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RESEARCH ARTICLE

The impacts of intra-individual daily sleep variability on daytime functioning and sleep architecture in healthy young adults: An experimental study

Wanqi Sun[1,2] | Forrest Tin Wai Cheung[1] | Ngan Yin Chan[3] | Jihui Zhang[3,4] | Joey Wing Yan Chan[3] | Kate Ching Ching Chan[5] | Yun Kwok Wing[3] | Shirley Xin Li[1,6]

[1]Sleep Research Clinic and Laboratory, Department of Psychology, The University of Hong Kong, Hong Kong, China

[2]Shanghai Mental Health Center, Shanghai, China

[3]Li Chiu Kong Family Sleep Assessment Unit, Department of Psychiatry, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, China

[4]Center for Sleep and Circadian Medicine, The Affiliated Brain Hospital of Guangzhou Medical University, Guangzhou, China

[5]Department of Paediatrics, Faculty of Medicine, The Chinese University of Hong Kong, Hong Kong, China

[6]The State Key Laboratory of Brain and Cognitive Sciences, The University of Hong Kong, Hong Kong, China

Correspondence
Shirley Xin Li, Department of Psychology, The University of Hong Kong, Pokfulam, Hong Kong, China.
Email: shirley.li@hku.hk

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Summary

Sleep variability is commonly seen in the young populations. This study aimed to examine the impacts of experimentally induced sleep variability on sleepiness, mood, cognitive performance and sleep architectures among young adults. Thirty-six healthy individuals (aged 18–22 years) were randomly assigned to either variable sleep schedule ($n = 20$) or control ($n = 16$) groups. The protocol involved 1 week of regular sleep (time in bed = 7.5 hr) in the home setting, followed by one adaptation night (time in bed = 7.5 hr), one baseline night (time in bed = 7.5 hr), and 6 nights of sleep manipulation in the laboratory monitored by polysomnography (three cycles of variable sleep schedule by changing daily time in bed alternating between 6 hr and 9 hr for variable sleep schedule group versus fixed sleep schedule with daily time in bed for 7.5 hr for control group). Sleepiness, mood, sustained attention, processing speed, response inhibition and working memory were measured every morning and evening. The variable sleep schedule group reported a higher level of sleepiness, especially in the mornings, and increased negative mood in the evenings. There were no significant differences in positive mood, cognitive performance and sleep macro- and micro-structures. Our results showed the negative effects of sleep variability on daytime functioning especially sleepiness and negative mood, suggesting the need to address variable sleep schedules through sleep intervention.

KEYWORDS
cognitive functioning, intra-individual variability, mood, sleep, sleep architecture, sleepiness

1 | INTRODUCTION

Sleep variability, i.e. changes in sleep duration, timing and quality daily, is a common phenomenon in modern society, especially in working adults and students. There has been some evidence showing the link between sleep variability and a wide range of poor daytime functioning (Becker et al., 2017; Bei et al., 2015). Previous observational studies showed that greater day-to-day sleep variability is associated with increased negative mood (Bei, Manber, et al., 2017), higher body mass index (Moore et al., 2011), altered stress responses (Bei, Seeman, et al., 2017) and cardiac autonomic modulation (Rodríguez-Colón et al., 2015), as well as impaired brain development (Telzer et al., 2015). Recent data from community-based studies has suggested sleep duration variability as a unique sleep problem associated with an increased risk of physical and mental health problems (Slavish et al., 2019), as well as non-suicidal self-injury (Hawali & Gouda, 2022). However, these observational studies could not delineate the causal relationship between sleep variability and impaired daytime functioning.

Sleep variability is also commonly observed in individuals with sleep disturbance, especially insomnia (Suh et al., 2012). Sleep-focused interventions, such as cognitive-behavioural treatment for insomnia (CBT-I), have been shown to result in a significant decrease in sleep variability as well as improved daytime functioning (Suh et al., 2012; Vestergaard et al., 2021). However, CBT-I program usually consists of multiple educational and therapeutic components (Schutte-Rodin et al., 2008). Whether the beneficial effects of the intervention on improving sleep and daytime functioning were due to regularizing sleep–wake patterns or the improvements of other behavioural or psychological aspects remained unclear.

To understand the implications of sleep variability, few interventional studies have investigated the effects of regularizing sleep on daytime functioning, but inconsistent findings have been reported. In a previous interventional study, college students were either asked to keep consistent bedtime and rise time (i.e. intervention group) or given no instruction on their sleep schedule (control group) while maintaining their time in bed (TIB) for 7.5 hr every night for 4 weeks. This experimental protocol resulted in a more regular sleep pattern in the intervention group. The intervention group had significantly lower subjective sleepiness than the control group immediately after the intervention and at the 5-week post-intervention follow-up. In addition, reduced sleep-onset latency (SOL) and increased sleep efficiency (SE) were observed in the intervention group (Manber et al., 1996). In another interventional study, a group of habitual irregular sleepers were asked to undergo a protocol of regularizing sleep for six consecutive days. Decreased negative mood and reduced daytime heart rate variability were found immediately after the intervention. However, there were no significant changes observed in daytime sleepiness and overall sleep quality, and the improvements in negative mood and heart rate variability could not sustain once the participants resumed their irregular sleep schedule (Takasu et al., 2012). An experimental study found that keeping a strict regular sleep schedule (i.e. going to bed and getting up at the same time each day) in the lab for 38 nights did not result in significant positive effects in university students with habitual irregular sleep schedule in terms of cognitive performance and night-time sleep macro-structures (Bonnet & Alter, 1982).

Given the inconclusive evidence of the current literature, we conducted an experimental study, which aimed to directly test the impacts of sleep variability on individuals' daytime functioning (e.g. sleepiness, mood and cognitive performance) and nocturnal sleep macro- and micro-architectures while controlling for the potential confounding factors, such as average sleep duration. Specifically, the hypotheses of the current study were as follows:

1. Participants with variable sleep schedule (VSS) would have a higher level of daytime sleepiness and worse mood state than those with regular sleep schedule.
2. Participants with VSS would show poorer cognitive performance than those with regular sleep schedule.
3. Participants with VSS would have more disrupted nocturnal sleep than those with regular sleep schedule. As previous research indicated that sleep disruption could be reflected by both sleep macro-structures (e.g. longer SOL, lower SE, increased night awakenings) and micro-structures (e.g. lower electroencephalogram [EEG] delta power, higher EEG beta power during non-rapid eye movement sleep [NREM]; Kline et al., 2013), both sleep macro- and micro-structures were examined in the current study.

2 | METHODS

This study was approved by the Human Research Ethics Committee at the University of Hong Kong (EA1708030). Written informed consent was collected from all the participants. The study protocol was registered at ClinicalTrials.gov (NCT04263428).

