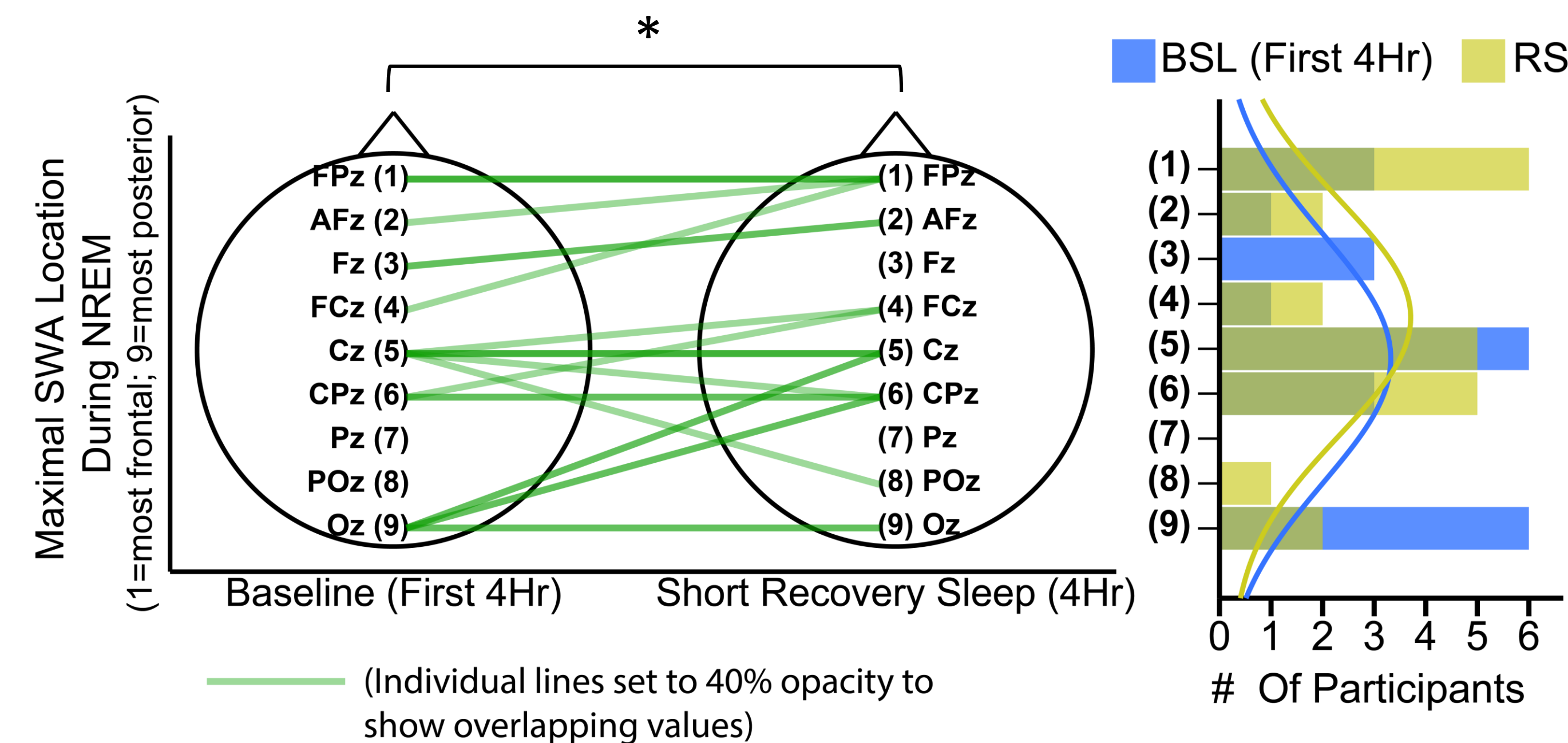
DURING STUDY: Two consecutive in-lab nights:



25-channel EEG w/ EMG & EOG at 256Hz sampling allowed R&K sleep staging and spectral power/topography analyses

When 10-13-year-olds are kept up longer and later than usual, their later sleep slow-wave activity (a sleep pressure marker) is stronger and more frontal than in rested sleep

