SLEEPJ, 2019, 1–9

doi: 10.1093/sleep/zsy200
Advance Access Publication Date: 20 October 2018
Original Article

ORIGINAL ARTICLE

Sustained vigilance is negatively affected by mild and acute sleep loss reflected by reduced capacity for decision making, motor preparation, and execution

Bobby Stojanoski[1], Antoine Benoit[2,3], Nicholas Van Den Berg[2,3], Laura B. Ray[2], Adrian M. Owen[1], Ali Shahidi Zandi[4], Azhar Quddus[4], Felix J. E. Comeau[4] and Stuart M. Fogel[1–3,5,*]

[1]Brain and Mind Institute, Western University, London, N6A 5B7, Canada, [2]The Royal's Institute of Mental Health Research, Ottawa, K1Z 7K4, Canada, [3]School of Psychology, University of Ottawa, Ottawa, K1N 6N5, Canada, [4]Alcohol Countermeasures Systems Corp (ACS), Toronto, M9W 6J2, Canada and [5]University of Ottawa Brain and Mind Research Institute, Ottawa, K1H 8M5, Canada

Work Performed: 1151 Richmond St, Brain and Mind Institute, Western University, London, N6A 5B7, Canada

*Corresponding author. Stuart M. Fogel, School of Psychology, University of Ottawa, 136 Jean-Jacques Lussier, Ottawa, K1N 6N5, Canada. Email: sfogel@uottawa.ca.

Abstract

Study Objectives: The behavioral and cognitive consequences of severe sleep deprivation are well understood. Surprisingly, relatively little is known about the neural correlates of mild and acute sleep restriction on tasks that require sustained vigilance for prolonged periods of time during the day.

Methods and Results: Event-related potential (ERP) paradigms can reveal insight into the neural correlates underlying visual processing and behavioral responding that is impaired with reduced alertness, as a consequence of sleep loss. Here, we investigated the impact of reduced vigilance following at-home mild sleep restriction to better understand the associated behavioral consequences and changes in information processing revealed by ERPs. As expected, vigilance was reduced (e.g. increased lapses and response slowing) that increased over the course of the experiment in the "sleep restricted" (5 hr sleep) compared with the "sleep-extension" (9 hr sleep) condition. Corresponding to these lapses, we found decreased positivity of visually evoked potentials in the Sleep Restriction vs. Sleep Extension condition emerging from 316 to 449 ms, maximal over parietal/occipital cortex. We also investigated electrophysiological signs of motor-related processing by comparing lateralized readiness potentials (LRPs) and found reduced positivity of LRPs in the Sleep Restriction vs. Sleep Extension condition at 70–40 ms before, and 115–158 ms after a response was made.

Conclusions: These results suggest that even a single night of mild sleep restriction can negatively affect vigilance, reflected by reduced processing capacity for decision making, and dulls motor preparation and execution.

Statement of Significance

Even a small amount of sleep loss can affect daytime performance, particularly in the face of monotonous tasks. However, relatively little is known about the neural basis of mild and acute sleep restriction. We investigated the electrophysiological correlates and behavioral consequences of only 2 hr of at-home sleep restriction. This amount of sleep loss negatively affected sustained vigilance. Event-related brain potentials showed that sleep loss reduced processing capacity for decision making, motor preparation, and execution. These may serve as an electrophysiological index of drowsiness. Thus, even a seemingly innocuous amount of sleep loss could be hazardous in certain situations (e.g. following daylight savings, in the workplace, and long-haul highway driving). Future studies could employ functional neuroimaging techniques to better understand the brain regions and functional brain connectivity affected by only a small amount of sleep restriction.

Key words: sleep restriction; arousal; event-related potentials; vigilance; sleepiness; psychomotor

Submitted: 20 April, 2018; **Revised:** 24 July, 2018

Introduction

As a society, we live increasingly sleepless lives. It is more common than ever for individuals to have restricted time in bed or time asleep. Disordered sleep has reached epidemic proportions in North America. Over 3 million Canadians met the criteria for insomnia in a 2002 survey [1], and it is estimated that between 50 and 70 million Americans suffer from sleep disorders [2]. Up to 40% of the population reports daytime sleepiness or problems falling asleep [3]. In the last century, average sleep duration has decreased by ~20% [4]. On average, more than 30% of adults get less than 7 hr of the recommended 7–9 hr of sleep [5, 6]. Taken together, as a consequence, we spend less and less time sleeping.

