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EEG and behavioural correlates of mild sleep deprivation and vigilance

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HIGHLIGHTS

- Mild, acute sleep restriction has surprisingly robust, negative consequences to sustained vigilance.
- Alpha burst activity is a sensitive electrophysiological index of drowsiness.
- These findings have direct implications for scenarios demanding sustained vigilance.

ABSTRACT

Objective: The current study investigated the behavioral, cognitive, and electrophysiological impact of mild (only a few hours) and acute (one night) sleep loss via simultaneously recorded behavioural and physiological measures of vigilance.
Methods: Participants (N = 23) came into the lab for two testing days where their brain activity and vigilance were recorded and assessed. The night before the testing session, participants either slept from 12am to 9am (Normally Rested), or from 1am to 6am (Sleep Restriction).
Results: Vigilance was reduced and sleepiness was increased in the Sleep Restricted *vs.* Normally Rested condition, and this was exacerbated over the course of performing the vigilance task. As well, sleep restriction resulted in more intense alpha bursts. Lastly, EEG spectral power differed in Sleep Restricted vs. Normally Rested conditions as sleep onset progressed, particularly for frequencies reflecting arousal (*e.g.*, delta, alpha, beta).
Conclusions: The findings of this study suggest that only one night of mild sleep loss significantly increases sleepiness and, importantly, reduces vigilance. In addition, this sleep loss has a clear impact on the physiology of the brain in ways that reflect reduced arousal.
Significance: Understanding the neural correlates and cognitive processes associated with loss of sleep may lead to important advancements in identifying and preventing deleterious or potentially dangerous, sleep-related lapses in vigilance.

1. Introduction

Not only has the average sleep duration of adults decreased by ~20% over the last century (Roth, 1995), but more than 30% of adults report sleeping less than the recommended 7–9 h per night (Foundation, 2013; Hafner et al., 2016). The impact of chronic sleep loss on behavioural and cognitive function is well-documented (Alhola and Polo-Kantola, 2007; Goel et al., 2009; Killgore, 2010; Hafner et al., 2016; Krause et al., 2017). Likewise, the impact of severe and acute sleep deprivation on daytime functioning is also rel-

Abbreviations: PVT, psychomotor vigilance test; SSS, Stanford Sleepiness Scale; EEG, electroencephalography; EOG, electro-oculogram; EMG, electromyogram; RT, reaction time; FFT, Fast Fourier Transform.

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atively well-understood (Killgore, 2010; Ftouni et al., 2013; Krause et al., 2017). At the extreme, severe and chronic sleep deprivation are considered dangerous, and potentially life-threatening (for a review, see Tobaldini et al., 2017). In contrast, the behavioural, cognitive, and physiological consequences of mild and acute sleep loss (*e.g.,* only a couple of hours, for only a single night) have received relatively little attention. This is surprising, given that this is a commonplace form of sleep loss; one that many around the world reluctantly have to endure following the clock change when we "spring forward" from standard time to daylight savings time (Smith, 2016). This may, in part, be due to the fact that mild, acute sleep loss is generally thought to be innocuous, despite research suggesting that small amounts of sleep loss can negatively affect performance (Cote et al., 2009; Anderson and Horne, 2013), emotional processing (Lustig et al., 2018), as well as visual attention (Kuo et al., 2019), behavioral preparedness and responding (Stojanoski et al., 2019). Furthermore, compared to more extreme forms of sleep loss (*i.e.*, sleep deprivation), mild and acute sleep restriction has much higher ecological validity, and has direct implications for any situation that requires continuous attention during a monotonous task (*e.g.*, long highway drives, etc.). Therefore, a better understanding of cognitive processes and neural markers related to mild and acute sleep loss may make possible important advancements in identifying and mitigating situations in which lapses in vigilance are potentially dangerous (*e.g.,* when driving a vehicle, workplace safety, etc.) or when sustained vigilance is required or advantageous (e.g., classroom settings).

The consequences of mild and acute sleep loss on factors that impact daytime performance such as vigilance, sleepiness, and arousal can be measured both *behaviourally* and *physiologically*. Behaviourally, vigilance can be measured objectively (*e.g.,* Psychomotor Vigilance Task (PVT); Dinges and Powell, 1985) and sleepiness, subjectively (*e.g.,* Stanford Sleepiness Scale (SSS); MacLean et al., 1992). The PVT is a highly reliable measure of sustained vigilance in the face of a long and monotonous task. The PVT requires the individual to attend to a stimulus and respond, when prompted, as quickly as possible. The SSS is a one-item self-report rating scale that measures an individual's level of sleepiness at a given point in time. Physiologically, arousal can be measured by means of electroencephalography (EEG), a temporally sensitive, non-invasive method for recording electrical activity of the brain. This activity has characteristic frequency ranges which have distinct spatial distributions and are associated with different states of brain functioning that can reflect arousal-related activity. For example, increases in posterior alpha activity (8–12 Hz) are often associated with drowsiness. Increases in frontal delta activity (0.5–4 Hz) are associated with reduced information processing. In addition, localized decreases in frontal beta activity (12.5–30 Hz) are often associated with active concentration and active, busy, or anxious thinking, and are associated with increased arousal (Blake et al., 1939; Dustman et al., 1962; Ogilvie et al., 1991; Klimesch et al., 1998; Aeschbach et al., 1999; Klimesch, 1999; Cajochen et al., 2000; Ogilvie, 2001; Harmony, 2013). Thus, these EEG changes are potential electrophysiological markers of the impact of sleep loss on vigilance, sleepiness and arousal.

The consequences of mild sleep loss on objective vigilance are well documented (Jewett et al., 1999; Van Dongen et al., 2001; Belenky et al., 2003; Cote et al., 2009; Basner and Dinges, 2011; Anderson and Horne, 2013; Stojanoski et al., 2019). However, the effect that mild, acute sleep loss has on the electrophysiological markers of reduced vigilance is relatively unknown. For example, Cote et al. (2009) examined how mild (3 or 5 h of sleep) and acute (1 or 2 nights) sleep restriction impacted the EEG power spectra, identifying that even a single night of sleep restriction caused performance deficits and EEG slowing. Similarly, our group (Stojanoski et al., 2019) examined the impact of mild and acute sleep restriction (5 h of sleep for 1 night) on visual processing and behavioural responding. It was found that even one night of mild sleep restriction reduced processing capacity for decision making. Event-related EEG brain responses for both motor preparation and motor execution were reduced, negatively impacting vigilance (Stojanoski et al., 2019). In addition, the few studies that have recorded EEG simultaneously during vigilance testing (for a review, see; Hudson et al., 2020), have only done so using severe, acute sleep deprivation paradigms (*e.g.,* >24 h of continuous wakefulness; Corsi-Cabrera et al., 1996; Belenky et al., 2003; Caldwell et al., 2003; Van Dongen et al., 2003; Banks and Dinges, 2007; Ftouni et al., 2013; Schulze et al., 2013), chronic sleep restriction conditions (Cote et al., 2008), or testing when sleep pressure is greatest, during the nocturnal sleep period (Hoedlmoser et al., 2011). However, these types of sleep deprivation situations are rare outside of laboratory settings, or only occur in extreme conditions (*e.g.*, shift work, trans-meridian flights, etc.). Thus, despite mild and acute sleep loss being more widespread and ecologically valid, the impact this type of sleep loss has on behavioural and cognitive processing during tasks that require sustained vigilance remains unclear.