2.1 | Participants

Forty healthy young Chinese university students (17 males) were recruited through mass e-mails, posters and promotion on social media. All interested participants initially completed an online survey as part of the screening procedure, including the reduced Morningness–Eveningness Questionnaire (rMEQ), the Pittsburgh Sleep Quality Index (PSQI), the Insomnia Severity Index (ISI), the Hospital Anxiety and Depression Scale (HADS), and the Epworth Sleepiness Scale (ESS). Participants were recruited if they met the following criteria: (1) aged between 18 and 25 years old; (2) habitual sleep duration > 7 hr. Exclusion criteria included: (1) extreme morning- or extreme evening-chronotype (i.e. rMEQ score < 8 or > 21; Adan & Almirall, 1991); (2) presence of sleep disturbance based on the self-report questionnaires (i.e. PSQI score > 5 [Buysse et al., 1989], ISI score ≥ 9 [Chung et al., 2011] or ESS score ≥ 10 [Johns, 1992]) and as confirmed by the clinical interview; (3) presence of any mood-related problems or psychiatric disorders based on the self-report questionnaires (i.e. HADS depression score > 8 or HADS anxiety score > 8; Snaith, 2003) and as confirmed by the

clinical interview; (4) presence of any chronic medical condition; and (5) on regular medication. Semi-structured clinical interviews were conducted by the trained researchers with the participants to ensure that they did not have any sleep disorders (as assessed by the Diagnostic Interview for Sleep Patterns and Disorders) or psychiatric disorders (as assessed by the Mini-International Neuropsychiatric Interview).

The targeted sample size was estimated in a priori power analysis using the online calculator GLIMMPSE (glimmpse.samplesizeshop.org). Based on a previous study by Manber et al. (1996), a sample size of 12 participants per group was required to achieve 90% power and a type I error rate of 0.01 using general linear mixed model. Therefore, the minimum number of participants required was 24.

2.2 | Two-week study protocol

Eligible participants were randomly assigned to either the VSS group or the control group upon recruitment. One week prior to the experimental session, participants were asked to wear actigraphy and complete a sleep diary on a daily basis, and were instructed to follow a regular 7.5-hr sleep schedule (0:00–07:30) and to avoid daytime napping to minimize the effect of prior sleep on sleep quality and daytime functioning during the experimental period. Compliance with this study procedure was verified by actigraphy. Participants with night-time sleep over 8.5 hr or less than 7.5 hr for more than 2 nights over the week were regarded as ineligible to undergo the second week of the laboratory protocol and were excluded from the study.

The second week of laboratory protocol (Figure 1) was carried out in the Sleep Research Clinic and Laboratory at the University of Hong Kong. The first 2 nights in the laboratory served as the adaptation night and baseline night, respectively, during which participants were allowed to have 7.5 hr of sleep opportunity (0:00–07:30). During the first adaptation night, participants underwent a full polysomnography (PSG) examination to verify the absence of sleep disorders. Following the first 2 nights, participants in the VSS group were asked to sleep on an irregular sleep schedule for 6 nights with variable TIB (i.e. three cycles of VSS, each cycle with 6 hr of TIB [01:30–07:30] for 1 night followed by 9 hr of TIB [22:30–07:30] for another night). Participants in the control group were asked to maintain a regular sleep schedule. Their overnight sleep during their stay in the laboratory was monitored by PSG. This experimental protocol aimed to simulate the typical irregular sleep–wake pattern commonly observed in college students (Fischer et al., 2020), especially during the semesters when the students need to get up at a fixed time for classes, while minimizing the shifts in the circadian phase by fixing the rise time for morning light exposure. To take into consideration the diurnal changes in sleepiness, mood and cognitive performance, and given that previous experimental study showed that sleep manipulation (e.g. sleep restriction) affected one's performance in