There are real-world implications for a society that is less vigilant as a consequence of being chronically sleep deprived. Even mild, acute sleep loss, such as the 1 hr time shift to daylight savings time (DST) in the spring and fall, affects sleep quality and daytime vigilance. For example, on Monday following the spring DST change, when clocks are set forward 1 hr (i.e. sleep duration is reduced by 1 hr), the incidence of minor workplace accidents is significantly increased compared with a regular work day [7–13]. Similarly, a significant increase in fatal vehicle crashes has also been reported to occur for 1 week following the spring DST change [14].

There are two principle measures for detecting the degree to which an individual shows signs of reduced alertness. Firstly, behavioral measures of alertness can be assessed using subjective ratings like the Stanford Sleepiness Scale (SSS) [15], or objective measures of alertness using the Psychomotor Vigilance Task (PVT) [16], which requires an individual to attend to a stimulus and respond as quickly as possible. The latter requires sustained attention, visual processing, feature detection, and motor response preparation, initiation, and execution. All of which could putatively be negatively affected by sleep loss. The PVT has been shown to be a highly reliable measure of sustained vigilance [17, 18]. Secondly, physiological measures of brain activity recorded using electroencephalography (EEG) can be used to assess the physiological signs of reduced alertness. In addition to fluctuations in power in specific frequency bands (e.g. increases in α and decreases in β) associated with differing levels of alertness [19–26], event-related potentials (ERPs) can provide insight into impaired information processing as a consequence of reduced alertness, such as in the case of sleep loss. For instance, early ERP components such as the P1 and the N1, which reflect sensory processing, are modulated in response to various levels of alertness [25, 27, 28]. However, disruptions at these early stages can affect downstream processing at later stages. Components such as the P3 and the lateralized readiness potential (LRP) are useful in detecting changes in decision making, processing capacity [29], and can provide information about motor response preparation, initiation, and execution [30, 31] as a function of alertness.

A rich body of knowledge exists on the impact of mild sleep restriction on objective vigilance [17, 18, 32–35]. However, relatively little is known about the electrophysiological markers of reduced vigilance as a result of mild and acute sleep loss (a seemingly innocuous, but potentially hazardous scenario). One study by Cote et al. examined the impact of mild (3 or 5 hr of sleep) and acute (one or two nights) of sleep restriction, employing power spectral analyses to investigate the EEG frequency characteristics affected by sleep loss [35]. They found that only one night of sleep restriction led to performance deficits and EEG slowing. Another study observed a larger N170 in response to sad images, and a larger amplitude late positive potential to positive images after only one night of sleep mild restriction (4 hr sleep) [34], thus, suggesting that mild and acute sleep restriction affects emotional processing in terms of neural reactivity and attention, respectively. However, few studies have simultaneously recorded EEG with the PVT, and the few that have tested electrophysiological correlates of arousal in the face of sleep restriction did so under conditions of severe and acute sleep deprivation (e.g. >24 hr of continuous wakefulness) [33, 36–40], chronic sleep restriction [41], or by testing throughout the normal nocturnal sleep period, when sleep pressure is maximal [42]. This type of sleep deprivation is not very common outside of highly controlled laboratory situations or under extreme conditions (e.g. shift work or long, transmeridian flights). What remains unclear is how mild and acute sleep loss, which more accurately reflects the growing trend of sleep habits of modern society, affects cognitive and behavioral processing during tasks which demand sustained vigilance for prolonged periods of time.