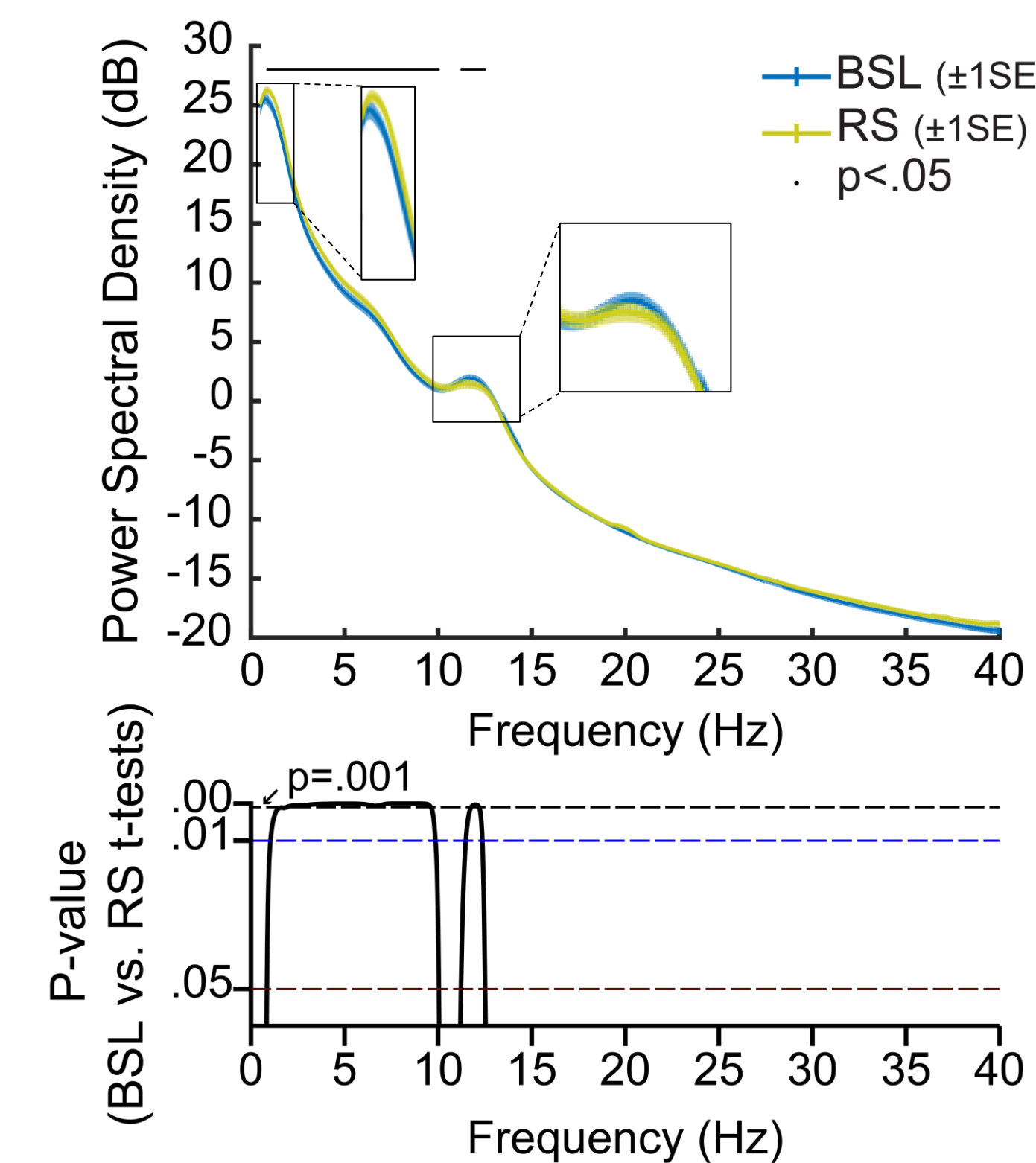
RESULTS (CONT.): MAXIMAL SLOW-WAVE ACTIVITY IN NON-REM SHIFTS FRONTALLY BETWEEN BSL (FIRST 4 HR.) AND RS



Maximal SWA location shifted from centroparietal sites during BSL (M[SD]: 5.32±2.78, i.e., Cz/CPz) to frontocentral sites during RS (M[SD]: 4.46±2.58; i.e., FCz/Cz).

This difference was significant, $t(21)=2.39, p=.027, d=.51$, indicating a shift in maximal midline SWA to more frontal areas during RS.