Here, we simultaneously recorded EEG during a monotonous, sustained attention task (the PVT) for a prolonged period during the daytime following one night of mild sleep restriction, to better understand the behavioral, cognitive, and neural consequences resulting from mild and acute sleep loss. We investigated both behavioural (objective and subjective) and physiological (*i.e.,* EEG) measures of daytime performance in mildly sleep restricted (*i.e.*, 5 h sleep opportunity; "Sleep Restriction" condition) *vs*. normal sleep (*i.e.*, 9 h sleep opportunity; "Normally Rested" condition) conditions in a repeated-measures design. We provided participants a slightly longer sleep opportunity than the "typical" ~8 h of sleep in the Normally Rested condition, to ensure participants were well-rested (but within recommended, normal sleep amounts for that age group; Hirshkowitz et al., 2015). This also ensured that participants who normally sleep slightly longer than 8 h/night, did not experience mild sleep restriction and were well satiated. We predicted that: (**1**) there would be an increase in (**a**) objective and (**b**) subjective sleepiness following sleep restriction; (**2**) throughout the testing session, participants would show an increasing number of drowsy-related visually-scored EEG characteristics associated with deeper/later sleep onset (*e.g.*, Hori Stages 3–9) and fewer drowsy-related EEG characteristics of lighter/early sleep onset (*e.g.*, Hori stages 1 & 2) which would change as a function of time while performing the sustained vigilance task; (**3**) EEG characteristics of drowsiness (*e.g.,* occipital alpha bursts) would be increased in the Sleep Restricted *vs.* the Normally Rested condition; and finally, (**4**) alertness-related EEG spectral characteristics would increase for frontal delta and occipital alpha, and decrease for frontal beta in Sleep Restriction *vs*. Normally-Rested conditions.

2. Methods

Findings from this data set, using many of the same methodological details, including participant information, data collection procedures, and data processing steps have previously been published in Stojanoski et al. (2019).

2.1. Participants

All participants were between 20 and 35 years of age. Participants were screened via a telephone interview to exclude participants whose sleep schedules were outside the hours of 10:00 PM to 9:00 AM and those obtaining <7 or >9 h of sleep/night (*i.e.*, within the normal, recommended range of sleep for this age

group). The telephone interview was also used to screen out participants who were left-handed, had any hand mobility problems, worked shift work, used medications known to affect sleep, considered themselves a "smoker", consumed >2 caffeinated beverages/-day, or consumed >7 alcoholic beverages/week, or had a history of chronic pain, seizures or head injury. Participants were required to refrain from the use of any recreational drugs (including but not limited to nicotine and alcohol) at least three days prior to, and throughout the duration of the study. Participants noted these behaviours in their sleep log, which was confirmed by the researcher on each testing day. During the study, participants consumed no more than a single caffeinated beverage per day upon awakening and refrained from consuming nicotine or other stimulants. Actigraphy and sleep logs, filled out by the participant, were used to confirm the participants' sleep and activity cycles throughout the study. Participants also completed the Sleep Disorders Questionnaire (Douglass et al., 1994), as well as the Beck Depression (Beck et al., 1988b) and Anxiety Inventories (Beck et al., 1988a) to rule out those with signs of depression and/or anxiety and ensure normal sleep-wake patterns.

A total of 26 participants met inclusion criteria for this study. From these, three participants were excluded because of missing or poor-quality behavioural data, and an additional two participants were excluded from the EEG analysis because of poor quality EEG recordings or excessive movement artifacts. Therefore, the behavioural analyses included a total of 23 participants (18 females, mean age ± SD = 22 ± 3 years), and EEG analyses included 21 participants (17 females, mean age ± SD = 22 ± 3 years).

2.2. Ethics statement

This study was approved by Western University's Health Science Research Ethics Board. Participants were given a letter with details of the study, provided informed consent and were financially compensated for their participation.

2.3. Behavioural measures

2.3.1. Psychomotor vigilance task

The PVT (Dinges and Powell, 1985) is a simple, visual reaction time test used to assess objective vigilance in the face of a long and monotonous task. Participants were instructed to focus their attention on an on-screen plus sign '+' (*i.e.,* the fixation point), and press the space bar on a computer keyboard (*i.e.*, the "response" button) as quickly as possible upon the appearance of a numerical timer (*i.e.*, the "stimulus"). To make the timing of each trial unpredictable, the onset of the stimulus was presented on-screen at random intervals ranging between 2 and 10 s in duration. Participants performed 6 blocks of 100 trials of the PVT, which took approximately 70 minutes to complete, in total. EEG was continuously recorded throughout the PVT testing session (see below for methodological details on EEG recording and analysis). This length of testing was necessary to examine the impact of sleep restriction on sustained vigilance in the face of a very long and monotonous task. Consistent with the extant literature (Dinges and Powell, 1985; Basner and Dinges, 2011; Yun et al., 2015), any trials in which the participant responded before the fixation cross appeared were considered "false reactions". Trials where reactions were longer than 1000 ms were considered "no response" trials. Both false reactions and no response trials were excluded from all subsequent analyses. Reaction times (RTs) greater than 500 ms, but less than 1000 ms, were included as valid reaction times but were also counted as "lapses" and analyzed separately. RT (ms) was inversely transformed to normalize the distribution of values for subsequent statistical analyses (Stojanoski et al., 2019; Dinges and Powell, 1985; Dinges et al., 1997; Jewett et al., 1999; Van Dongen et al., 2001; Basner and Dinges, 2011; Jongen et al., 2015; Yun et al., 2015). Also, consistent with previous behavioural studies (Stojanoski et al., 2019; Jewett et al., 1999; Lim and Dinges, 2008; Mollicone et al., 2010; Basner and Dinges, 2011), the visual PVT was used to assess the impact of mild and acute sleep restriction on cognitive and electrophysiological processes. The variables of interest for the PVT included the mean response speed, the number of lapses, the mean 10% fastest trials, and the mean 10% slowest trials in which a valid response occurred.

2.3.2. Stanford sleepiness scale

The Stanford Sleepiness Scale (SSS; MacLean et al., 1992) is a single-item scale used to determine the subjective sleepiness of the individual. The scale ratings vary from "Feeling active, vital, alert, or wide awake" to "Asleep". Participants rate how they felt when completing the questionnaire on a scale from 1 to 7. Scale ratings of 1, indicates peak alertness; 2–4, indicates a potential lack of sleep; and 5–7, a serious sleep debt. The variable of interest for the SSS is the single score reported by the participant.

2.4. Physiological measures & analysis

2.4.1. Electroencephalography

Electroencephalographic (EEG) recordings were obtained using a 32-channel Embla Titanium amplifier (Natus Medical Inc, Pleasanton, CA, USA) from 16 scalp derivations (EEG channels M1, M2, Fp1, Fp2, Fpz, F3, F4, Fz, C3, C4, Cz, P3, P4, Pz, O1, and O2) based on the international 10–20 electrode placement system (Jasper, 1958). EEG was digitized at 512 Hz, with an online high pass filter of 0.1 Hz. Referential EOG recordings were collected from electrodes placed on the outer canthus of the eyes (referenced to Fpz). In addition, a bipolar channel was used to record submental EMG activity. Following acquisition, EEG data were re-referenced offline to the averaged mastoid derivations (M1 and M2), and bandpass filtered between 0.3 Hz and 35 Hz in two passes using a zero phase, hamming-windowed sinc finite impulse response (FIR) filter implemented in EEGLAB (Delorme and Makeig, 2004). The EMG recording was bandpass filtered offline between 10 and 50 Hz in two passes using a zero phase, hamming-windowed sinc FIR filter. Eye movements were removed from the data using ICA decomposition. Movement artifacts were automatically detected using custom MATLAB (The Mathworks Inc., Natick, MA, United States) scripts, which employ a variance-based (*i.e.*, first-derivative) transformation of the EMG channels, then visually inspected by an expert to validate the automatic detection algorithm before being used to exclude EEG marked as movement from further analyses.