Here, we aim to understand the behavioral, cognitive, and neural consequences of mild and acute sleep loss while performing a monotonous sustained attention task (PVT) for a prolonged period during the daytime. By employing simultaneous EEG and vigilance testing following one night of mild sleep restriction, we can better understand the physiological signs of reduced vigilance under such common conditions. Specifically, we investigated both the perceptual (visual-evoked responses) and behavioral (response-locked) ERPs during a sustained vigilance task in mildly sleep restricted vs. mildly sleep-extended conditions in a repeated-measures design. To be comparable to previous studies employing fine-grained sleep restriction protocols [33, 43–46], we employed a 5 hr sleep opportunity when compared with a 9 hr sleep opportunity, with the latter designed to ensure that participants were indeed well-rested by providing them a slightly longer sleep opportunity than the participants typical ~8 hr of sleep. This procedure also ensured that those participants who typically sleep 9 hr/night did not experience 1 hr of sleep restriction. PVT and SSS testing took place at the circadian trough (i.e. the “mid-afternoon dip”) with simultaneous EEG recording. Together, these techniques may reveal insight into the cognitive processes which are impaired with commonly experienced levels of sleep loss, and to identify the physiological signs which predict reduced vigilance with high temporal precision. We expected that (1) sleep restriction would lead to increased subjective and objective sleepiness, and (2) we hypothesized that this reduced vigilance would be reflected in both the visual-evoked potentials (VEPs) and motor response–locked LRP brain responses.

Methods

Participants

All participants were between the ages of 20 and 35. An initial telephone screening interview was used to exclude participants

for irregular sleep schedules (i.e. sleep beyond the recommended 7–9 hr of sleep, or outside the hours of 10:00 pm to 9:00 am) in order to include participants who slept, on average 8 hr, at normal times. Based on the results of the screening interview, participants were included only if they were right-handed, had no hand mobility problems, did not do shift work, did not use medications known to affect sleep, did not consume excessive nicotine (i.e. considered themselves a "non-smoker"), excessive caffeine (i.e. consumed <1–2 drinks/day) or excessive alcohol (i.e. consumed <7 drinks/week), or have a history of chronic pain, seizures, or head injury. Participants were required to abstain from drug use, nicotine, and alcohol at least 3 days prior to, and throughout the duration of the study, logged by the participant in their sleep journal and confirmed with the participant (by the researcher) prior to each testing session. Participants were instructed to consume no more than a single caffeinated beverage per day in the am, upon awakening. Participants' sleep routines throughout the study were confirmed by actigraphy and sleep diaries. To ensure normal sleep–wake patterns and rule out anxiety and depression, participants who met the initial screening were also asked to fill out the Sleep Disorders Questionnaire [47], as well as the Beck Depression [48] and Anxiety Inventories [49].

In total, 26 participants met the inclusion criteria for this study. Of these, six had either poor quality or missing EEG data, and two had too few artifact-free trials for analysis purposes. Thus, a total of eight participants were not included in the analyses. There were no demographic or sleep habit differences between those included and those with missing or poor quality data. Thus, 18 individuals (median age 21, range 20–26) were included (N = 14 females). Written and informed consent was obtained prior to participation in the study. This study was approved by the Western University Research Ethics Board.

Behavioral tasks

Stanford Sleepiness Scale

SSS was used as a subjective measure of sleepiness [15]. It is an 8-item scale that asks the individual to indicate the scale rating (from "Feeling active, vital, alert, or wide awake" to "Asleep") that best describes how they are feeling at that particular moment in time. A scale rating of 1 indicates that the individual is at peak alertness. A scale rating from 2 to 4 indicates that the individual could be suffering from a lack of sleep. A scale rating of 5 to 7 could indicate that they have a serious sleep debt and need more sleep, especially if this individual should be feeling alert at that time of day.

Psychomotor Vigilance Task

PVT was used as an objective measure of sustained vigilance [16]. The PVT is a simple, visual reaction time (RT) test, whereby participants are instructed to focus their gaze on a fixation point (e.g. an on-screen plus sign "+") and respond as quickly as possible, by pressing the space bar (i.e. the "response"), to the appearance of a numerical timer (i.e. the "stimulus") which was presented on-screen at a random interval between 2 and 10 s long. Participants performed six sessions of 100 trials, taking approximately 70 min in order to have a sufficient number of events for the analyses, but also importantly, to examine the impact of sleep restriction on extended periods of time when vigilance is required in the face of a monotonous task. These trials were also later categorized as the fastest 15% and slowest 15% RTs (see "ERP Analyses" section for details), as extreme responses have been found to be sensitive to sleep restriction [18]. Consistent with the extant literature [16, 18, 50], any RT < 100 ms were considered false starts, and RTs > 500 ms were considered lapses, which were excluded from subsequent behavioral and ERP analyses. As done in previous studies employing the PVT, RT (ms) was transformed using an inverse transformation [16–18, 32, 43, 50, 51]. Also, consistent with previous behavioral studies [17, 44, 52, 53], and the one previous ERP study employing the PVT and sleep restriction [54], we employed a visual PVT to assess the electrophysiological and cognitive processes affected by mild and acute sleep restriction. The choice of a visual PVT task was also made as this study was intended to serve as a "proof-of-concept" study to be adapted to other settings where visual attention is required, such as in a driving simulator environment.

