

## REGULAR RESEARCH PAPER

# Trait-like characteristics of sleep EEG power spectra in adolescents across sleep opportunity manipulations

Ju Lynn Ong  | June C. Lo  | Amiya Patanaik  | Michael W. L. Chee 

Centre for Cognitive Neuroscience,  
Neuroscience and Behavioral Disorders  
Program, Duke-NUS Medical School,  
Singapore

**Correspondence**

Dr Michael W. L. Chee, Centre for Cognitive  
Neuroscience, Duke-NUS Medical School,  
Singapore.  
Email: michael.chee@duke-nus.edu.sg

**Funding information**

This work was supported by the National  
Medical Research Council, Singapore  
(NMRC/STaR/0004/2008 and NMRC/  
STaR/015/2013) and The Far East  
Organization.

**Abstract**

The electroencephalographic power spectra of non-rapid eye movement sleep in adults demonstrate trait-like consistency within participants across multiple nights, even when prior sleep deprivation is present. Here, we examined the extent to which this finding applies to adolescents who are habitually sleep restricted on school-days and sleep longer on weekends. We evaluated 78 adolescents across three sleep restriction groups who underwent different permutations of adequate sleep (9 hr time-in-bed), sleep restriction (5 hr time-in-bed), afternoon naps (1 hr afternoon) and recovery sleep (9 hr time-in-bed) that simulate behaviour on school-days and weekends. The control group comprised a further 22 adolescents who had 9 hr of sleep opportunity each night. Intra-class correlation coefficients showed moderate to almost perfect within-subject stability in electroencephalographic power spectra across multiple nights in both sleep restriction and control groups, even when changes to sleep macrostructure were observed. While nocturnal intra-class correlation metrics were lower in the low-frequency and spindle frequency bins in the sleep restriction compared with the control group, hierarchical clustering measures could still identify multi-night electroencephalographic spectra as originating from the same individual. The trait-like characteristics of electroencephalographic spectra from an adolescent remain identifiable despite the disruptive effects of multi-night sleep restriction to sleep architecture.

**KEYWORDS**

baseline sleep, daytime nap, inter-individual differences, recovery sleep, sleep restriction

## 1 | INTRODUCTION

Sleep electroencephalographic (EEG) spectra show a high degree of inter-individual variability, but within individuals they are remarkably consistent across multiple nights of recording akin to an electrophysiological “fingerprint” (Buckelmuller, Landolt, Stassen, & Achermann, 2006; De Gennaro, Ferrara, Vecchio, Curcio, & Bertini, 2005). In support of this idea, cluster analysis of EEG spectra obtained from 4 nights of nocturnal sleep separated by several weeks could distinguish recordings from a particular individual from a mixture of

recordings from different participants (Buckelmuller et al., 2006). “Trait-like” EEG characteristics are present in both young and old adults across multiple nights of recording (Tan, Campbell, & Feinberg, 2001; Tan, Campbell, Palagini, & Feinberg, 2000), and remain despite systematic changes in sleep architecture induced by manipulation of sleep schedules (De Gennaro et al., 2005; Tarokh, Rusterholz, Achermann, & Van Dongen, 2015), drug administration (Palagini, Campbell, Tan, Guazzelli, & Feinberg, 2000) or developmental changes (Tarokh, Carskadon, & Achermann, 2011). The level of trait-like stability in nocturnal sleep EEG spectra is important to

This is an open access article under the terms of the Creative Commons Attribution-NonCommercial License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes.

© 2019 The Authors. *Journal of Sleep Research* published by John Wiley & Sons Ltd on behalf of European Sleep Research Society

establish in order to properly assess the effects of a particular sleep manipulation (e.g. sleep restriction, daytime napping or enhancement of sleep via pharmacological or non-pharmacological means). The lack of such consistency would make it difficult or impossible to attribute a treatment effect to the experimental manipulation as opposed to random inter-session fluctuation in EEG spectra from night to night.

The present work seeks to determine if such “trait-likeness” of the sleep EEG persists across sleep restriction, naps and recovery nights in adolescents who typically shorten sleep on weeknights and compensate by taking daytime naps or extending sleep on weekends (Gradisar, Wright, Robinson, Paine, & Gamble, 2008; Lo et al., 2017). Work to date has shown high within- compared with between-subject correlations across baseline, slow-wave sleep deprivation and recovery nights of equal time-in-bed (TIB; De Gennaro et al., 2005; Tarokh et al., 2015). However, work investigating shorter TIB periods, for example during sleep restriction and naps, is lacking.

In this work, we use EEG data collected from multiple sleep manipulation protocols (combinations of 9 hr baseline nights, 5 hr sleep restriction nights, 1 hr daytime naps and 9 hr recovery nights) in order to investigate “trait-like” characteristics of the spectra during restricted and recovery sleep in adolescents. We predicted that the shape of the EEG spectrum would remain stable despite changes to sleep architecture caused by experimental manipulations replicating previous work showing trait-like stability for recovery nights following total sleep deprivation in adults (Tarokh et al., 2015). However, we expected that the degree of this “trait-likeness” would be reduced compared with a control group who did not undergo any sleep manipulation.