The EEG data recorded during the PVT testing session were visually scored offline by a single, expert registered polysomnographic technologist (RPSGT) with >20 years of research and clinical experience, in 5-second epochs continuously from the beginning to the end of the PVT testing session. EEG data were scored according to the Hori method of sleep onset stage scoring (Tanaka et al., 1996) and visually verified by a second expert, also with >20 years of sleep scoring experience. Prior to conducting sleep stage scoring, the technologist was first trained on the scoring method to be employed, then independently scored four separate stage scoring training files. Inter-rater reliability was established by comparing these scored files to the gold standard scoring conducted by an expert scorer. Any discrepancies were discussed in order to clarify any reasons for disagreement. This process was repeated until an inter-rater reliability of at least 90% was established. The Hori method categorizes sleep onset into 9 distinct sleep stages (termed "Hori Stage 1" to "Hori Stage 9"), each

characterized by unique electrophysiological signatures and markers (*e.g.*, H1 = alpha waves train, H2 = alpha wave intermittent >50%, H3 = alpha wave intermittent <50%, H4 = EEG flattening, H5 = ripples, H6-9 = sharp waves and spindles; see Fig. 1 for illustrative examples of EEG activity for each stage). The Hori method is intended to precisely identify the subtle changes that accompany the transition from wakefulness to sleep (Tanaka et al., 1996). Because the Hori stages were intended to characterize sleep onset only, and the majority of the EEG recording includes alert wake with eyes open, an additional stage, termed "active/alert wake", was used to describe when participants were alert and awake with eyes open (i.e., no alpha, desynchronized, low amplitude EEG characterized by beta activity, elevated EMG and eye blinks) according to standard guidelines (Iber et al., 2007).

2.4.2. Alpha burst analysis

Automatic alpha burst detection was performed on movement artifact-free, Hori scored 5-second epochs of EEG data using a previously validated method (Ray et al., 2015). Briefly, sleep phenomena in the frequency range of interest were automatically detected using a complex demodulation transformation of the EEG signal (see Ray et al., 2015 for a more detailed account). This approach was adapted to detect alpha bursts by setting the bandwidth to 8–12 Hz for O1 and O2 electrode derivations. Alpha burst detection was done using custom, EEGLAB-compatible (Delorme and Makeig, 2004) software written for MATLAB R2014a (The MathWorks Inc., Natick, MA, United States). To ensure accurate detection, alpha burst detection was visually verified by an expert RPSGT. The variables of interest extracted from this method include alpha burst activity (amplitude × duration) and number of bursts for each participant at the averaged O1-O2 derivation (the scalp locations where arousal-related alpha is maximal).

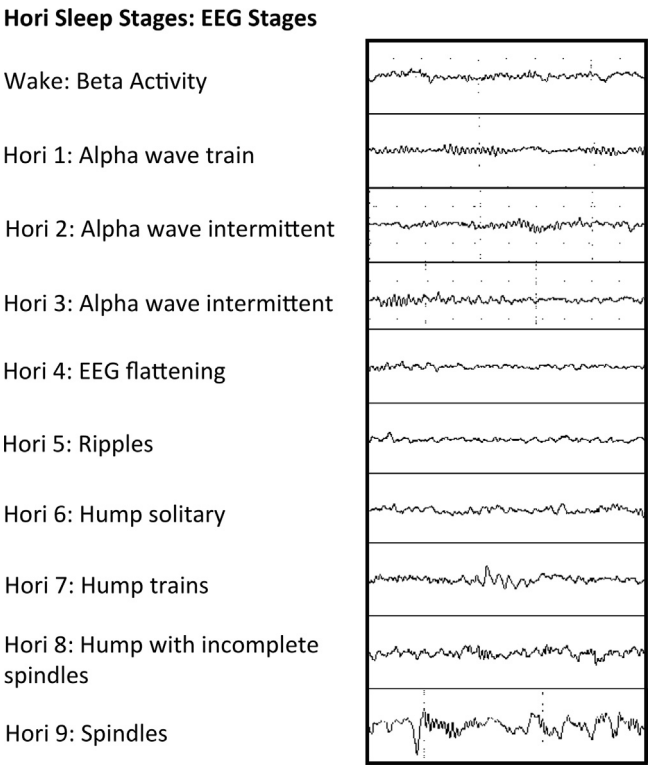

Fig. 1. Hori Sleep Stages: Representative examples of EEG data (5-sec epoch) of the unique electrophysiological signatures for each Hori sleep stage with corresponding conventional sleep stage descriptions.

2.4.3. Power spectral analysis

Power spectral analysis of the EEG data was done using Fast Fourier Transformation (FFT) techniques on active/alert wake data, as well as all sleep onset data, and data for each Hori stage 1–5 separately, at each electrode (note: there was insufficient data in H6-9 to be included in the analysis). FFT analyses were conducted on all recorded artifact-free EEG across the entire testing session using 2-second Hann windows, with a 75% overlap. Spectral power was then binned into five frequency ranges: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), sigma (12–16 Hz), and beta (16–35 Hz). Prior to statistical analyses, spectral power values were log transformed to normalize the distribution of scores.

2.5. Procedure

All participants who met the screening criteria *(see "Participants" section for details)* were required to come into the lab for two testing days (from ~12 pm to 3 pm) where their brain activity and vigilance were simultaneously recorded. On the night prior to the testing days, participants slept from 1 am to 6 am (*i.e.*, obtained 5 h of sleep in the Sleep Restriction condition), or from 12 am to 9 am (*i.e.*, obtained 9 h of sleep in the Normally Rested condition) in a repeated-measures design. The order of the sleep conditions was counter-balanced across participants. The 9-h sleep opportunity avoided the possibility of imposing a 1-hour sleep restriction for those individuals who habitually slept 8–9 h/night and is within recommended sleep amounts for this age group. Thus, this sleep period was considered to be a "normal" night of sleep. A minimum of 3 days occurred between testing days. Actigraphy and sleep logs were used to verify that each participant strictly adhered to the sleep timing prior to each testing day. Participants arrived at the sleep laboratory on each testing day at approximately 12:00 pm, for EEG setup and electrode application. Testing began at approximately 1:15 pm and lasted for, on average, approximately 70 min. For testing, participants were asked to complete six consecutive PVT sessions with 100 trials each, during which EEG was simultaneously and continuously recorded throughout the entire testing session (*see "Physiological Measures" section for more details*). In addition, participants completed the SSS a total of 7 times; once before each PVT session, and a final time after all PVT sessions were complete.

2.6. Statistical analyses

To investigate objective vigilance, a repeated measures ANOVA using sleep condition (Normally Rested, Sleep Restriction) × block (blocks 1–6) as factors was conducted on the inverse RTs from the PVT task. Similarly, a sleep condition (Normally Rested, Sleep Restriction) × block (blocks 1–7) ANOVA was used to investigate subjective sleepiness (*i.e.,* SSS scores) across blocks, as a function of sleep condition. Thirdly, sleep condition (Normally Rested, Sleep Restriction) × block (blocks 1–6) ANOVAs were conducted separately for each Hori stage to investigate changes in alertness (*i.e.,* Hori stages 1–5).

Hori stages 6–9 were excluded from all analyses as a result of inadequate amounts of data. See Fig. 1 for examples of EEG traces for each Hori sleep stage.