Procedure

All participants were initially screened to verify that they met inclusion criteria (see "Participants" section for details). For the night prior to each testing day, all participants were instructed to either sleep from 1 am to 6 am (e.g. 5 hr of sleep in the "sleep-restricted" condition), or from 12 am to 9 am (e.g. 9 hr of sleep in the "sleep-extended" condition). We allowed a 9 hr sleep opportunity so that those who habitually tend sleep 9 hr per night would not experience 1 hr of sleep restriction (n.b., participants were young adults, and those that typically slept less than 7 hr, or more than 9 hr were initially screened out from participating in the study). One week occurred between testing conditions and conditions were counterbalanced across participants. Wrist actigraphy and sleep diaries were used to verify that participants adhered to the sleep timing instructions. On each testing day, participants arrived at the sleep laboratory at 12:00 pm. Upon arrival, electrodes were applied to their scalp and face. Testing began at 1:15 pm. Participants were asked to sit approximately 60 cm away from the testing computer screen. Six sessions (100 trials each) were completed in total where participants' brain activity was recorded via EEG (see "EEG Acquisition and Pre-Processing Procedures" section for details). Participants completed the SSS prior to the first PVT session and following each PVT session thereafter. The PVT testing session lasted, on average, approximately 1 hr 10 min.

EEG acquisition and preprocessing procedures

Data were acquired from a 24-channel electroencephalographic (EEG) Embla Titanium (Natus, Pleasanton, CA) amplifier system. EEG was recorded at a sampling rate of 512 Hz, with a high pass filter of 0.1 Hz and a low pass filter of 220 Hz. EEG (M1, M2, Fp1, Fp2, Fpz, F3, F4, Fz, C3, C4, Cz, P3, P4, Pz, O1, and O2) and electrooculogram (EOG; placed on the outer canthus of the eyes) referential recordings (reference Fpz) were re-referenced offline to the averaged mastoid derivations (M1 and M2), placed according

to the international 10–20 electrode placement system [55]. A submental electromyogram (EMG) channel was recorded as a bipolar derivation.

ERP analyses

For the event-related analyses, the data were segmented into 696 ms single trial "epochs" time-locked to the onset of each stimulus (100 ms prestimulus plus 596 ms poststimulus) for visual ERPs and segmented into 969 ms epochs time-locked to motor responses (500 ms preresponse plus 500 ms postresponse). Epochs were re-referenced to the average of both mastoids and baseline corrected. Trials containing movement artifacts were visually identified and excluded from analysis. Bad channels were visually identified, removed, and interpolated using EEGLAB [56]. There were a total of 16 EEG channels that were included in the analysis, but no more than three existing channels that were present but noisy were interpolated per data set. Six data sets required no interpolation, and eight required only one channel to be interpolated, three recordings required two channels to be interpolated, and only one data set had three channels interpolated. Blink and other ocular artifacts were subsequently removed using Independent Components Analysis implemented in EEGLAB. All ocular ICs were visually verified prior to correction. Epochs were first grouped into two vigilance states: (1) sleep-extended (Sleep Extension) and (2) sleep-restricted (Sleep Restriction), and further divided into fast and slow responses by selecting trials corresponding to reactions times in the fastest 15% and slowest 15%, respectively. (Extreme slowest and fastest responded are conventionally taken as the most extreme 10%; however, to have a sufficient number of trials ($N = 100$) for analysis purposes, here, we included the most extreme 15% of trials.) This produced four experimental conditions: (1) Sleep Extension, fastest responses; (2) Sleep Extension, slowest responses; (3) Sleep Restriction, fastest responses; and (4) Sleep Restriction, slowest responses. To examine whether these conditions differed in perceptual and/or motor processing, we analyzed VEPs and LRPs. All preprocessing steps were performed using MATLAB and EEGLAB.