## 2 | MATERIALS AND METHODS

### 2.1 | Participants

Data were acquired from adolescents participating in two protocols – the Need for Sleep Studies (NFS) 1 and 2 (Lo, Ong, Leong, Gooley, & Chee, 2016; Lo et al., 2017; Ong, Lo, Gooley, & Chee, 2016, 2017). Participants met the following screening criteria: (i) between 15 and 19 years old; (ii) no history of chronic medical conditions, psychiatric illness or sleep disorders; (iii) a body mass index (BMI) of  $\leq 30 \text{ kg m}^{-2}$ ; (iv) not habitual short sleepers (i.e. not individuals who both [a] had an average actigraphically assessed TIB of  $< 6 \text{ hr}$ ; and [b] showed no sign of sleep extension for more than 1 hr on weekend compared with weekday nights); (v) daily consumption of fewer than five cups of caffeinated beverages; and (vi) no history of travel across more than two time zones 1 month prior to the experiment. Full details of the recruitment and screening criteria are detailed in our earlier work (Lo et al., 2016, 2017). The study was approved by the Institutional Review Board of the National University of Singapore, and informed consent was obtained from both participants and their legal guardian.

Data from a total of 100 participants were analysed after removal of 12 individuals whose polysomnographic (PSG) data were

heavily contaminated by artefacts (see Section 2.3). The contributing individuals were exposed to four different sleep schedules: (i) Control (13  $\times$  9 hr TIB nights,  $n = 22$ ); (ii) Sleep Restriction Group 1 (SR1; three  $\times$  9 hr TIB baseline nights, seven  $\times$  5 hr TIB sleep restriction nights, and three  $\times$  9 hr TIB recovery nights,  $n = 25$ ); (iii) Sleep Restriction Group 2 (SR2; two  $\times$  9 hr TIB baseline nights, eight  $\times$  5 hr TIB sleep restriction nights, and four  $\times$  9 hr TIB recovery nights split into two cycles,  $n = 26$ ); and (iv) Sleep Restriction Group 3 (SR3; two  $\times$  9 hr TIB baseline nights, eight  $\times$  5 hr TIB sleep restriction nights, eight  $\times$  1 hr TIB afternoon naps, and four  $\times$  9 hr TIB recovery nights split into two cycles,  $n = 27$ ). Participant characteristics and protocol details for each group are shown in Table 1 and Figure 1, respectively, and nights where PSG was recorded are indicated with an asterisk. TIB nights of 9 hr were scheduled from 23:00 hours to 08:00 hours, while 5 hr TIB nights were scheduled from 01:00 hours to 06:00 hours, and 1 hr TIB afternoon naps took place between 14:00 hours and 15:00 hours.

### 2.2 | Polysomnography

Sleep was recorded using portable EEG recording devices (SOMNOtouch RESP, SOMNOmedics GmbH, Germany). EEG was recorded from two main channels (C3 and C4 in the international 10–20 system of electrode placement) referenced to the contralateral mastoids. The common ground and reference electrode were placed at Fpz and Cz. Electrooculography (EOG; right and left outer canthi) and submental electromyography (EMG) were also used for sleep stage classification. Signals were sampled at 256 Hz and band-pass filtered between 0.2 and 35 Hz (EEG and EOG) or 1 and 128 Hz (EMG).

### 2.3 | Sleep staging and electroencephalographic spectral analysis

Sleep scoring was performed in 30-s epochs using the FASST toolbox (Leclercq, Schrouff, Noirhomme, Maquet, & Phillips, 2011). Scoring was performed by trained technicians following the criteria set by the AASM Manual for the Scoring of Sleep and Associated Events (Iber, Ancoli-Israel, Chesson, & Quan, 2007). EEG recordings were visually inspected to identify artefact-free 5-s epochs. Adaptation night recordings (Figure 1), recordings containing more than 10% artefacts (from epochs scored as sleep) and recordings from participants with unusable baseline recordings were excluded from further analyses. Each participant contributed to a minimum of 4 nights of data, which included one baseline night, and at least one sleep restriction and one recovery night. Participants in SR3 additionally had a minimum of four nap sessions. The mean, standard deviation and range of the number of nights for each group are shown in Supporting Information Table S2.