To explore the effects of sleep restriction on alpha burst activity during a sustained vigilance task, a two-way, repeated-measures ANOVA was conducted with sleep condition (Normally Rested, Sleep Restriction) and Hori stage (H1–H5) as factors for both alpha burst activity (duration × amplitude) and number separately. Paired samples t-tests were used to follow-up significant main effects of sleep condition across Hori stages 1–5.

To explore the effects of mild, acute sleep restriction on spectral power in the EEG signal during a sustained vigilance task, a three-

way repeated-measures ANOVA was conducted on spectral power in each frequency band (delta, theta, alpha, sigma, beta), separately, with electrode site (F3, F4, Fz, C3, C4, Cz, Fp1, Fp2, Fpz, P3, P4, Pz, O1, and O2), arousal (Active/Alert Wake vs. Sleep Onset), and sleep condition (Normally Rested, Sleep Restriction) as factors. This analysis was used to establish if statistically significant effects were observed between sleep conditions. To further characterize the more nuanced features of the sleep onset process, a second series of ANOVAs were conducted, on spectral power in each frequency band, with electrode site, sleep condition, and Hori stage (Active/Alert wake and Stages H1-H5) as factors. Statistically significant findings from the spectral analysis were investigated further to determine where the sleep condition-related differences in spectral power were distributed across the scalp. For this, a more descriptive approach was employed to follow-up statistically significant ANOVA results to assess the topographic pattern of these effects. Each significant finding reported was followed up using paired t-tests. To this end, for the stages in which spectral power differed between sleep conditions, statistically significant results ($p < 0.05$) were reported for each electrode location and used to display thresholded t-score values between conditions in order to visually illustrate the spatial distribution of power spectral differences.

For all statistical analyses, tests of homogeneity/sphericity were conducted when applicable, and corrected values were reported when necessary.

3. Results

3.1. Behavioural results

3.1.1. Objective vigilance: psychomotor vigilance task (PVT)

In the Sleep Restriction condition, participants produced significantly more lapses than they did in the Normally Rested condition ($F(1, 22) = 17.06$, $p < 0.001$, $\eta^2 = 0.44$) in overall PVT performance (Table 1). Also, participants produced an increasing number of lapses over the course of the 6 PVT blocks in both sleep conditions ($F(5, 110) = 14.40$, $p < 0.001$, $\eta^2 = 0.40$) (Fig. 2**a**). There was no significant condition by block interaction.

Overall, response speed was significantly faster in the Normally Rested than in the Sleep Restriction condition ($F(1, 22) = 9.02$, $p = 0.007$, $\eta^2 = 0.30$) and became slower across the 6 blocks ($F(5, 110) = 8.12$, $p < 0.001$, $\eta^2 = 0.27$) regardless of sleep condition (Fig. 2**b**). Response speed for the slowest responses also showed a similar pattern, whereby responses in the Sleep Restriction condition were significantly slower than in the Normally Rested condition ($F(1, 22) = 9.10$, $p = 0.006$, $\eta^2 = 0.30$). Response speed of the slowest responses slowed in both conditions over the course of the 6 blocks of trials ($F(5, 110) = 6.42$, $p < 0.001$, $\eta^2 = 0.23$) (Fig. 2**c**). Sleep condition did not significantly affect response speed for the fastest responses ($F(1, 22) = 0.02$, $p = 0.886$, $\eta^2 < 0.01$), but again, performance slowed over the course of the blocks of trials ($F(5, 110) = 2.34$, $p = 0.047$, $\eta^2 = 0.10$) (Fig. 2**d**). No condition by block interactions were significant.

Table 1
Overall Psychomotor Vigilance Task (PVT) performance and Standford Sleepiness Scale (SSS) scores (mean over blocks of trials) in the normal sleep (Normally Rested) and sleep restriction (Sleep Restriction) conditions.

	Normally Rested		Sleep Restriction	
	M	SD	M	SD
SSS	3.54	0.73	4.43*	0.66
Number of lapses	39.38	25.10	47.28***	25.41
Mean response speed	0.49	0.09	0.52**	0.10
Mean fastest	0.38	0.04	0.38	0.05
Mean slowest	0.76	0.31	0.90*	0.49

Note: Speed expressed as reaction time (s); *, **, *** indicate significant differences between the Normally Rested and Sleep Restriction conditions at $p < 0.05$, 0.01, & 0.001 respectively.

3.1.2. Subjective sleepiness: Stanford sleepiness scale (SSS)

Participants reported significantly higher subjective sleepiness (Table 1) in the Sleep Restriction condition compared to the Normally Rested condition ($F(1, 22) = 6.20$, $p = 0.021$, $\eta^2 = 0.22$). Sleepiness increased over the course of the testing session ($F(6, 132) = 14.61$, $p < 0.001$, $\eta^2 = 0.40$) for both sleep conditions (Fig. 3). There was no significant condition by block interaction.

3.2. Physiological results

3.2.1. EEG architecture

No main effect of sleep condition was found for any Hori stage, but a significant main effect of block was found for H3 ($F(5, 110) = 3.36$, $p < 0.01$, $\eta^2 = 0.13$) and H4 ($F(5, 110) = 2.73$, $p < 0.05$, $\eta^2 = 0.11$), whereby participants spent increasingly less time in H3 and more time spent in H4 in the later PVT blocks, regardless of sleep condition. This suggests that over the course of the PVT testing session, as predicted, EEG associated with lighter Hori stages (H3) was reduced, and EEG associated with deeper Hori stages (H4) was increased, irrespective of sleep restriction condition (Table 2). The interaction between sleep condition and block was not statistically significant for any of the Hori stages (Fig. 4). Remarkably, individuals spent on average only 76.9% of the time in active/alert wakefulness, and correspondingly 23.1% of the time in some form of sleep microstate or sleep onset. Thus, suggesting that participants were not able to sustain a high level of vigilance in the face of a long, monotonous task.

3.2.2. Alpha bursts

Alpha burst activity (*i.e.*, strength/intensity) was significantly greater in the Sleep Restriction condition than in the Normally Rested condition irrespective of Hori Stage ($F(1, 20) = 6.19$, $p = 0.022$, $\eta^2 = 0.24$; Fig. 5), and differed significantly between Hori stages ($F(4,80) = 41.04$, $p < 0.001$, $\eta^2 = 0.67$). To further explore the nature of these effects, paired-samples t-tests were conducted to compare alpha burst variables (activity, number) between sleep conditions (Normally Rested, Sleep Restriction), for each Hori sleep stage. Participants showed significantly greater alpha burst activity in the Sleep Restriction condition compared to the Normally Rested condition during H1 ($t(20) = 2.14$, $p = 0.04$) and H2 ($t(20) = 2.30$, $p = 0.03$; Table 3). No other significant differences were found for alpha burst activity.

Although the number of alpha bursts differed between Hori stages ($F(4, 80)$, $p < 0.001$, $\eta^2 = 0.64$), there was no significant main effect of sleep condition ($F(1, 20) = 0.81$, $p = 0.379$, $\eta^2 = 0.04$). Taken together, these results indicate that there are not necessarily more alpha bursts when sleep restricted, but the bursts that do occur are stronger/more intense, overall, compared to when normally rested, regardless of Hori stage (Fig. 5).

3.2.3. EEG power spectral analysis

3.2.3.1. EEG characteristics in active/alert wake vs. sleep onset. Spectral power differed significantly across electrode site in all frequency bands (all $F > 2.25$, $p < 0.008$; Fig. 6). Power also differed between periods of active/alert wakefulness and sleep onset (all $F > 8.23$, $p < 0.010$). In both delta and theta bands, power differed between the normally rested and sleep restriction conditions ($Fs > 4.34$, $p < 0.050$), indicating that mild, acute sleep restriction had an effect on low frequency neural oscillations.