RESULTS (CONT.): SLEEP SPECTRAL POWER (SWA AND OTHER FREQUENCIES) DIFFERS BETWEEN BSL (FIRST 4 HR.) AND RS



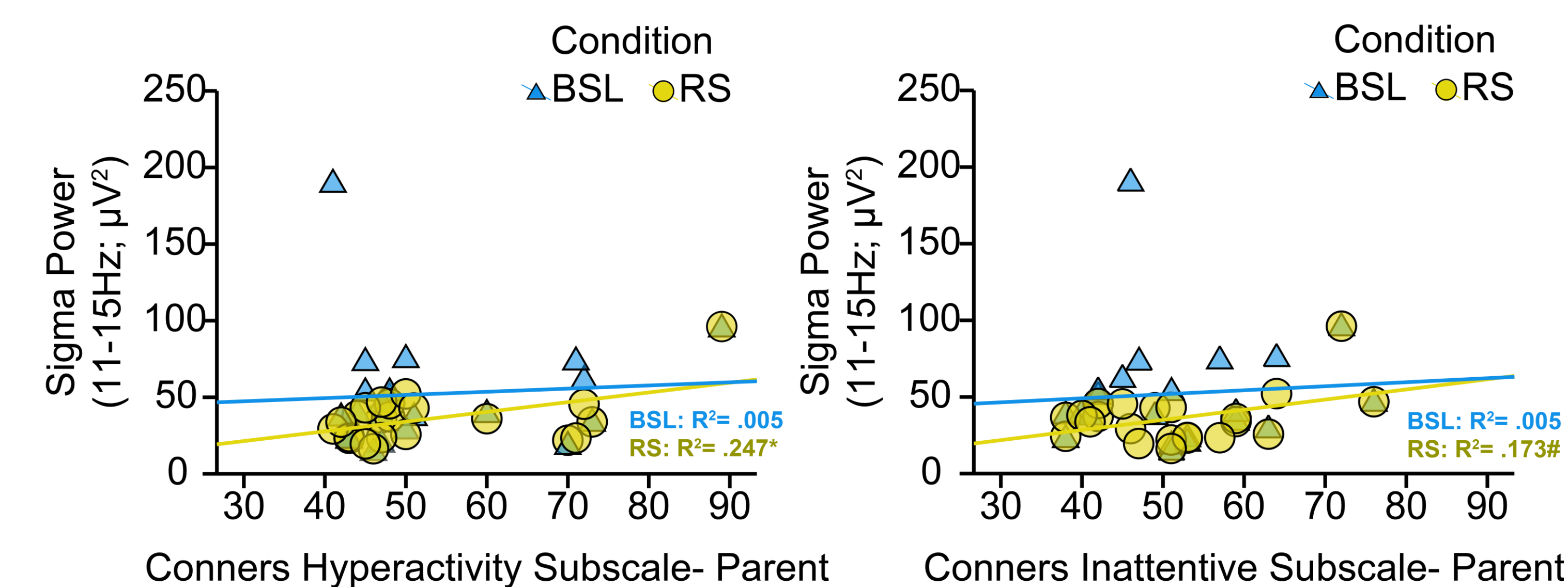
T-tests compared power differences between BSL & RS for each frequency using a sliding window of .03125Hz.

SWA (0.88Hz-4Hz) and theta (4-8Hz) bands significantly increased in RS

Conversely, sigma power decreased during RS (1.25Hz-12.5Hz)

SWA change (0.5-4Hz) was significant in 22/25 electrodes (Bonferroni-corrected significance; channels not meeting threshold= F7, FCz, T6)

EXPLORATORY: RELATIONS BETWEEN CHILD ADHD TRAITS AND SLEEP SPECTRA ACROSS BSL (FIRST 4 HR.) AND RS



Neither child inattentiveness nor hyperactivity predicted absolute global BSL or RS NREM SWA, the change in NREM SWA between conditions, or topography/shifts in maximal midline SWA between conditions (all $ps > .16$).

However, hyperactivity predicted greater sigma during RS NREM ($r=.497, p=.019$). The relation between inattentiveness and sigma was marginal ($r=.416, p=.054$).

DISCUSSION

Young adolescents show a robust homeostatic response to wake extension, indicated by 1) increased SWA and 2) decreased sigma power in subsequent recovery sleep. Adolescent ADHD traits such as hyperactivity may influence sleep characteristics during recovery sleep, including higher sigma power. Comparing maximal SWA topography between baseline and recovery sleep, we found a small frontal shift in maximal SWA over RS—shifting towards not yet fully mature cortical areas.

Future directions include assessing relations between child ADHD and cycle-by-cycle changes in SWA and sigma across sleep, to examine whether recovery sleep dissipates sleep pressure differently across the scalp and in children with varying baseline clinical profiles.

RESULTS: SLEEP LENGTH AND ARCHITECTURE CHANGES BETWEEN BASELINE (BSL) AND SHORT RECOVERY SLEEP (RS)

Sleep Variable	M(SD) BSL	M(SD) RS	T (df=21)	p(d)
Sleep Period Time (SPT; min)	546.41	237.34	60.40	<.001(12.88)***
Total Sleep Time (TST; min)	512.14	235.05	33.98	<.001(7.25)***
Sleep Efficiency (TST/SPT*100)	90.37	98.29	-5.45	<.001(-1.16)***
Sleep Stage N1 (% TST)	8.51	2.61	5.91	<.001(1.26)***
Sleep Stage N2 (% TST)	47.36	37.04	6.87	<.001(1.46)***
Slow-wave sleep (SWS; % TST)	26.76	51.65	-17.09	<.001(-3.65)***
REM sleep (% TST)	17.36	8.72	8.37	<.001(1.78)***

Our sleep manipulation successfully reduced total sleep time and increased slow-wave sleep, at the cost of REM and lighter NREM (N1 & N2)

1. Kurth, S., Ringli, M., Geiger, A., LeBourgeois, M., Jenni, O. G., & Huber, R. (2010). Mapping of cortical activity in the first two decades of life: A high-density sleep electroencephalogram study. *Journal of Neuroscience*, 30(40), 13211–13219. <https://doi.org/10.1523/JNEUROSCI.2532-10.2010>
2. Kurth, S., Ringli, M., LeBourgeois, M. K., Geiger, A., Buchmann, A., Jenni, O. G., & Huber, R. (2012). Mapping the electrophysiological marker of sleep depth reveals skill maturation in children and adolescents. *NeuroImage*, 63(2), 959–965. <https://doi.org/10.1016/j.neuroimage.2012.03.053>
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