Electroencephalographic spectral analysis was then performed on non-overlapping 5-s epochs using custom routines written in

**TABLE 1** Characteristics of the Control and the three SR groups

	Control group		SR1 group		SR2 group		SR3 group		$F/\chi^2$	$p$
	Mean	SD	Mean	SD	Mean	SD	Mean	SD		
N	22.00		25.00		27.00		26.00			
Age (years)	17.00	1.15	16.40	0.87	16.91	1.16	16.67	0.88	1.69	0.17
Gender (% males)	45.45		44.00		57.70		51.90		1.19	0.75
BMI (kg m <sup>-2</sup> )	20.40	2.73	20.06	2.89	20.97	2.87	20.17	2.80	0.54	0.66
Caffeinated drinks per day	0.45	0.65	0.72	0.60	0.81	0.92	0.80	0.78	1.11	0.35
Morningness–Eveningness Questionnaire score	48.64	6.69	48.04	7.65	50.23	7.93	52.33	7.46	1.70	0.17
Self-reported sleep										
Total sleep time on weekdays (hr)	5.40	1.65	5.97	0.98	6.18	0.73	6.06	0.94	2.28	0.09
Total sleep time on weekends (hr)	8.84	2.30	8.51	1.11	8.37	1.03	8.59	1.06	0.44	0.73
Pittsburgh Sleep Quality Index global score	4.82	2.81	5.20	2.42	5.35	2.31	5.26	1.95	0.22	0.88
Raven's Advanced Progressive Matrices score	10.32	1.09	9.92	1.89	9.58	2.00	9.07	1.41	2.51	0.06

SD, standard deviation; BMI, body mass index.

Matlab R2012a (The MathWorks, Natick, MA, USA). Analysis was conducted primarily using C3/A2, unless data from C4/A1 had fewer artefacts (4.6% of all records). For each epoch, EEG spectra were computed in a manner similar to a previous study (Tarokh et al., 2011), where each individual's spectra were normalized by dividing power at each frequency (0.2 Hz bin resolution) by the total power from 0.6 to 16 Hz separately for each night/nap recording and then log-transformed. Normalization was conducted as we were more interested in characterizing changes to the morphology of the EEG spectra rather than gross changes in signal amplitude.

## 2.4 | Quantification of sleep stability

We used two approaches to quantify stability across multiple sleep periods, i.e. hierarchical cluster analysis (Tarokh et al., 2011) and the intra-class correlation coefficient (ICC). The former provides an objective measure of similarity that can simultaneously take into account the shape of the entire spectrum (Tarokh et al., 2011), while the latter is an appropriate correlational measure for studies involving repeated-measures designs (Tucker, Dinges, & Van Dongen, 2007) but is limited by taking into account information from only one frequency bin at a time. To control for the number as well as placement of recordings during the protocol, clustering and ICC metrics were additionally computed for participants with five artefact-free recordings comprising different combinations of naps, 5 hr and 9 hr nights (Supporting Information). As the results did not considerably differ, we only discuss results from the full sample in the main text.

## 2.5 | Cluster analysis

Hierarchical clustering was carried out by computing cosine distances (one minus the cosine of the included angle between observations) between pairs of log-transformed normalized EEG spectra represented as vectors (1 × 78 dimensions representing power in each frequency bin). This was implemented using the MATLAB function PDIST and LINKAGE. Dendrograms were plotted for visualization, where the height of each U-shaped line connecting two observations represents the distance between these data points (Tarokh et al., 2011). The number of participants whose EEG spectra across multiple nights clustered correctly in the three SR groups was then compared with the number from the Control group using Fisher's exact test. For SR3, we computed clustering metrics separately for the night and nap spectra, which we termed SR3-nights and SR3-naps, respectively.

## 2.6 | Intra-class correlation coefficient analysis

The ICC was additionally computed using the ratio of between-subject variance to the sum of between- and within-subject variances. Variance components were estimated using a linear mixed model fit ("lmer" function from the lme4 package in R) with restricted maximum likelihood estimation criterion and subject factor as a random intercept. ICC values were interpreted using the standard ranges (Landis & Koch, 1977): "slight" (0.0–0.2); "fair" (0.2–0.4); "moderate" (0.4–0.6); "substantial" (0.6–0.8); and "almost perfect" (0.8–1.0). Ninety-five percent confidence intervals (CIs) were also computed using the bootstrap function "bootMer" with 1,000 iterations. Frequency bins where 95% CIs did not overlap between the

Control	*		*	*			*			*	*		*	
	B1	B2	B3	C1	C2	C3	C4	C5	C6	C7	C8	C9	C10	
SR1	*		*	*			*			*	*		*	
	B1	B2	B3	M1	M2	M3	M4	M5	M6	M7	R1	R2	R3	
SR2	*	*	*		*		*	*		*		*	*	
	B1	B2	M11	M12	M13	M14	M15	R11	R12	M21	M22	M23	R21	R22
SR3	*	*	*		*		*	*		*		*	*	
	B1	B2	M11	M12	M13	M14	M15	R11	R12	M21	M22	M23	R21	R22
			N11	N12	N13	N14	N15			N21	N22	N23		
	1	2	3	4	5	6	7	8	9	10	11	12	13	14
	Day													

B 9h TIB (Baseline)  
 C 9h TIB (Control)  
 M 5h TIB (Sleep Restriction)  
 R 9h TIB (Recovery)  
 N 1h TIB (Afternoon Naps)  
 \* PSG recorded sessions

**FIGURE 1** Experimental protocols for Control, sleep restriction (SR)1, SR2 and SR3 groups. Asterisks indicate days when polysomnographic (PSG) recordings were acquired. TIB, time-in-bed

SR groups and the Control group were considered to be significantly different. For SR3, we computed ICC measures separately for the night and nap spectra, which we termed SR3-nights and SR3-naps, respectively.