In addition, the spatial distribution of spectral power across electrodes differed between states of active/alert wakefulness

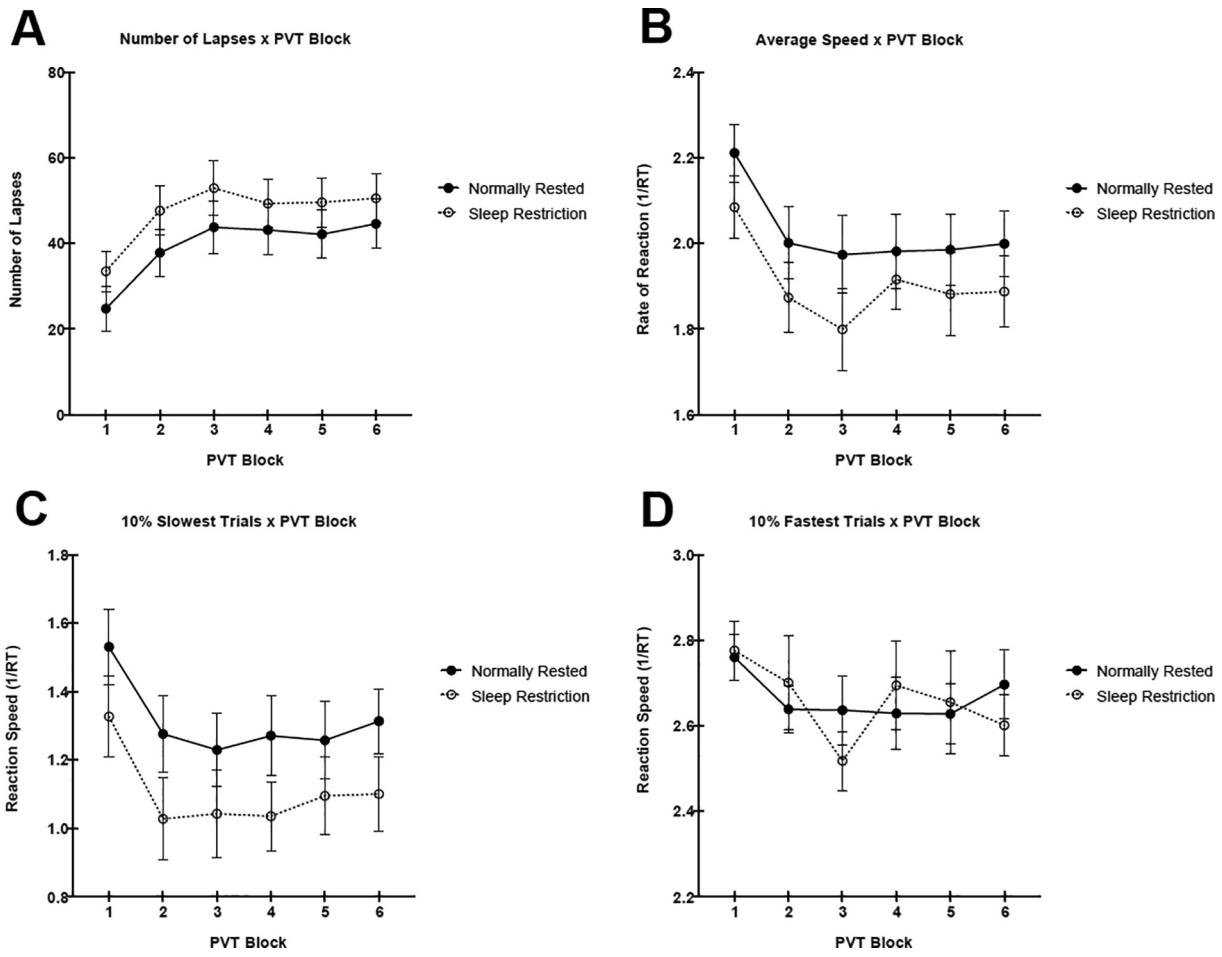

Fig. 2. PVT Performance: Mean (±SE) Psychomotor Vigilance Task (PVT) performance in both sleep-related conditions; normally rested condition (Normally Rested; closed marker with the solid line) and sleep restriction condition (Sleep Restriction; open marker with the dotted line) by 6 blocks. **A:** The number of lapses per block. **B:** The average speed of all trials per block (1/RT × 1000). **C:** The average speed of the 10 slowest trials per block (1/RT × 1000). **D:** The average speed of the 10 fastest trials per block (1/RT × 1000).

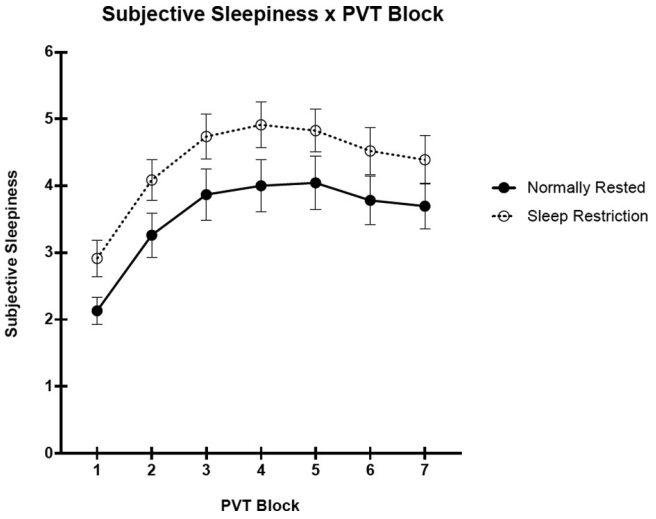

Fig. 3. Subjective Sleepiness: Mean (±SE) Stanford Sleepiness Scale (SSS) scores in both sleep related conditions; normal sleep condition (Normally Rested; closed marker with the solid line) and sleep restriction condition (Sleep Restriction; open marker with the dotted line) measured prior to the start of each Psychomotor Vigilance Task (PVT) blocks and after the last PVT block.

and sleep onset ($F > 6.71$, $p < 0.001$), in a two-way interaction between electrode and arousal in the delta, theta, and beta bands. Importantly, spectral power differences between active/alert wakefulness and sleep onset were significantly affected by mild, acute sleep restriction ($F(1, 20) = 5.81$, $p = 0.026$), in a two-way interaction between arousal and sleep condition in the alpha band. Lastly, spectral power differed significantly, not only across electrode sites, but such that the pattern of differences at each site differed as a function of active/alert wakefulness and sleep onset ($F(13, 260) = 3.54$, $p < 0.001$) in a three-way interaction between electrode site, arousal, and sleep condition in the beta band.

Follow-up simple effects analyses of sleep condition were performed to further explore the significant main effects and interactions reported above, and in particular, to describe the electrode where the effects were maximal. The effect of sleep restriction was maximal in all frequency bands during sleep onset. In the delta band, sleep restriction increased power maximally in occipital regions at O2 ($t(20) = 3.29$, $p = 0.004$). In the theta band sleep restriction increased power maximally frontally at F4 ($t(20) = 2.87$, $p = 0.002$). In the alpha band, sleep restriction maximally increased power occipitally at O2 ($t(20) = 3.15$, $p = 0.005$). In the beta band, sleep restriction maximally decreased power frontally at F3 ($t(20) = -2.86$, $p = 0.010$).

Table 2
Overall average time awake and in stages of sleep onset during Psychomotor Vigilance Task (PVT) blocks in the Normally Rested (NR) and Sleep Restricted (SR) conditions.