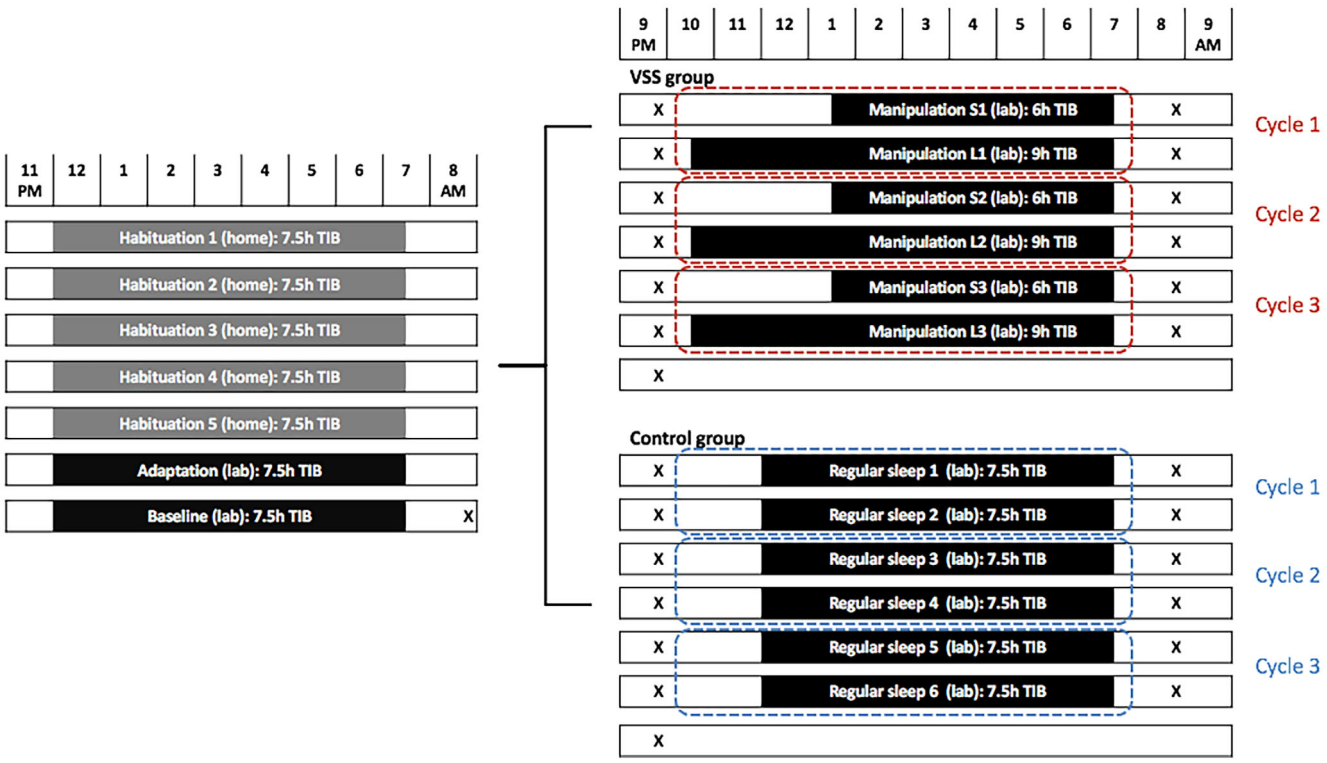

FIGURE 1 Experimental protocol. The 2-week experimental protocol consisted of 7 nights of habituation (5 nights at home setting and 2 nights in the sleep laboratory) and 6 nights of regular versus irregular sleep schedule in the sleep laboratory. Cognitive performance (marked as "x") was assessed twice a day at 08:30 and 21:30 at baseline and during the manipulation period, as well as in the morning and the evening following the baseline night and last experimental night. TIB, time in bed (TIB); S, night with short sleep (i.e. 6 hr TIB); L, night with long sleep (i.e. 9 hr TIB); VSS, variable sleep schedule

the morning more than in the evening (Lo et al., 2017), we measured and analysed the outcomes in both the mornings and evenings during the experimental period.

All the participants slept in a single bed in an air-conditioned and sound-attenuated room with an en-suite bathroom. Light-blocking curtains were closed to prevent morning sunlight during the scheduled sleep periods, while natural and artificial lighting were allowed except during the sleep periods. Artificial light in the bedroom was comparable to usual indoor lighting. During their stay in the sleep lab, participants were allowed to read and use electronic devices during the light-on period. Light snacks were provided upon request. During the daytime, participants were allowed to leave the lab for classes and were instructed to return to the lab by 20:00. Throughout the study, sleep–wake patterns and activity were continuously monitored by wrist-actigraphy to ensure that participants did not have any unscheduled sleep. Participants were asked to avoid caffeine and alcohol consumption during the entire 2 weeks. A total of HKD$ 1000 (7HKD = 1USD) was given to the participants upon completing the experimental protocol.

3 | MEASURES

3.1 | Daytime functioning

A computerized battery of cognitive tasks and two self-report measures on sleepiness and mood were administered twice a day (08:30 and 21:30) during the manipulation period. The tasks were programmed using E-prime and the whole battery lasted for approximately 30 min. The measures were completed in the following order: the Sanford Sleepiness Scale (SSS); the Positive and Negative Affect Scale (PANAS); the Digit Symbol Substitution Task (DSST); the Stop Signal Task; the Verbal 2-back task; and a 10-min Psychomotor Vigilance Task (PVT).

The SSS is a seven-point scale used to measure subjective sleepiness (Carskadon & Dement, 1981). The PANAS was used to assess positive and negative mood. Higher scores on PANAS negative mood subscale suggest more negative affect, and higher scores on PANAS positive mood subscale suggest more positive affect. Low levels of positive affect and high levels of negative mood have been found in individuals with depression (Watson et al., 1988). Details about the neurobehavioural tasks were described in the supplemental materials.

3.2 | Sleep measures

3.2.1 | Actigraphy

Participants were asked to record their sleep–wake time daily in a sleep diary and wear an actigraphy (Actiwatch Spectrum Pro, Philips Respironics, USA) on their non-dominant wrist throughout the study period. Sleep parameters were calculated based on the recorded activity counts in 1-min epoch intervals. When scoring actigraphy data in Actiware software, we determined and set the rest intervals using the inputs in the following order of importance: (1) event marker; (2) sleep diary; (3) white light intensity; (4) activity level, with reference to a previous study (Patel et al., 2015). Sleep parameters generated for the analysis included the mean and within-subject standard deviation of total TIB, total sleep time (TST), SE, SOL, wake after sleep onset (WASO) and fragmentation index. Due to the technical failure, the actigraphy data of two participants were not valid for analysis.

3.2.2 | Polysomnography

The PSG recordings were conducted in a standardized sleep lab during the second week of laboratory protocol using Compumedics E-series (Compumedics, Australia), including EEG (F3, F4, C3, C4, P3, P4, O1 and O2 in the international 10–20 system of electrode placement), electrooculography, electromyography and electrocardiography. Signals were sampled at 512 Hz, and filtered between 0.3 and 35 Hz for EEG. Sleep staging was manually scored for each 30-s epoch as Wake, N1, N2, slow-wave sleep (SWS) or rapid eye movement (REM) sleep, following the criteria set by the AASM Manual for the Scoring of Sleep and Associated Events. In addition, SE, SOL, REM latency, WASO and total arousal index were calculated. Due to technical failure, 1 night of sleep data from one participant was not included in the analyses, and five participants had 1 night of sleep data that were partially valid (i.e. sleep latency) for analyses.

The analysis of EEG spectral power during NREM of two main EEG channels (C3 and C4) was performed using MATLAB R2015b (The MathWorks, USA) and EEGLab toolbox. EEG power density was calculated using non-overlapping 4-s epochs (Tarokh et al., 2014). The spectral power was computed using the modified periodogram method (Hamming window; 0.25-Hz bin resolution) and integrated using the trapezoidal rule for integral approximation. The following spectral power in the classical EEG bands was calculated: delta (1–4 Hz); theta (4–8 Hz); alpha (8–12 Hz); sigma (12–15 Hz); and beta (15–30 Hz).

Both sleep macro- and micro-structural parameters were calculated for the entire night and in the first 6 hr of the night, respectively (Ong et al., 2016). The percentage of WASO, REM, N1, N2 and SWS sleep over the entire night and in the first 6 hr of the night between the two groups were calculated.

3.3 | Statistical analysis

To examine the cumulative effects of sleep variability (i.e. repeated cycles of VSS) on sleep and daytime functioning, mixed-effect models were conducted in which the number of the cycle of VSS, group, and group × cycle interaction were entered as fixed effects and the participant was entered as a random effect. Baseline performance was controlled in the analyses as covariates. The average performance in the morning and evening on neurobehavioural tests during the first day

TABLE 1 Characteristics of the sample

	VSS group ($N = 20$)	Control group ($N = 16$)	Statistical value (p)
Age, years, mean (SD)	20.55 (1.11)	19.73 (1.39)	$t = 1.94$ (0.062)
Male, n (%)	10 (50.0)	7 (43.8)	$\chi^2 = 0.14$ (0.709)
BMI (kg m^{-2}), mean (SD)	20.48 (1.76)	20.81 (2.90)	$t = -0.43$ (0.691)
rMEQ score, mean (SD)	14.55 (2.37)	13.94 (3.38)	$t = 0.64$ (0.527)
ISI score, mean (SD)	3.85 (2.11)	3.50 (2.66)	$t = 0.44$ (0.662)
ESS score, mean (SD)	5.10 (2.08)	5.75 (2.54)	$t = -0.85$ (0.404)
PSQI total score, mean (SD)	2.95 (1.36)	3.75 (1.88)	$t = -1.48$ (0.147)
HADS scores			
Anxiety subscale score, mean (SD)	3.35 (2.16)	4.25 (2.35)	$t = -1.20$ (0.241)
Depression subscale score, mean (SD)	3.25 (1.94)	3.00 (1.90)	$t = 0.39$ (0.701)

Note: There were no significant differences in demographic characteristics, habitual sleep and mood status between the two groups.
Abbreviations: BMI, body mass index; ESS, Epworth Sleepiness Scale; HADS, Hospital Anxiety and Depression Scale; ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; rMEQ, reduced Morningness–Eveningness Questionnaire; VSS, variable sleep schedule.