### 3 | RESULTS

#### 3.1 | Participants

Participants in the four groups were similar in terms of age, sex distribution, BMI, number of caffeinated drinks consumed per day, morningness–eveningness preference, self-reported sleep quality and duration, or non-verbal intelligence (Table 1).

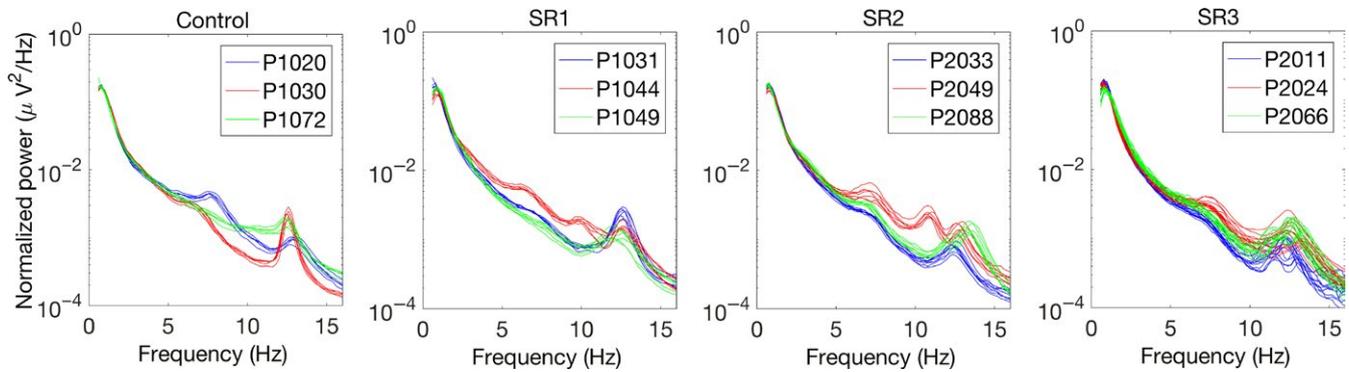
#### 3.2 | Sleep macrostructure

Electroencephalographic dynamics across nights of sleep restriction and recovery have been reported previously (Ong et al., 2016, 2017). Briefly, sleep restriction resulted in a curtailment of most sleep stages, with N3 duration remaining relatively stable. In the recovery period, we observed increased total sleep time (particularly N2 and rapid eye movement) and reduced wake after sleep onset relative to baseline, even when naps were taken during

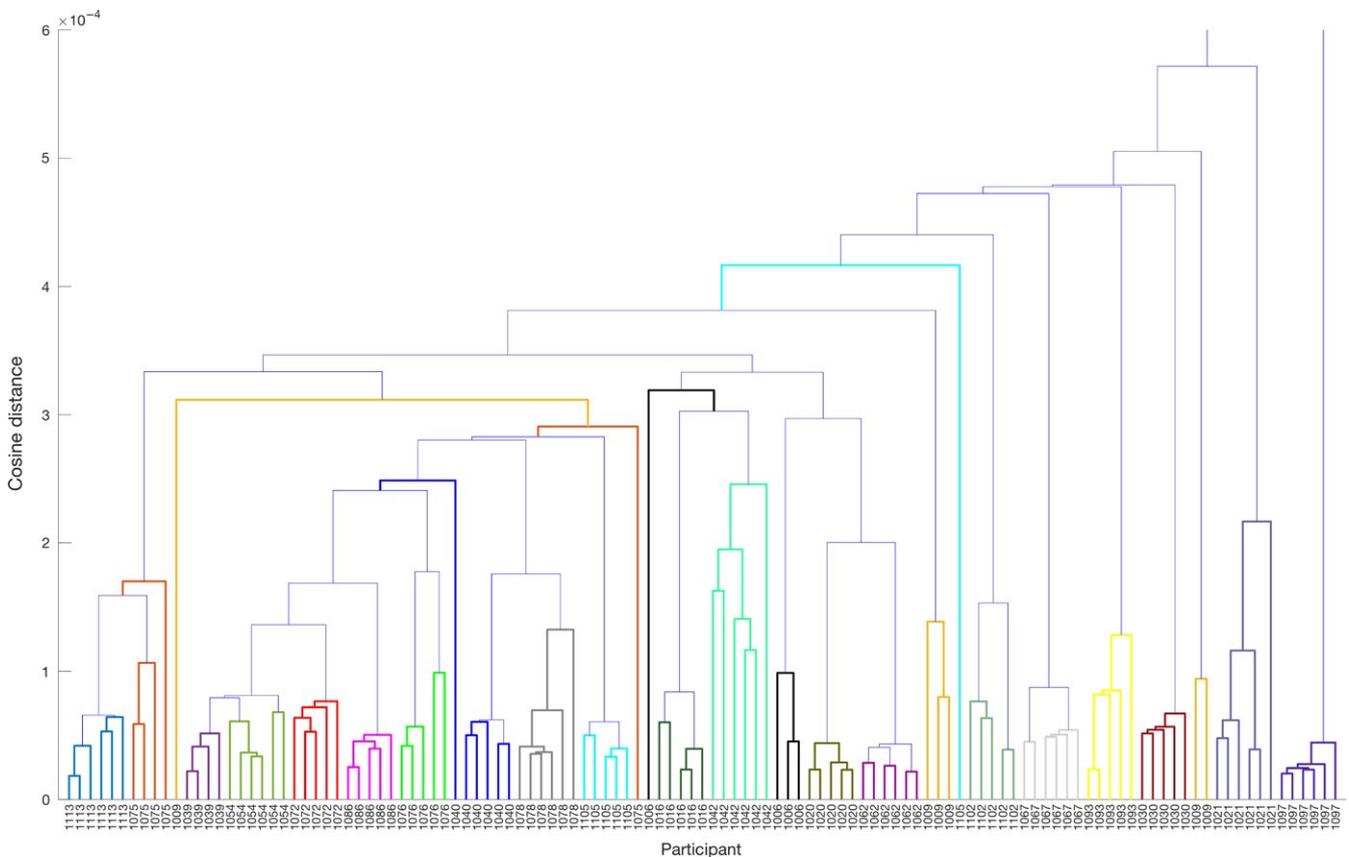
the preceding period. N3 duration again remained relatively preserved. Summaries of macrostructure parameters averaged within each subject for the baseline, sleep restriction, recovery and daytime napping periods for all four groups of participants are presented in Table S1.