ERPs are conventionally analyzed by identifying maximum and minimum peaks at a particular poststimulus time. The average of all activity in the prestimulus interval serves as a zero-voltage baseline from which each data point is measured against. However, this approach assumes that a cognitive process occurs within a highly selective time interval (e.g. at the latency of P1). In the present study, we examined all data points within the poststimulus epoch. Separate *t*-tests could then be run on each of the almost 1000 data points to compare the two conditions. This, of course, will result in inflating the chances of making Type I errors. Data were thus analyzed in two ways. First, the visually evoked responses were analyzed using the cluster-mass procedure [57] implemented in FieldTrip [58]. Briefly, this procedure compares adjacent spatiotemporal data points across conditions using *t*-tests. Single-subject ERP averages (across all trials and channels) elicited by each condition were compared using paired-samples *t*-tests. Although the *t*-test step is parametric, FieldTrip employs a secondary nonparametric clustering method to address the issue of multiple comparisons. Specifically, *t*-values of adjacent spatiotemporal points whose *p* values were less than 0.05 were clustered together by summating their *t*-values, and the largest such cluster was retained. A minimum of two neighboring electrodes had to pass this threshold to form a cluster, with neighborhood defined as other electrodes within a 4 cm radius. This entire procedure, i.e. calculation of *t*-values at each spatiotemporal point followed by clustering of adjacent *t*-values, was then repeated 1000 times, with recombination and randomized resampling of the ERP data before each repetition. This Monte Carlo method generated a nonparametric estimate of the *p* value representing the statistical significance of the originally identified cluster. This approach provides increased power relative to other corrections for multiple comparisons such as Bonferroni correction and false discovery rate. All analyses were two-tailed and included data from 100 ms prestimulus until the end of the epoch (596 ms). Second, LRPs were measured with respect to the response, rather than the stimulus. Because all responses were made with the right hand, a negative readiness potential occurring prior to response should be larger over the left than the right hemisphere (i.e. it will be lateralized). This LRP was measured in a difference wave calculated by subtracting activity at ipsilateral (right hemisphere) sites from that of contralateral (left hemisphere) sites. That is, LRP time courses were computed by subtracting channels C3, F3, Fp1, O1, P3 from C4, F4, Fp2, O2, P4, respectively. These data were then analyzed using a cluster-based approach of successive *t*-tests, whereby *t*-tests were performed across all trials and electrodes at each time point, ranging from 500 ms before to 500 ms after the response, for each condition. A criterion of 12 or more consecutive time frames (approximately 24 ms), where $p < 0.05$ was used to assess statistical significance [59].

Results

Behavioral results

When considering changes in PVT performance (Table 1) across the six PVT blocks of trials as a function of sleep condition, a sleep condition (Sleep Extension, Sleep Restriction) × PVT block (blocks 1–6) ANOVA revealed significantly more lapses in the Sleep Restriction vs. Sleep Extension condition ($F_{(1,17)} = 8.89$, $p = 0.008$, $\eta_2 = 0.34$) and an increasing number of lapses over the course of the six blocks of PVT trials ($F_{(5,85)} = 10.05$, $p < 0.001$, $\eta_2 = 0.37$). A similar pattern of results was observed for the slowest responses in the Sleep Restriction vs. Sleep Extension condition ($F_{(1,17)} = 4.78$, $p = 0.043$, $\eta_2 = 0.22$) and over the course of the six blocks of trials ($F_{(5,85)} = 4.49$, $p = 0.001$, $\eta_2 = 0.22$). Overall response speed was marginally significantly faster in the Sleep Extension vs. Sleep Restriction condition ($F_{(1,17)} = 4.33$, $p = 0.053$, $\eta_2 = 0.20$) and became slower across the six blocks ($F_{(5,85)} = 5.75$, $p < 0.001$, $\eta_2 = 0.25$). There was no significant effect of sleep condition for the fastest responses ($F_{(1,17)} = 0.12$, $p = 0.736$, $\eta_2 = 0.01$), but performance did slow over the course of the blocks of trials ($F_{(5,85)} = 2.63$, $p = 0.029$, $\eta_2 = 0.13$).