TABLE 2 Mixed-effect models of the effect of sleep variability on subjective sleepiness, mood and cognitive functioning

	Descriptive analysis (mean, SD)		Fixed-effects (F, p)		
	VSS group	Control group	Group	Cycle	Group × cycle
Daily average					
SSS score	2.79 (1.23)	2.22 (0.90)	**5.12 (0.030)**	1.86 (0.136)	1.77 (0.153)
Positive affect	19.46 (6.05)	22.94 (5.96)	0.16 (0.688)	**6.88 (< 0.001)**	0.72 (0.541)
Negative affect	16.00 (4.06)	18.92 (5.09)	0.30 (0.587)	**2.76 (0.042)**	1.33 (0.265)
PVT reaction time (ms)	319.65 (94.60)	321.42 (86.12)	0.32 (0.575)	**6.87 (< 0.001)**	0.09 (0.964)
SSRT (ms)	264.90 (69.35)	240.46 (67.25)	1.52 (0.226)	1.78 (0.150)	0.48 (0.695)
DSST (N)	77.18 (9.06)	72.82 (8.05)	0.18 (0.677)	**63.97 (< 0.001)**	0.95 (0.418)
Verbal 2-back A'	0.96 (0.04)	0.96 (0.04)	0.07 (0.794)	**3.98 (0.008)**	0.73 (0.536)
Morning					
SSS score	2.93 (1.17)	2.31 (0.86)	**4.96 (0.033)**	0.54 (0.659)	1.54 (0.205)
Positive affect	18.96 (5.66)	22.90 (6.25)	0.57 (0.456)	**7.73 (< 0.001)**	0.39 (0.759)
Negative affect	15.74 (3.98)	18.86 (5.11)	0.20 (0.655)	2.19 (0.091)	0.23 (0.876)
PVT reaction time (ms)	335.10 (124.47)	335.19 (106.46)	0.33 (0.572)	**5.19 (0.002)**	0.07 (0.977)
SSRT (ms)	264.40 (66.88)	239.21 (71.63)	1.42 (0.242)	1.47 (0.224)	0.24 (0.865)
DSST (N)	76.13 (9.43)	71.91 (7.27)	0.31 (0.579)	**41.25 (< 0.001)**	0.20 (0.893)
Verbal 2-back A'	0.96 (0.05)	0.96 (0.04)	0.47 (0.499)	**8.92 (< 0.001)**	0.55 (0.650)
Evening					
SSS score	2.65 (1.27)	2.13 (0.93)	1.64 (0.210)	1.94 (0.124)	0.75 (0.524)
Positive affect	19.94 (6.40)	22.98 (5.68)	< 0.01 (0.967)	2.49 (0.061)	0.80 (0.493)
Negative affect	16.26 (4.14)	18.98 (5.10)	0.31 (0.580)	1.26 (0.289)	**3.02 (0.031)**
PVT reaction time (ms)	304.43 (45.76)	307.64 (56.44)	0.11 (0.738)	**5.30 (0.002)**	0.80 (0.496)
SSRT (ms)	265.40 (71.93)	241.72 (62.87)	0.99 (0.327)	0.47 (0.706)	0.75 (0.523)
DSST (N)	78.21 (8.59)	73.73 (8.70)	0.08 (0.785)	**29.81 (< 0.001)**	1.16 (0.325)
Verbal 2-back A'	0.97 (0.03)	0.96 (0.05)	0.91 (0.348)	0.53 (0.660)	0.23 (0.875)

Note: Significant results were marked in bold. Baseline cognitive performance in each task was controlled in the mixed models.
Abbreviations: DSST, Digit Symbol Substitution Task; PVT, Psychomotor Vigilance Task; SSRT, stop signal reaction time; SSS, Sanford Sleepiness Scale; VSS, variable sleep schedule.

was used as the baseline data. In addition to examining daily average performance, mixed-effect models were also conducted separately to examine participants' functioning in the mornings and evenings because there might be diurnal differences in the effects of sleep variability on daytime functioning. Planned comparisons with Bonferroni adjustment were performed, when: (1) there was a significant group effect, to compare the group differences within each cycle; or (2) a group × cycle interaction effect, to compare the group differences within each cycle, as well as the cycle differences within each group. All the analyses were performed using the Statistical Package for Social Sciences (SPSS) for Windows 22.0 (SPSS, Chicago, IL, USA). The statistical significance level was set at 0.05.

4 | RESULTS

4.1 | Sample characteristics and baseline sleep features during the habituation week

Four participants (two in the VSS group) quit during the second week of laboratory protocol due to physical illness ($n = 2$), unwillingness to continue ($n = 1$), and non-compliance with the experimental procedure ($n = 1$). There were no differences in demographic features, mood status and habitual sleep between the completers and non-completers. Less than 10% of the data were missing for each outcome variable and the data were missing at random (Table S1). The final