#### 3.3 | Clustering metrics

In spite of large sleep macrostructure changes observed, clustering of nocturnal EEG spectra (Figure 2) was highly consistent within the same individual. In the Control group, only four of 22 participants did not have their EEG spectra across multiple nights correctly clustered. A portion of the dendrogram from the Control group is shown in Figure 3. This was compared with six of 25 participants in SR1, eight of 26 participants in SR2, and seven of 27 participants in SR3-nights. The degree of clustering was not significantly different from the Control group (Fisher's exact test,  $p = 0.73$ ,  $0.50$  and  $0.73$ , respectively). In contrast, when considering only nap records from SR3, clustering was reduced indicating greater within-subject variability. Fifteen of 27 participants in SR3-naps did not have EEG spectra across multiple nap recordings correctly clustered, and this was significantly reduced compared with the Control group ( $p = 0.0095$ ).



**FIGURE 2** Normalized non-rapid eye movement sleep spectra (in logarithmic scale) from three representative participants in each group (Control, sleep restriction [SR]1, SR2, SR3). Spectra from the same participant in each group are indicated in the same colours

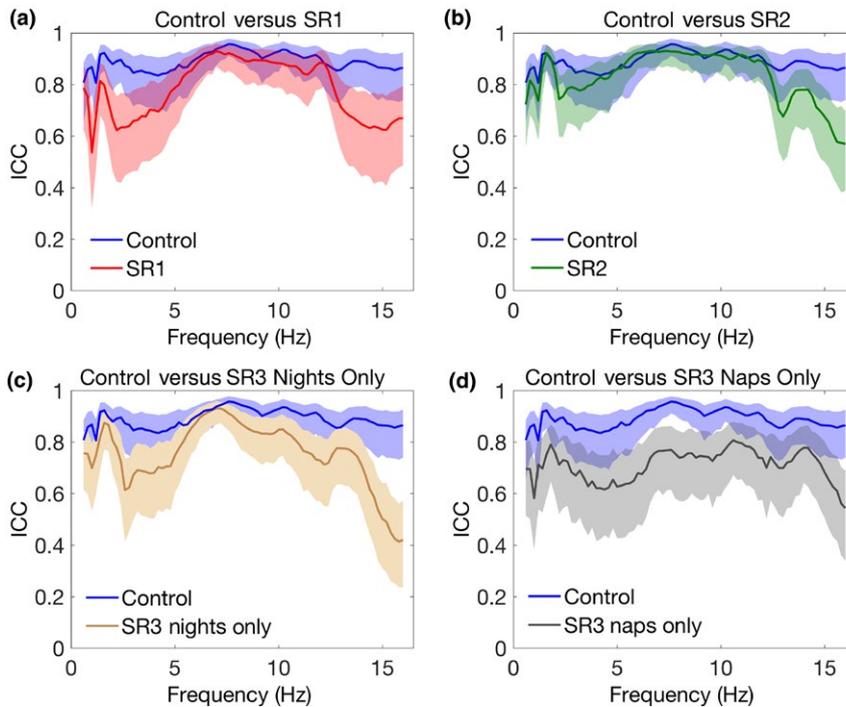


**FIGURE 3** Dendrogram from the Control group ( $N = 22$  participants), with participant numbers on the x-axis and cosine distance on the y-axis. Branches from the same participant are denoted in the same colour. In this group, 18 of 22 participants had complete clustering of all nights (i.e. all except for participants 1,006, 1,009, 1,075 and 1,105)