A similar analysis approach revealed that in terms of subjective sleepiness, the SSS scores (Table 1) were higher in the Sleep Restriction when compared with the Sleep Extension condition

($F_{(1,17)} = 5.01$, $p = 0.039$, $\eta_2 = 0.23$) and increased over the course of the testing session ($F_{(6,102)} = 11.62$, $p < 0.001$, $\eta_2 = 0.41$).

Stimulus-locked ERPs

To examine whether vigilance state affects visually related processing, we compared VEPs for participants who had extended sleep to those who had restricted sleep. We found electrophysiological differences associated with performing the simple, visual PVT task under the different vigilance-related states for a late positivity following stimulus onset (i.e. time-locked to the appearance of the numerical timer; Figure 1, top). This positivity corresponds well to the timing of the P3. Grand-averaged ERPs (the average of all participant's ERPs) are illustrated at the bottom of Figure 1A. Consistent with the usual scalp distribution of the P3, its amplitude was maximum over parietal/occipital cortex (Figure 1, bottom). More specifically, ERPs were sorted for the fastest and slowest RTs. For the fastest RTs, the ERP revealed sleep condition differences for a positivity occurring between 344 and 418 ms (Figure 1A, top). This late positivity was larger for the fastest response times in the Sleep Extension condition compared with fastest response times in the Sleep Restriction condition ($p = 0.039$; mean difference = 0.91; Cohen's $d = 0.98$). Similarly, for the slowest RTs, ERPs also revealed a significantly larger late positivity between 316 and 449 ms (Figure 1B, top) for the Sleep Extension condition in contrast to those with restricted sleep ($p = 0.004$; mean difference = 0.67; Cohen's $d = 0.69$). Conversely, we found no difference in amplitude at any point throughout the epoch between the fastest and slowest 15% within each condition. Therefore, completing the PVT in a state of reduced vigilance induced by restricted sleep results in a reduced positivity corresponding to the P3 compared with a sleep-extended state. The reduction in this late positivity was observed for both the fastest and the slowest RTs. Together, this suggests that regardless of the speed of the response to the visual stimuli (e.g. fastest responses or slowest responses), the P3-like component was reduced in amplitude following mild acute sleep restriction. Moreover, sleep condition differences at earlier time periods, corresponding to the P1 and N1 (shown in Figure 1), were not significant for trials sorted according to the fastest and slowest RTs.

Table 1. Overall PVT performance and SSS scores (mean of blocks of trials) in the sleep extension (Sleep Extension) and sleep restriction (Sleep Restriction) conditions

	Sleep Extension		Sleep Restriction	
	M	*SD*	M	*SD*
SSS	3.60	1.28	4.47	0.97
Number of lapses	34.02	24.48	40.88	23.79
Mean response speed	2.08	0.37	1.98	0.34
Mean fastest	2.61	0.29	2.62	0.28
Mean slowest	1.49	0.47	1.31	0.45

Speed expressed as the reciprocal of reaction time (ms) × 1000.