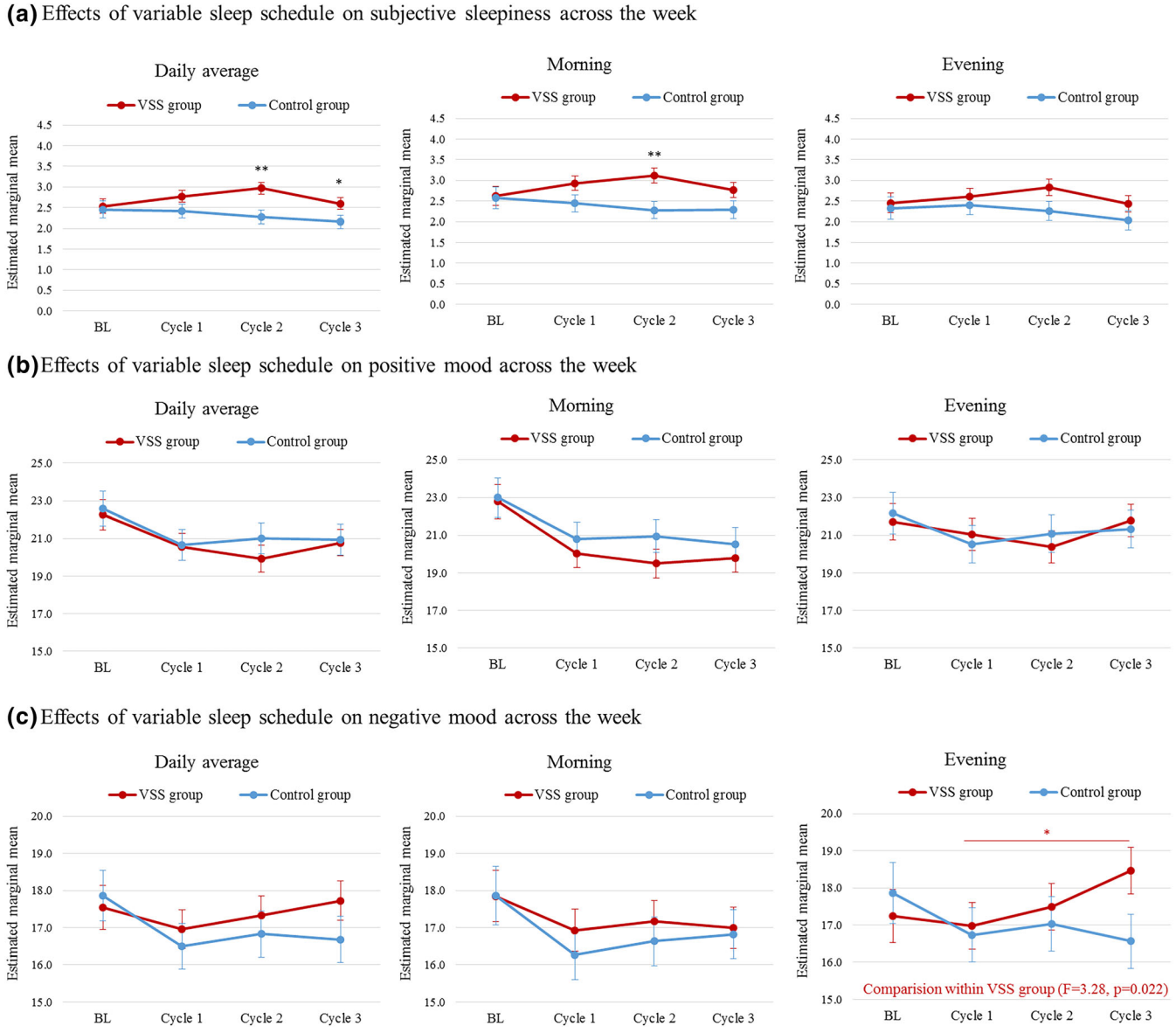

FIGURE 2 Effects of VSS on subjective sleepiness and mood across the experimental week. (a) Subjective sleepiness was measured by the Sanford Sleepiness Scale (SSS), with higher scores indicating more sleepiness. (b) Positive mood was measured by the positive affect subscale from Positive and Negative Affect Scale (PANAS), with higher scores suggesting more positive affect. (c) Negative mood was measured by the negative affect subscale from PANAS, with higher scores suggesting more negative affect. Estimated marginal mean ± SEM from the mixed-effect models of the VSS group (red) and the control (blue) groups were plotted. Red asterisks indicate significant differences across sleep cycles within the VSS group; black asterisks indicate significant differences between groups within each cycle (*$p < 0.05$, **$p < 0.01$). BL, baseline; VSS, variable sleep schedule

sample comprised of 36 participants (mean age ± SD: 20.5 ± 1.1 years, 47.2% male). There were no significant differences in demographic features, mood and habitual sleep between the two groups (Table 1). Details on baseline sleep features during the habituation week are described in the supplemental materials.

4.2 | Effects of irregular sleep schedule on daytime functioning

The results of the mixed-effect models on the effects of sleep manipulation on daytime functioning are shown in Table 2 and Figure 2. Participants in the VSS group showed higher average daily sleepiness level than those in the control group during the experimental period ($F = 5.12$, $p = 0.030$). Planned comparison showed that average daytime sleepiness was significantly higher in the VSS group than the control group in the second cycle ($p = 0.002$) and in the last cycle ($p = 0.048$; Figure 2a). Notably, the differences in sleepiness between the two groups were observed in the morning ($F = 4.96$, $p = 0.033$), and planned comparison showed that average daytime sleepiness was significantly higher in the VSS group than the control group in the second cycle of morning assessment ($p = 0.003$; Figure 2b).

There was no significant group effect and interaction effect on positive mood, whilst there was a significant cycle effect on positive mood ($F = 6.88$, $p < 0.001$), especially in the mornings ($F = 7.73$, $p < 0.001$; Table 2). As shown in Figure 2(b), both groups showed reduced positive mood over the course of the experiment.

Although there were no group effect and group × cycle effects on the daily average negative mood rating, further analyses separating testing time suggested a significant effect of group × cycle interaction on negative mood in the evenings ($F = 3.02$, $p = 0.031$). In particular, negative mood reported by the participants in the VSS group was significantly higher in the evening in the third cycle than that of the first cycle ($p = 0.019$; Figure 2c), indicating an increase in the level of negative mood across the 1-week period with VSS (comparison within VSS group: $F = 3.28$, $p = 0.022$). However, there was no such elevation of negative mood in the control group across the three cycles (Figure 2c).

Overall, there were no significant main effects of group or group × cycle interaction on cognitive abilities ($p > 0.05$; Figure S1; Table 2). However, there was a significant cycle effect on the daily average performance of PVT ($F = 6.87$, $p < 0.001$), DSST ($F = 63.97$, $p < 0.001$) and 2-back tasks ($F = 3.98$, $p = 0.008$) over the experimental period. In particular, the reaction time on the PVT task (Figure S1a) and the correct number in the DSST task (Figure S1c) were increasing across cycles, indicating decremented sustained attention but improved processing speed. A significant improvement in working memory as measured by the 2-back task from baseline to the first cycle in the morning was observed in both groups (Figure S1d), which might be due to the practice effect. There was no significant cycle effect on stop signal reaction time (SSRT; Figure S1b).

4.3 | Effects of irregular sleep schedule on sleep architecture

Comparisons of PSG sleep parameters in minutes are shown in Table S4. In brief, the VSS group had greater variability in several sleep parameters (TIB, TST, N1, N2, REM) and longer average N2 sleep than the control group ($p < 0.05$).