### 3.4 | Intra-class correlation coefficient metrics

Intra-class correlation coefficient metrics for all groups were significantly different from 0 at the  $p < 0.05$  level, indicating the presence of trait-like characteristics in spite of changes to sleep architecture. For all frequency bins considered, ICCs for the nocturnal recordings were consistently above 0.80 for the Control group, but dropped to above 0.54 for SR1, above 0.57 for SR2, and above 0.42 for SR3-nights, and above 0.54 for SR3-naps (Figure 4a–d). Bootstrapped statistics

indicated that the SR1 group differed from the Control group in 8/78 bins, comprising the delta (1, 2.2–2.4 Hz) and fast-spindle bands (13.6–14.4 Hz; Figure 4a). The SR2 group differed from the Control group in 4/78 bins (Figure 4b), comprising the fast-spindle band (15.4–16 Hz), while the SR3-nights group differed from the Control group in 12/78 bins (Figure 4c), comprising the delta (2.6–2.8 Hz) and spindle bands (11.8, 14.4–16 Hz). However, recordings from SR3-naps differed in 25/78 bins compared with the Control group, in a range of mixed frequency bins (1, 1.4–1.6, 5.8–8.8, 9.6–10.2, 15.8–16 Hz; Figure 4d).



**FIGURE 4** Intra-class correlation coefficients (ICC) values for frequency bins 0.6–16 Hz derived from the electroencephalographic (EEG) spectra. Shaded regions indicate 95% confidence intervals (CIs) for each plot. Non-overlapping regions between two plots indicate where these groups significantly differed ( $p < 0.05$ )

## 4 | DISCUSSION

Both ICC and clustering methods revealed moderate to almost perfect within-subject stability across multiple recording *nights* in spite of perturbation of sleep by experimental sleep restriction and nap opportunity. That these trait-like metrics remain relatively preserved even in adolescents who ought to exhibit high neural plasticity and adaptation to environmental stressors is reminiscent of prior work demonstrating trait-like stability in preadolescents and adolescents 1.5–3 years later (Tarokh et al., 2011). It would seem that even in the face of cortical changes during the critical neurodevelopmental period, brain oscillators underlying sleep generation remain relatively stable.

This within-subject consistency in nocturnal sleep EEG spectra is critical for the assessment of the effect of a treatment designed to alter sleep (such as acoustic stimulation or a drug), as the lack of such consistency would make it difficult or impossible to attribute a treatment effect to the experimental manipulation as opposed to random fluctuation in EEG spectra from night to night.

When comparing nocturnal recordings from the SR and Control groups, ICC metrics revealed that spectral differences associated with condition differences occurred mainly in the low-frequency and spindle frequency bins. This might be expected as these frequency bands are markers of sleep propensity that might be expected to change with the duration of wakefulness. Specifically, low-frequency activity tends to rise as a function of time awake, while spindle frequency activity is typically reduced after sleep deprivation (Aeschbach & Borbely, 1993; Dijk, Hayes, & Czeisler, 1993; Knoblach, Krauchi, Renz, Wirz-Justice, & Cajochen, 2002).

These stable trait-like measures across subjects also highlight the importance of considering the relative effect size of an experimental manipulation relative to the large inter-individual variability observed

in the EEG spectra (Tarokh et al., 2015). Within-subject investigations are clearly more appropriate if the goal is to characterize the effect of an experimental manipulation on sleep architecture that might otherwise be obscured by large between-subject differences.

Our results also reveal a reduction in the aforesaid trait-like stability in nap recordings; naps being less trait-like than nocturnal sleep. In frequency bins where ICC values were low and, thus, stability was poor, even within-subject investigations could lead to ambiguous interpretations if the effect of a particular sleep manipulation or intervention itself is masked by naturally occurring inter-session variation in nap EEG spectra.

The underlying mechanisms of these stable inter-individual differences are still unclear, and may involve genetic factors (De Gennaro et al., 2008; Landolt, 2011) or differences in structural anatomy (Buchmann et al., 2011). This is further expanded upon by findings showing that the dynamics of the homeostatic process itself are trait-like (Rusterholz, Durr, & Achermann, 2010; Rusterholz, Tarokh, Van Dongen, & Achermann, 2017). How these features relate to trait-like characteristics observed with IQ, vulnerability to sleep loss or progression of disease remain to be elucidated.