% total (# epochs)		PVT Block											
		1		2		3		4		5		6	
		NR	SR	NR	SR	NR	SR	NR	SR	NR	SR	NR	SR
Sleep	Onset	H5	0.54 (5.22)	0.91 (10.00)	0.51 (5.00)	0.90 (8.70)	0.80 (9.13)	1.34 (13.04)	0.87 (9.13)	0.45 (3.70)	0.95 (11.30)	1.18 (15.65)
		0.52 (5.87)	1.02 (12.17)										
	H4	0.21 (1.96)	0.07 (0.65)	0.81 (8.48)	0.37 (3.48)	0.27 (2.83)	0.94 (9.35)	0.28 (2.61)	0.83 (6.96)	0.09 (0.87)	0.49 (5.22)	0.07 (0.65)	0.37 (3.48)
	H3	2.16 (19.78)	2.13 (20.43)	3.15 (29.57)	4.58 (42.17)	2.50 (24.78)	4.51 (42.61)	2.48 (22.39)	3.07 (25.43)	1.45 (13.91)	2.32 (23.48)	1.48 (13.26)	2.20 (20.00)
	H2	4.70 (40.65)	5.66 (49.13)	5.38 (47.39)	5.81 (50.65)	4.79 (41.74)	6.41 (57.17)	5.84 (48.91)	6.39 (51.96)	4.65 (38.48)	5.36 (46.09)	4.54 (36.74)	4.59 (39.13)
H1		12.90 (111.96)	11.43 (97.61)	12.70 (106.96)	11.47 (97.83)	11.89 (99.57)	11.51 (98.48)	13.12 (106.52)	12.27 (100.00)	14.12 (113.48)	12.43 (100.65)	14.05 (111.09)	12.37 (99.57)
Active/Alert		78.87 (678.91)	77.79 (673.04)	76.08 (646.30)	74.64 (640.87)	79.13 (656.30)	73.65 (625.87)	76.01 (627.61)	76.56 (622.61)	76.78 (630.00)	76.33 (623.26)	78.97 (640.22)	78.16 (626.30)

Note: amount of time spent in each wakeful or sleep onset stage expressed as both a percent of the total time of the PVT block (above) and in mean number of 5 second epochs (below; in parentheses).

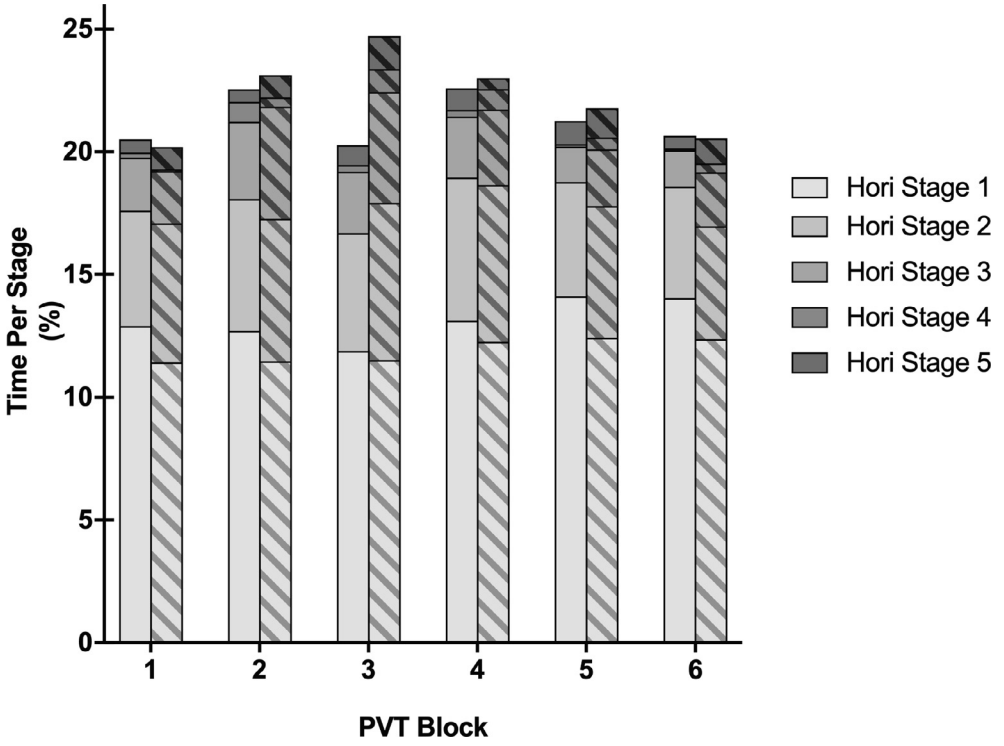

Fig. 4. Hori Stages: Percentage of time participants spent in H1 (alpha waves train), H2 (alpha wave intermittent > 50%), H3 (alpha wave intermittent < 50%), H4 (EEG flattening), and H5 (ripples) across the 6 Psychomotor Vigilance Task (PVT) blocks of trials as a function of sleep conditions; normal sleep condition (Normally Rested; solid bars) and sleep restriction condition (Sleep Restriction; striped bars).

3.2.3.2. EEG characteristics as a function of sleep onset stages. To further investigate the impact of sleep restriction on EEG characteristics systematically over the sleep onset process, using a more nuanced approach, we also analyzed the FFT results according to Hori sleep onset stages. Power differed significantly across electrode site in each frequency band (all $F(13, 260) > 2.09$, $p < 0.015$). Spectral power also differed significantly across Hori stages in each frequency band (all $F(5, 100) > 7.41$, $p < 0.001$). In all frequency bands except sigma ($F(1, 20) = 0.01$, $p = 0.939$), power differed between the normally rested and sleep restriction conditions, indicating that sleep restriction had an effect on EEG spectral power (all $F(1, 20) > 5.52$, $p < 0.029$).

The analyses also revealed that the spatial distribution of spectral power across electrodes differed as a function of Hori stage (all $F(65, 1300) > 1.75$, $p < 0.001$) in a two-way interaction between electrode site and Hori stage in all frequency bands. Lastly, spectral power differed significantly, not only across electrode sites, but such that the pattern of differences at each site differed as a function of Hori stage ($F(65, 1300) = 2.02$, $p < 0.001$) in a three-way interaction between electrode site, Hori stage, and sleep condition in the beta band.

Follow-up simple effects analyses of sleep condition were performed to further explore the significant main effects and interactions reported above, and in particular, to describe the electrode