There was no significant group or interaction effect on % of different sleep stages, SOL and SE (Table 3). Significant cycle effects were found for % of N2 and REM sleep in the entire night and in the first 6 hr, and % of SWS in the first 6 hr. Specifically, both groups showed a gradual increase in REM sleep in the first 6 hr and entire night, an increase in SWS in the first 6 hr, and a decrease in N2 sleep in the first 6 hr and entire night over the experimental period ($p < 0.05$; Figure 3). There was no significant group or interaction effect on power spectrum density (PSD; Table 3). There was a decrease in beta band power during the whole night over the experimental period in both groups as indicated by the significant cycle effect ($F = 3.53$, $p = 0.016$; Figure S2).

We further conducted the sensitivity analysis by adjusting for TST SD in the mixed-effect models, and the main results regarding the effects on daytime functioning and sleep architecture remained comparable (Table S5).

5 | DISCUSSION

The present study examined the effects of experimentally induced variability in sleep schedule on daytime functioning and sleep macro- and micro-structures in healthy young adults. The experimental protocol involved three cycles of day-to-day sleep variability, particularly in sleep duration, which were found to result in significant changes in daily average daytime sleepiness and negative mood in the evenings, albeit no significant differences in overnight sleep architecture and the cognitive abilities tested in this study.

5.1 | Effects of irregular sleep schedule on daytime functioning

In the current study, experimentally induced sleep variability resulted in increased self-rated sleepiness, without affecting objectively alertness as reflected by the performance on the PVT task. In contrast, previous studies showed that a typical sleep restriction paradigm resulted in greater impairment in objectively assessed alertness as compared with subjective rating of alertness level (Zhou et al., 2012). These observations suggested that the impact of sleep variability might be distinct from that of sleep deprivation with a different underlying mechanism. It was also possible that the effects of VSS might take time to consolidate to affect objective performance. Interestingly, our results were in line with the observations noted in some clinical populations. For example, individuals with insomnia, often presenting with increased sleep variability, were found to have a higher level of self-reported daytime sleepiness but show a

comparable behavioural performance on the vigilance task as compared with healthy sleepers (Cohen et al., 2017).

Our results were consistent with a previous naturalistic observational study conducted in university students (Bei, Manber, et al., 2017), which showed that negative mood, especially in the evenings, was affected by irregular sleep schedules. It is possible that changes in sleep pattern might have differential impacts on mood with distinct valences. In a previous study conducted in the adolescents that involved an experimental protocol of 5 nights of sleep restriction and 2 nights of recovery, positive mood was found to restore, but increased negative mood induced by sleep restriction became even more prominent after recovery sleep (Booth et al., 2021). This result was similar to the current observation where a change in negative mood could not be fully restored when being exposed to variable day-to-day sleep schedule characterized by alternating between sleep restriction and sleep recovery. Notably, sleep variability, especially in sleep duration, is commonly observed in the clinical population with mental health conditions, such as depression and substance use (Kwon et al., 2019; Slavish et al., 2019). Taken together, our findings might provide an insight into the role of sleep variability in the risk of psychopathology.

In terms of cognitive performance, there were no significant changes in several cognitive abilities, including sustained attention, processing speed, response inhibition and working memory, upon the experimental manipulation. Our results were in line with previous research suggesting that sleep variability per se without inducing sleep loss might have a limited influence on daytime cognitive performance such as vigilance and math (Bonnet & Alter, 1982). It was possible that mild sleep variability might not be sufficient to induce changes in cognitive abilities at a behavioural level. In this regard, a recent study showed that youth with insomnia had a comparable performance on the cued go/no-go task as compared with healthy sleepers, but showed altered brain activities as measured by event-related potential, suggesting impaired inhibitory

TABLE 3 Mixed-effect models of the effect of sleep variability on PSG sleep parameters

	Descriptive analysis (mean, SD)		Fixed-effects (*F*, *p*)		
	VSS group	Control group	Group	Cycle	Group × cycle
TST (min)	417.59 (76.14)	413.61 (21.30)	<0.01 (0.986)	0.16 (0.923)	0.05 (0.987)
SOL (min)	14.18 (19.62)	18.10 (15.59)	0.05 (0.833)	2.29 (0.079)	0.10 (0.962)
REM latency (min)	72.96 (24.90)	71.34 (21.92)	0.15 (0.704)	0.66 (0.581)	1.85 (0.140)
Entire night					
SE (%)	93.17 (4.01)	92.08 (4.51)	0.09 (0.768)	0.85 (0.467)	0.50 (0.682)
WASO (%)	3.79 (2.47)	3.81 (2.57)	0.68 (0.417)	1.19 (0.314)	1.87 (0.137)
REM sleep (%)	26.51 (3.59)	28.02 (4.74)	3.41 (0.073)	**3.89 (0.010)**	2.01 (0.114)
N1 sleep (%)	4.92 (1.83)	5.28 (1.49)	0.25 (0.618)	0.68 (0.568)	0.44 (0.726)
N2 sleep (%)	47.07 (7.79)	42.44 (8.10)	3.17 (0.084)	**3.38 (0.019)**	0.42 (0.736)
SWS (%)	21.14 (6.80)	23.78 (5.31)	0.04 (0.838)	2.38 (0.071)	0.53 (0.665)
Delta band PSD	2.56 (0.19)	2.56 (0.18)	0.16 (0.695)	1.29 (0.279)	0.11 (0.953)
Theta band PSD	1.60 (0.14)	1.59 (0.12)	0.33 (0.570)	1.28 (0.282)	0.31 (0.817)
Alpha band PSD	1.06 (0.17)	1.03 (0.14)	0.35 (0.556)	2.13 (0.097)	0.85 (0.466)
Sigma band PSD	0.70 (0.15)	0.70 (0.16)	0.01 (0.940)	1.27 (0.287)	1.11 (0.345)
Beta band PSD	0.48 (0.12)	0.54 (0.13)	1.40 (0.245)	**3.53 (0.016)**	0.50 (0.685)
First 6 hr					
SE (%)	93.18 (5.44)	91.77 (5.60)	0.04 (0.844)	1.00 (0.394)	0.84 (0.475)
WASO (%)	3.40 (2.50)	3.35 (1.62)	0.92 (0.343)	0.33 (0.802)	2.11 (0.101)
REM sleep (%)	23.17 (4.81)	24.91 (4.66)	2.98 (0.093)	**2.89 (0.037)**	0.91 (0.439)
N1 sleep (%)	4.68 (1.84)	5.33 (1.59)	0.54 (0.469)	0.50 (0.683)	0.17 (0.920)
N2 sleep (%)	47.25 (7.36)	41.90 (7.54)	3.05 (0.090)	**5.89 (0.001)**	0.56 (0.640)
SWS (%)	24.54 (6.56)	27.85 (5.88)	0.73 (0.397)	**2.77 (0.043)**	0.39 (0.759)
Delta band PSD	2.60 (0.19)	2.59 (0.18)	0.09 (0.772)	1.08 (0.357)	0.17 (0.917)
Theta band PSD	1.63 (0.14)	1.61 (0.11)	0.20 (0.654)	1.05 (0.372)	0.27 (0.844)
Alpha band PSD	1.07 (0.17)	1.04 (0.15)	0.43 (0.516)	1.71 (0.166)	0.85 (0.468)
Sigma band PSD	0.70 (0.15)	0.71 (0.16)	0.09 (0.770)	0.92 (0.432)	1.65 (0.179)
Beta band PSD	0.48 (0.12)	0.54 (0.13)	0.27 (0.609)	2.09 (0.103)	0.66 (0.579)