### 4.1 | Study limitations

Our findings are based on exposure to a particular combination of sleep restriction, nap and recovery nights. As sleep debt accumulates across sleep restriction nights/cycles, this could account for some of the increased within-subject variability observed in the three SR groups. Future studies that allow for a full recovery period before repeated exposures to sleep restriction/napping will allow better characterization of trait-like differences in response to sleep restriction/napping alone.

Due to the nature of the study, it was also not feasible to record EEG from more than two electrode sites. In addition to maturational changes of homeostatic sleep regulation (Jenni, Achermann, & Carskadon, 2005), high-density EEG studies have shown that regions of maximal slow-wave activity undergo a shift from posterior to anterior regions from early childhood to late adolescence (Kurth et al., 2010). As such, it is possible that the brain regions expressing trait-like features could shift with age within a wider temporal window of observation.

## 5 | CONCLUSION

The present study highlights significant trait-like characteristics in the spectra of EEG data across multiple baseline, sleep restriction and recovery nights, and across daytime naps. Nevertheless, the degree of trait-likeness differed depending on the extent of the manipulation, and was the lowest across multiple daytime naps. These findings highlight the importance of considering inter-individual differences when designing sleep studies as a significant portion of the observed variance could be attributable to inter-individual variability in sleep physiology.

## ACKNOWLEDGEMENTS

The authors would like to thank the research assistants at the Cognitive Neuroscience Laboratory and the Neuroergonomics and Cognition Laboratory for their efforts in data collection and processing.

## CONFLICTS OF INTEREST

All authors declare no conflicts of interest.

## AUTHORS' CONTRIBUTIONS

JCL and MWC designed the study. JCL and JLO collected the data. JLO analysed the data and prepared the manuscript. JCL, JLO, AP and MWC interpreted the findings and provided critical comments on the manuscript.

## ORCID

Ju Lynn Ong  <https://orcid.org/0000-0001-5115-4112>

June C. Lo  <https://orcid.org/0000-0001-7707-7557>

Amiya Patanaik  <https://orcid.org/0000-0003-2340-983X>

Michael W. L. Chee  <https://orcid.org/0000-0002-6087-0548>

## REFERENCES

Aeschbach, D., & Borbely, A. A. (1993). All-night dynamics of the human sleep EEG. *Journal of Sleep Research*, 2, 70–81. <https://doi.org/10.1111/j.1365-2869.1993.tb00065.x>

- Buchmann, A., Kurth, S., Ringli, M., Geiger, A., Jenni, O. G., & Huber, R. (2011). Anatomical markers of sleep slow wave activity derived from structural magnetic resonance images. *Journal of Sleep Research*, 20, 506–513. <https://doi.org/10.1111/j.1365-2869.2011.00916.x>
- Buckelmuller, J., Landolt, H. P., Stassen, H. H., & Achermann, P. (2006). Trait-like individual differences in the human sleep electroencephalogram. *Neuroscience*, 138, 351–356. <https://doi.org/10.1016/j.neuroscience.2005.11.005>
- De Gennaro, L., Ferrara, M., Vecchio, F., Curcio, G., & Bertini, M. (2005). An electroencephalographic fingerprint of human sleep. *NeuroImage*, 26, 114–122. <https://doi.org/10.1016/j.neuroimage.2005.01.020>
- De Gennaro, L., Marzano, C., Fratello, F., Moroni, F., Pellicciari, M. C., Ferlazzo, F., ... Malafosse, A. (2008). The electroencephalographic fingerprint of sleep is genetically determined: A twin study. *Annals of Neurology*, 64, 455–460. <https://doi.org/10.1002/ana.21434>
- Dijk, D. J., Hayes, B., & Czeisler, C. A. (1993). Dynamics of electroencephalographic sleep spindles and slow wave activity in men: Effect of sleep deprivation. *Brain Research*, 626, 190–199. [https://doi.org/10.1016/0006-8993\(93\)90579-C](https://doi.org/10.1016/0006-8993(93)90579-C)
- Gradisar, M., Wright, H., Robinson, J., Paine, S., & Gamble, A. (2008). Adolescent napping behavior: Comparisons of school week versus weekend sleep patterns. *Sleep and Biological Rhythms*, 6, 183–186. <https://doi.org/10.1111/j.1479-8425.2008.00351.x>
- Iber, C., Ancoli-Israel, S., Chesson, A., & Quan, S. F. (2007). *The AASM manual for the scoring of sleep and associated events: Rules, terminology, and technical specification* (1st edn). Westchester, IL: American Academy of Sleep Medicine.
- Jenni, O. G., Achermann, P., & Carskadon, M. A. (2005). Homeostatic sleep regulation in adolescents. *Sleep*, 28, 1446–1454. <https://doi.org/10.1093/sleep/28.11.1446>
- Knoblauch, V., Krauchi, K., Renz, C., Wirz-Justice, A., & Cajochen, C. (2002). Homeostatic control of slow-wave and spindle frequency activity during human sleep: Effect of differential sleep pressure and brain topography. *Cerebral Cortex*, 12, 1092–1100. <https://doi.org/10.1093/cercor/12.10.1092>
- 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, 13 211–13 219. <https://doi.org/10.1523/JNEUROSCI.2532-10.2010>
- Landis, J. R., & Koch, G. G. (1977). The measurement of observer agreement for categorical data. *Biometrics*, 33, 159–174. <https://doi.org/10.2307/2529310>
- Landolt, H. P. (2011). Genetic determination of sleep EEG profiles in healthy humans. *Progress in Brain Research*, 193, 51–61. <https://doi.org/10.1016/B978-0-444-53839-0.00004-1>
- Leclercq, Y., Schrouff, J., Noirhomme, Q., Maquet, P., & Phillips, C. (2011). fMRI artefact rejection and sleep scoring toolbox. *Computational Intelligence and Neuroscience*, 2011, 598 206.
- Lo, J. C., Lee, S. M., Teo, L. M., Lim, J., Gooley, J. J., & Chee, M. W. (2017). Neurobehavioral impact of successive cycles of sleep restriction with and without naps in adolescents. *Sleep*, 40, zsw042. <https://doi.org/10.1093/sleep/zsw042>
- Lo, J. C., Ong, J. L., Leong, R. L., Gooley, J. J., & Chee, M. W. (2016). Cognitive performance, sleepiness, and mood in partially sleep deprived adolescents: The need for sleep study. *Sleep*, 39, 687–698. <https://doi.org/10.5665/sleep.5552>
- Ong, J. L., Lo, J. C., Gooley, J. J., & Chee, M. W. (2016). EEG changes across multiple nights of sleep restriction and recovery in adolescents: The need for sleep study. *Sleep*, 39, 1233–1240. <https://doi.org/10.5665/sleep.5840>
- Ong, J. L., Lo, J. C., Gooley, J. J., & Chee, M. W. L. (2017). EEG changes accompanying successive cycles of sleep restriction with and without naps in adolescents. *Sleep*, 40, zsx030. <https://doi.org/10.1093/sleep/zsx030>
- Palagini, L., Campbell, I. G., Tan, X., Guazzelli, M., & Feinberg, I. (2000). Independence of sleep EEG responses to GABAergic hypnotics:

- Biological implications. *Journal of Psychiatric Research*, 34, 293–300. [https://doi.org/10.1016/S0022-3956\(00\)00019-4](https://doi.org/10.1016/S0022-3956(00)00019-4)
- Rusterholz, T., Durr, R., & Achermann, P. (2010). Inter-individual differences in the dynamics of sleep homeostasis. *Sleep*, 33, 491–498. <https://doi.org/10.1093/sleep/33.4.491>
- Rusterholz, T., Tarokh, L., Van Dongen, H. P., & Achermann, P. (2017). Interindividual differences in the dynamics of the homeostatic process are trait-like and distinct for sleep versus wakefulness. *Journal of Sleep Research*, 26, 171–178. <https://doi.org/10.1111/jsr.12483>
- Tan, X., Campbell, I. G., & Feinberg, I. (2001). Internight reliability and benchmark values for computer analyses of non-rapid eye movement (NREM) and REM EEG in normal young adult and elderly subjects. *Clinical Neurophysiology*, 112, 1540–1552. [https://doi.org/10.1016/S1388-2457\(01\)00570-3](https://doi.org/10.1016/S1388-2457(01)00570-3)
- Tan, X., Campbell, I. G., Palagini, L., & Feinberg, I. (2000). High inter-night reliability of computer-measured NREM delta, sigma, and beta: Biological implications. *Biological Psychiatry*, 48, 1010–1019. [https://doi.org/10.1016/S0006-3223\(00\)00873-8](https://doi.org/10.1016/S0006-3223(00)00873-8)
- Tarokh, L., Carskadon, M. A., & Achermann, P. (2011). Trait-like characteristics of the sleep EEG across adolescent development. *Journal of Neuroscience*, 31, 6371–6378. <https://doi.org/10.1523/JNEUROSCI.5533-10.2011>
- Tarokh, L., Rusterholz, T., Achermann, P., & Van Dongen, H. P. (2015). The spectrum of the non-rapid eye movement sleep electroencephalogram following total sleep deprivation is trait-like. *Journal of Sleep Research*, 24, 360–363. <https://doi.org/10.1111/jsr.12279>
- Tucker, A. M., Dinges, D. F., & Van Dongen, H. P. (2007). Trait interindividual differences in the sleep physiology of healthy young adults. *Journal of Sleep Research*, 16, 170–180. <https://doi.org/10.1111/j.1365-2869.2007.00594.x>

## SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

**How to cite this article:** Ong JL, Lo JC, Patanaik A, Chee MWL. Trait-like characteristics of sleep EEG power spectra in adolescents across sleep opportunity manipulations. *J Sleep Res*. 2019;e12824. <https://doi.org/10.1111/jsr.12824>