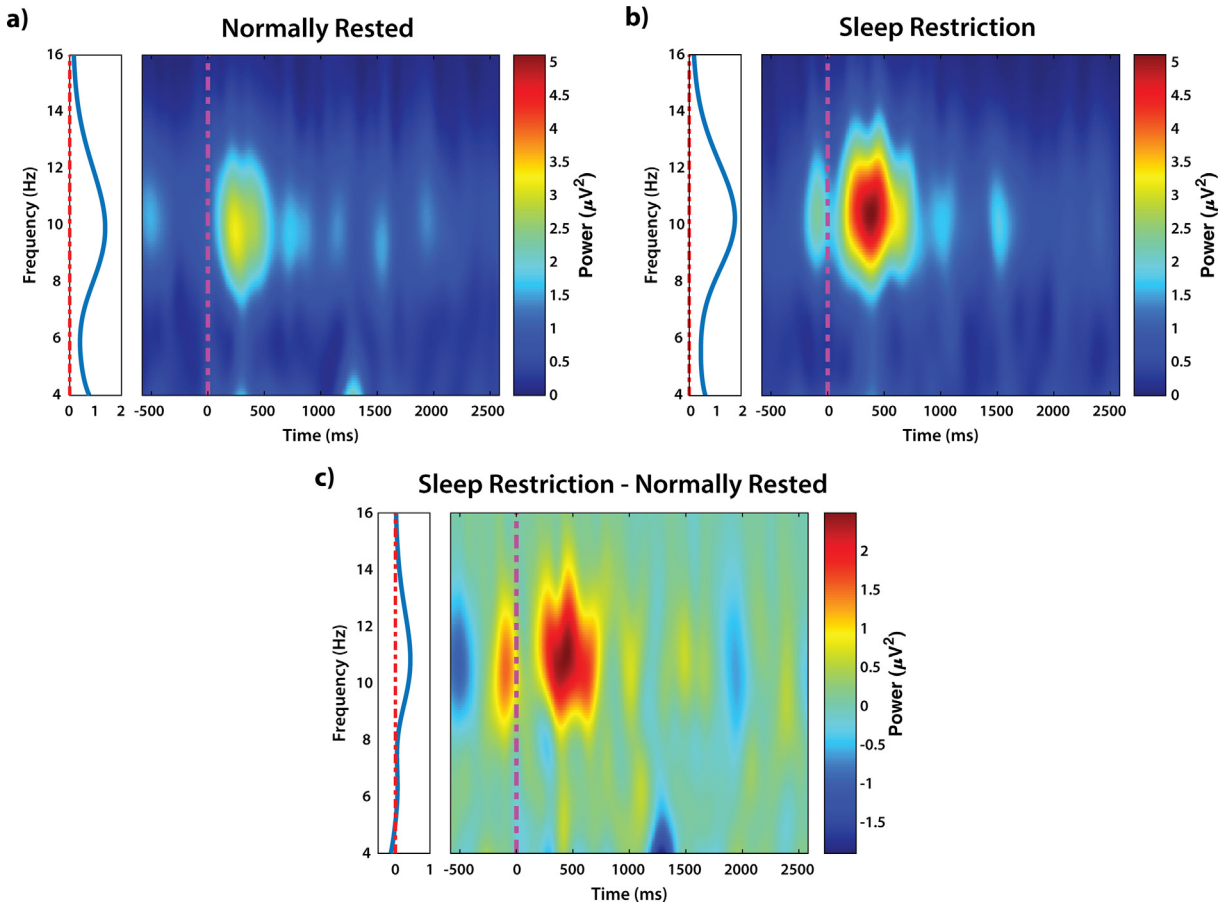

Fig. 5. EEG power time-locked to alpha bursts: Intensity of alpha burst activity averaged across all Hori Stages (H1-H5) in the **(A)** Normally Rested and **(B)** Sleep Restriction condition, and, **(C)** the difference between the Sleep Restriction – Normally Rested conditions. Each panel consists of a frequency spectrum (left, *n.b.,* x-axis of frequency spectrum denotes power), a time/frequency spectrogram (centre), and colour bar (right) indicating the power of the alpha burst. Spectrograms (centre) are time-locked to alpha burst onset (dashed magenta line). In panels (a) and (b) warm colours indicate larger spectral perturbations and cooler colours indicate smaller or no perturbations. In (a) and (b), dotted red line in frequency spectra (left) indicates zero perturbation. In panel (c) warm colours indicate Sleep Restriction > Normally Rested, whereas cool colours indicate normally rested > sleep restriction. In (c), dotted red line in frequency spectrum (left) indicates no difference in perturbation strength between Sleep Restriction and Normally Rested conditions. Note: scale of perturbation strength (right) in panel (c) is different than (a) and (b).

Table 3
Means and standard deviations for the significant differences identified between the alpha burst activity compared between conditions (Normally Rested, Sleep Restriction) by Hori sleep stage.

Alpha Burst Activity	Normally Rested		Sleep Restriction	
	M	SD	M	SD
All Sleep Onset stages	18.85	11.78	25.92	12.51
Hori stage 1	37.07	17.59	44.94*	16.63
Hori stage 2	29.13	20.98	42.38*	20.45

Note: * indicates significant differences between the Normally Rested and Sleep Restriction conditions at $p < 0.05$.

and sleep stages where the effects were maximal. The effect of sleep restriction was maximal in H5 (although see Fig. 6 for strikingly similar pattern in H3) for all frequency bands except sigma, which showed the largest difference between sleep condition in H3 (see Fig. 6). As expected, in the delta band during H5, sleep restriction increased power frontally at F4 ($t(20) = 2.36$, $p = 0.029$). In the theta band during H5, sleep restriction increased power at central areas, *e.g.*, at C4 ($t(20) = 2.58$, $p = 0.018$). As expected, sleep restriction increased power maximally at occipital regions (O2) in the alpha band during H5 ($t(20) = 2.53$, $p = 0.020$). Sleep restriction maximally increased power at Cz in the sigma band during H3 ($t(20) = 2.38$, $p = 0.027$). Lastly, as expected, sleep restriction maximally decreased power at Fp1 in beta band during H5 ($t(20) = -2.54$, $p = 0.012$).

4. Discussion

Here, we investigated the behavioral, cognitive and electrophysiological impact of mild and acute sleep loss (*i.e.*, only a couple of hours for only a single night) via *behavioural* (*i.e.,* objective and subjective) and *physiological* (*i.e.,* EEG) measures of vigilance. Chronic sleep loss has been the focus of the majority of past research investigating the behavioral and cognitive consequences of chronic sleep loss (Alhola and Polo-Kantola, 2007; Goel et al., 2009; Killgore, 2010; Hafner et al., 2016; Krause et al., 2017). In contrast, relatively little is known about the changes in brain activity that accompany reduced vigilance and increased sleepiness (*e.g.*, PVT performance and SSS scores, respectively) resulting from mild and acute sleep loss. Results of the current investigation revealed: (**1a**) reduced vigilance and, (**1b**) increased sleepiness in the Sleep Restricted condition compared to Normally Rested condition. These differences were exacerbated over the course of performing a long and monotonous sustained attention task; (**2**) participants in both Sleep Restriction and Normally Rested condi-