Note: Significant results were marked in bold. Baseline sleep was controlled in the mixed models.
Abbreviations: N1, non-REM sleep stage 1; N2, non-REM sleep stage 2; PSD, power spectrum density; REM, rapid eye movement sleep; SE, sleep efficiency; SOL, sleep-onset latency; SWS, slow-wave sleep; TST, total sleep time; VSS, variable sleep schedule; WASO, wake after sleep onset.

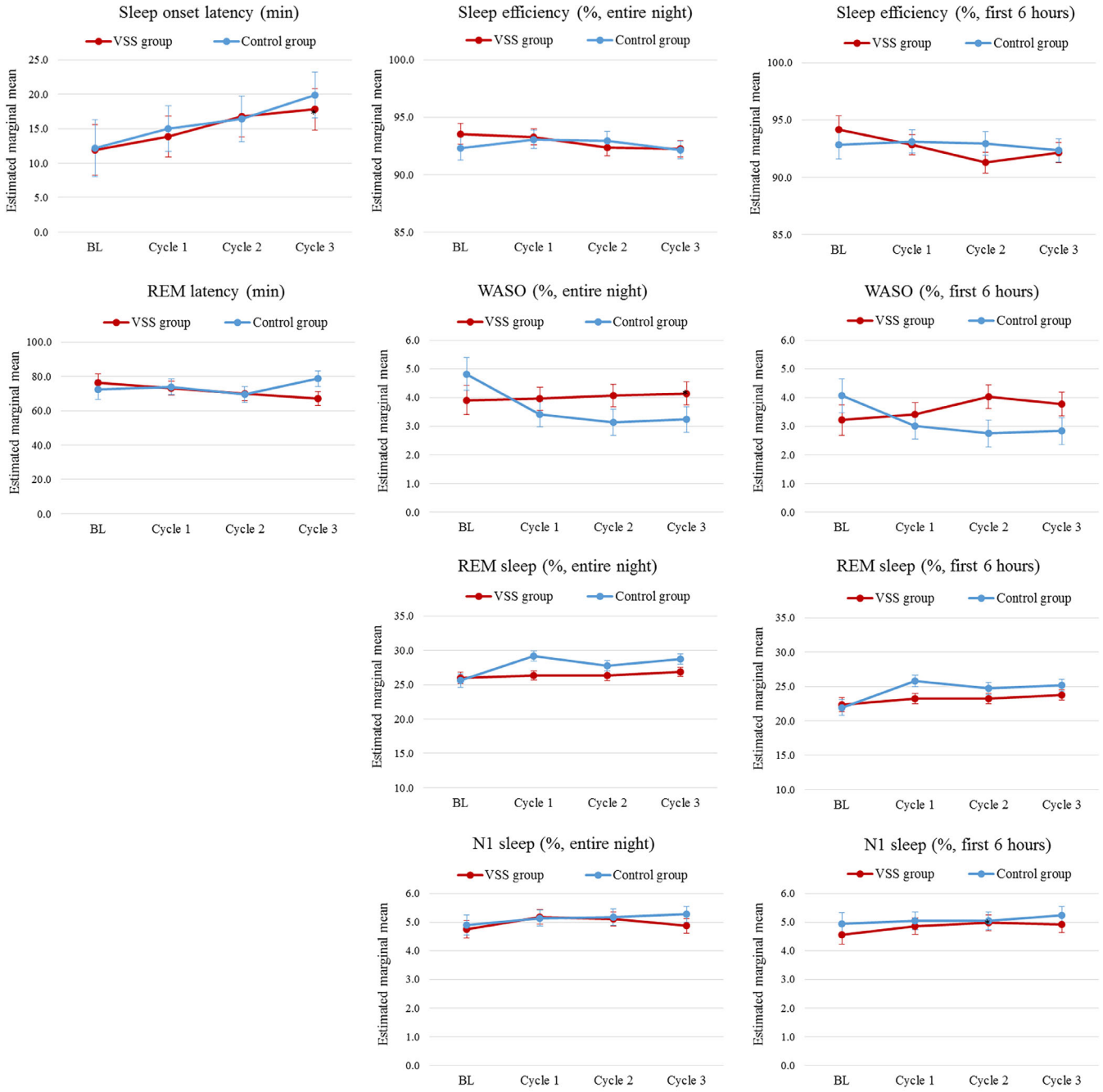

FIGURE 3 Effects of VSS on sleep macrostructure across the experimental week. Mean ± SEM of the VSS group (red) and the control (blue) groups were plotted. REM, rapid eye movement sleep; WASO, wake after sleep onset; N1, non-REM sleep stage 1; N2, non-REM sleep stage 2; SWS, slow-wave sleep; BL, baseline; VSS, variable sleep schedule

control (Ling et al., 2021). Future studies with neuroimaging techniques are needed to further examine possible underlying changes in brain activity in response to sleep variability.

5.2 | Effects of irregular sleep schedule on sleep architecture

This experimental study showed that 1 week of VSS had minimum effect on sleep architecture. Previous epidemiological studies showed that sleep variability is commonly observed in individuals with insomnia (Suh et al., 2012), and greater actigraphy-measured variability in WASO but not SOL was associated with insomnia symptoms (Buysse et al., 2010). Yet this experimental study did not find significant changes in insomnia-related sleep parameters as a result of sleep variability. It could be partly due to insufficient dose of sleep manipulation in the current study. It might be also because the sample recruited in the current study was healthy sleepers, which could be different from clinical populations.