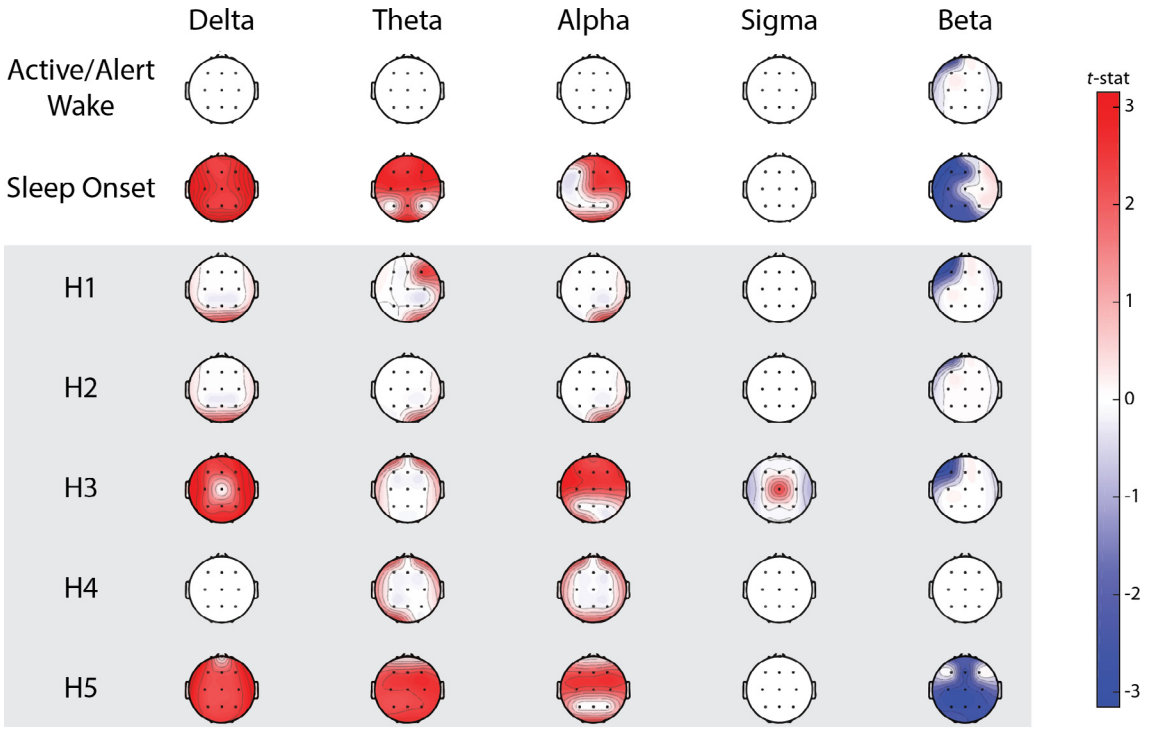

Fig. 6. Topography of EEG spectral power: Thresholded ($p < 0.05$) topographical maps of the difference in EEG frequency bands between Normally Rested and Sleep Restriction conditions across active/alert wake (top row), all sleep onset stages (second row), and sleep onset separated by Hori stage (H1-H5; grey area). Direction effects are displayed using t-scores. Red indicates sleep restriction > normally rested, whereas blue indicates sleep restriction < normally rested. White indicates no difference between conditions.

tions spent an increasing amount of time in the deeper Hori stages than in the lighter Hori stages over the course of performing the PVT task; (**3**) Sleep Restriction resulted in more alpha bursts than in the Normally Rested condition across Hori sleep stages; and lastly, (**4**) EEG spectral power differed between sleep conditions, particularly for frequencies that reflect arousal (*e.g.*, increased frontal delta, increased occipital alpha, and reduced frontal beta). Taken together, these results suggest that even a single night of sleep loss significantly reduces vigilance and increases sleepiness. In addition, this sleep loss has a clear impact on the physiology of the brain in multiple ways that reflect reduced arousal.

A large body of literature has established increased alpha activity as an electrophysiological correlate of drowsiness (Harrison and Horne, 1999; Connor et al., 2002; Simon et al., 2011) and demonstrated the relationship between sleep restriction and behavioural consequences to the outcomes of sustained attention/vigilance tasks (Jewett et al., 1999; Van Dongen et al., 2001; Belenky et al., 2003; Cote et al., 2009; Basner and Dinges, 2011; Stojanoski et al., 2019). However, these studies have mostly focussed on either chronic or severe sleep loss, which can occur, but is uncommon in day-to-day life. Although the PVT is a common cognitive task used in sleep deprivation studies, few have examined brain activity associated with the task, and only under conditions of extreme or chronic sleep restriction (Alhola and Polo-Kantola, 2007; Goel et al., 2009; Killgore, 2010; Ftouni et al., 2013; Hafner et al., 2016; Krause et al., 2017). Thus, ironically, the effect that more common mild or acute sleep restriction (*e.g.*, 2–3 h) has on the electrophysiological markers of arousal, and behaviour is relatively unknown.

As predicted, the results of the current study indicated more intense alpha burst activity in the Sleep Restriction than in the Normally Rested condition. The current findings of increased alpha burst activity in the sleep-restricted condition is consistent with previous literature suggesting that increased alpha activity is related to drowsiness (Harrison and Horne, 1999; Connor et al., 2002; Simon et al., 2011), inattention (Klimesch et al., 1998), and decreased task engagement (Bazanova and Vernon, 2014). Additionally, participants reported feeling sleepier (*i.e.*, higher SSS scores over time). Thus, these findings not only provide further evidence that alpha burst activity may be a useful electrophysiological marker of drowsiness, but also that even mild, acute sleep restriction is sufficient to alter brain activity in a meaningful way. Thus, the intensity of bursts of alpha activity may be a sensitive physiological marker of impairments in performance and cognition.

Here, RT was increased in the Sleep Restriction, compared to the Normally Rested condition across all blocks of the PVT task. These findings are in line with previous research on the cognitive effects of mild and acute sleep restriction, and suggest that even small amounts of sleep loss can have a significant impact on sustained vigilance (Stojanoski et al., 2019). Studies using total sleep deprivation have traditionally suggested that decreased performance on the PVT task after sleep restriction may reflect an inability to sustain attention to a stimulus (Hoedlmoser et al., 2011). However, recent investigations using mild and acute sleep restriction, more in line with the current study, have demonstrated that mild and acute sleep restriction does not affect early ERP components, which would reflect attentional differences (Stojanoski et al., 2019). Instead, the decreased performance after mild acute sleep restriction is thought to result from the attenuation of late stage ERP components associated with downstream information processing, such as decision making (Kok, 2001; Verleger et al., 2005; Stojanoski et al., 2019) and motor preparedness (Lutzenberger et al., 1985; Houlihan et al., 1994; Stojanoski et al., 2019).

Employing more ecologically valid testing paradigms, in combination with electrophysiological recording and vigilance testing (*e.g.,* using driving simulators; Arnedt et al., 2000, 2005; MacLean et al., 2003) may help to uncover performance impairments caused by sleep loss, and help identify neural markers of reduced vigi-

lance. Additionally, the effects of mild and acute sleep loss may not be consistent throughout the day. Thus, disentangling the interaction of sleep pressure and circadian rhythmicity on mild and acute sleep loss is crucial to understand when vigilance is maximally, or minimally, impacted by sleep loss. Here, we examined performance during the "mid-afternoon dip" to maximize the chance of detecting sleep-loss related effects on vigilance, information processing, and brain activity. It is possible that these effects would not be observed at other times of day. This possibility remains to be explored. In addition, to the best of our knowledge, the validity and inter-rater reliability of the Hori scoring method for sleep onset remains to be established. This work would be important to conduct in a future rigorous methodological validation study as the Hori scoring method is very detailed and requires highly-trained experts. Finally, by combining other brain imaging techniques (*e.g.*, fMRI, fNIRS, etc.) with vigilance testing, future studies could better understand the functional and neuroanatomical substrates impacted by sleep loss.

5. Conclusions

Although, mild and acute sleep loss is generally considered to be innocuous, it is very common, and as such, understanding the cognitive, behavioural, and electrophysiological impact of this type of ecologically valid sleep loss is important. The results of the present study demonstrate that mild, acute sleep restriction has surprisingly robust detrimental consequences on activities requiring sustained vigilance. This is reflected at behavioural, subjective and physiological levels. Additionally, our findings demonstrate that increased alpha burst intensity during impaired PVT performance may reflect a sensitive electrophysiological index of drowsiness. These findings have direct implications for a variety of scenarios, *e.g.*, workplace settings that require sustained vigilance for monitoring monotonous tasks, in classroom settings, for academic performance (*e.g.*, studying), and when driving (*e.g.*, long-haul highway driving, during morning/evening commutes, etc.). A better understanding of the neural correlates and cognitive processes associated with sleep loss may lead to important advances in identifying and preventing potentially deleterious or even dangerous, sleep-related lapses in vigilance in the workplace, classroom, or when reduced vigilance is life threatening (*e.g.*, driving).

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

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