In general, both groups showed improvements in sleep quality throughout the manipulation period, as indicated by more deep sleep

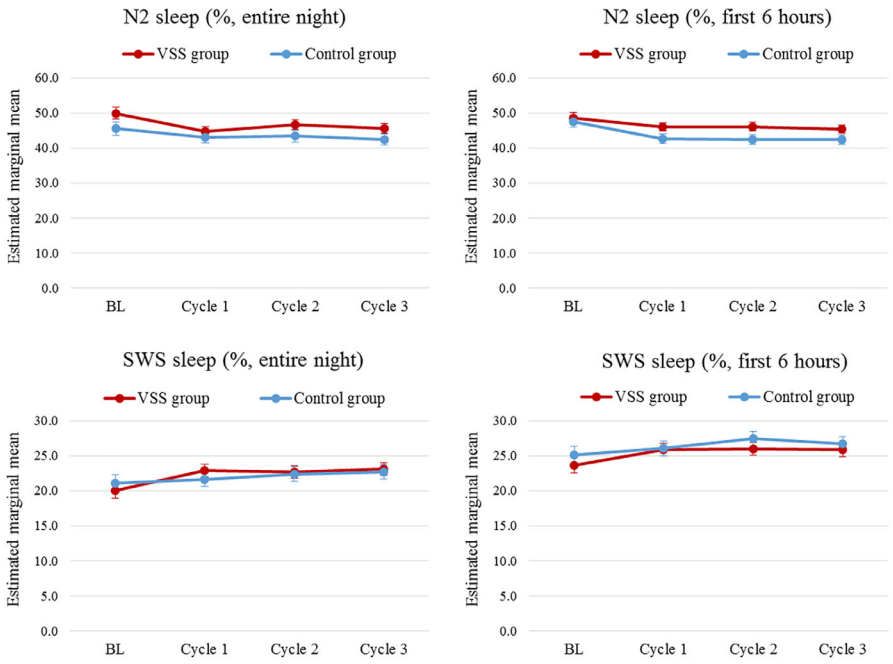

FIGURE 3 (Continued)

(i.e. SWS) and less light sleep (i.e. N2 sleep and beta spectrum power), which might suggest a gradual adaption to the laboratory environment by the study participants. Although it was unexpected that REM increased during the 1-week manipulation, it could also be an indicator for better sleep quality. It has been suggested that sufficient REM sleep at the end of sleep period is important for individuals to wake from sleep consciously (Kline et al., 2013). In addition, longer REM sleep duration could be related with better cognitive performances (Djonlagic et al., 2021), thus the increased REM sleep might be a reason for improved processing speed observed in the current study.

5.3 | Strength and limitations

This was a well-controlled experimental study on the effects of sleep variability on daytime functioning and nocturnal sleep, with the consideration of several potential confounding factors, including overall sleep duration and circadian factors. While this study primarily aimed to test the effects of experimentally induced day-to-day changes in sleep duration, the experimental condition might also reflect repeated cycles of acute sleep restriction followed by sleep compensation. Our results were similar to a previous study that incorporated an experimental protocol consisting of 3 weeks of 5 nights of sleep restriction (4 hr) followed by 2 nights of sleep recovery (8 hr), in which self-perceived recovery of daytime functioning from sleep loss was found (i.e. less increase in sleepiness) while the elevated physiological stress responses persisted (e.g. HPA axis functioning; Simpson et al., 2016).

Due to the nature of the study design, there were some limitations in generalizing the laboratory results to the real-world scenario. The study sample was highly selected (i.e. well-educated, healthy young adults) so the findings might not be able to generalize to other populations. In this study, we only adopted the behavioural tasks measuring the cognitive abilities, and some higher-level cognitive functioning (e.g. planning and decision making) was not assessed. It is possible that sleep variability could lead to alternation of brain activities, despite no change at the behavioural level. Therefore, future studies are suggested to explore the impacts of sleep variability on other specific domains of cognitive functioning and incorporate advanced brain imaging techniques to further explore the changes in brain activity in response to sleep variability. In addition, the current study only included 1 week of manipulation of sleep schedule. Such an experimental manipulation might only induce a mild degree of sleep variability within a limited time. Future studies may consider examining the dose–effect of sleep variability (i.e. by changing the severity/intensity and length of the exposure to sleep variability) on health-related outcomes. Although this was a well-controlled experimental study, it remained difficult to delineate all the related factors under the manipulation of sleep duration (e.g. accumulated sleep debt, sleep drive, circadian factors). For example, whilst the present study tried to minimize the effect of circadian shift in the experimental protocol and the differences in the first 6 hr of sleep were specifically examined, it was still possible that sleep architecture might be affected during different time periods due to unintended circadian factors. Despite our efforts to refine the experimental design and statistical plans of testing a priori hypotheses, the findings should be interpreted with caution given the potential risk of increased type 1 error with a number of outcome variables in the current study. Furthermore, the technologist was not blinded to the experimental condition when scoring PSG. Nonetheless, manual scoring of sleep stages

was compared with automatic scoring algorithm and showed an agreement over 85%.

5.4 | Implications and conclusion

The present study showed that experimentally induced sleep duration variability led to increased daytime sleepiness and elevated negative mood in the evenings among healthy young adults, albite limited impacts on cognitive functioning and nocturnal sleep. Our findings might have implications for understanding the links between sleep variability, insomnia and mood symptoms, given that individuals with sleep disturbances (e.g. circadian rhythm disorders and insomnia) and mental health problems (e.g. depression, bipolar disorders) often reported increased sleep variability (Burgess et al., 2017; Buysse et al., 2010; Geoffroy et al., 2014; Suh et al., 2012; Yokomaku et al., 2008). Our results also highlighted the need to address sleep variability to improve daytime functioning, especially in those at-risk individuals.

AUTHOR CONTRIBUTIONS

Wanqi sun: Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; writing – original draft; writing – review and editing. **Forrest Tin Wai Cheung:** Conceptualization; data curation; investigation; methodology; project administration; writing – review and editing. **Ngan Yin Chan:** Conceptualization; investigation; methodology; writing – review and editing. **Jihui Zhang:** Conceptualization; investigation; methodology; supervision; writing – review and editing. **Joey Wing Yan Chan:** Conceptualization; investigation; methodology; resources; writing – review and editing. **Kate Ching Ching Chan:** Conceptualization; investigation; methodology; writing – review and editing. **Yun Kwok Wing:** Conceptualization; investigation; methodology; resources; supervision; writing – review and editing. **Shirley Xin Li:** Conceptualization; formal analysis; funding acquisition; investigation; methodology; project administration; resources; supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors do not have any conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

ORCID

Yun Kwok Wing https://orcid.org/0000-0002-5745-5474
Shirley Xin Li https://orcid.org/0000-0002-7085-1850

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