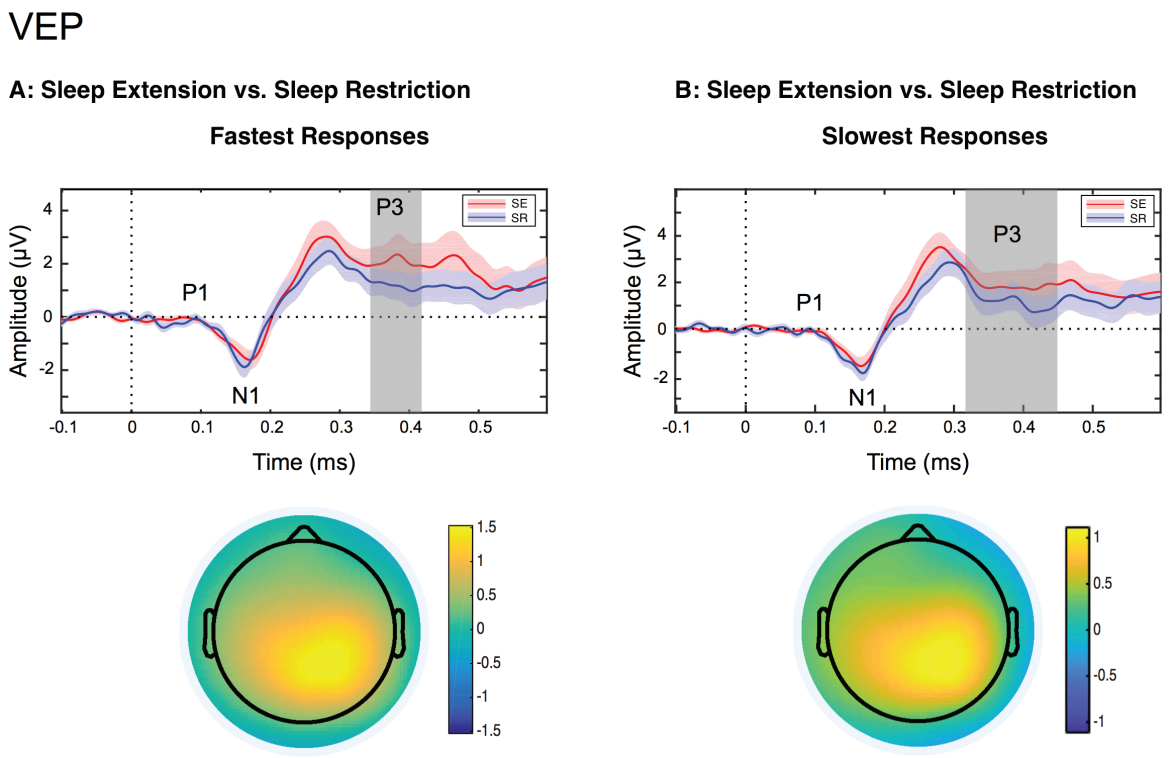

Figure 1. (A) Top panel are grand averaged visual-evoked potentials (N = 18) for the fastest trials in both sleep related conditions; sleep extension condition (Sleep Extension, Fastest Responses; red) and sleep restriction condition (Sleep Restriction, Fastest Responses; blue) for significant electrodes (marked by gray shaded region) using cluster-based permutation statistics. P1, N1, and P3 peaks are indicated. Bottom panel is the scalp topography reflecting mean activity (μV) during the significant time window for the Sleep Extension vs. Sleep Restriction conditions for the Fastest Responses. (B) Top and bottom panels reflect the same information presented in (A), comparing the slowest trials in both conditions (Sleep Extension vs. Sleep Restriction conditions for the Slowest Responses). Significant electrodes included in the average waveform for Figure 1A (top panel) include O1, O2, P3, P4, Pz, C3, C4, Cz, Fz, F4, and Fp2 and the significant electrodes for Figure 1B (top panel) include O1, O2, P3, P4, Pz, C3, C4, Cz, Fz, F3, and Fp1.

Lateralized readiness potential

In addition to visual processing-related ERP differences, we also examined how the two vigilance states affected motor-related processing by computing the brain activity prior to (Figure 2A) and following (Figure 2B) each button press. We found prestimulus LRP condition increases in positvity from –70 to –40 ms for the fastest responses in the Sleep Extension vs. the Sleep Restriction condition. A significant postresponse increase in positivity was also found in the 115–118 ms interval for the Sleep Extension condition compared with the Sleep Restriction condition (Figure 2B). On the other hand, both preresponse and postresponse LRP differences were not significant for the slowest responses (Figure 2C). Additionally, further investigation revealed a significantly larger positivity for the fastest compared with the slowest responses in the 115 to 152 ms postresponse interval (Figure 2D). This was only the case for the Sleep Extension condition, as no differences were apparent at any interval for the fastest vs. slowest responses in the Sleep Restriction condition (Figure 2E). No other comparisons revealed statistically significant results. These results suggest that restricted sleep may affect processes involved with both preparing and executing a motor response, and that very slowest responses executed by those under sleep-extended conditions, and produces a pattern of activity that resembles those with restricted sleep.

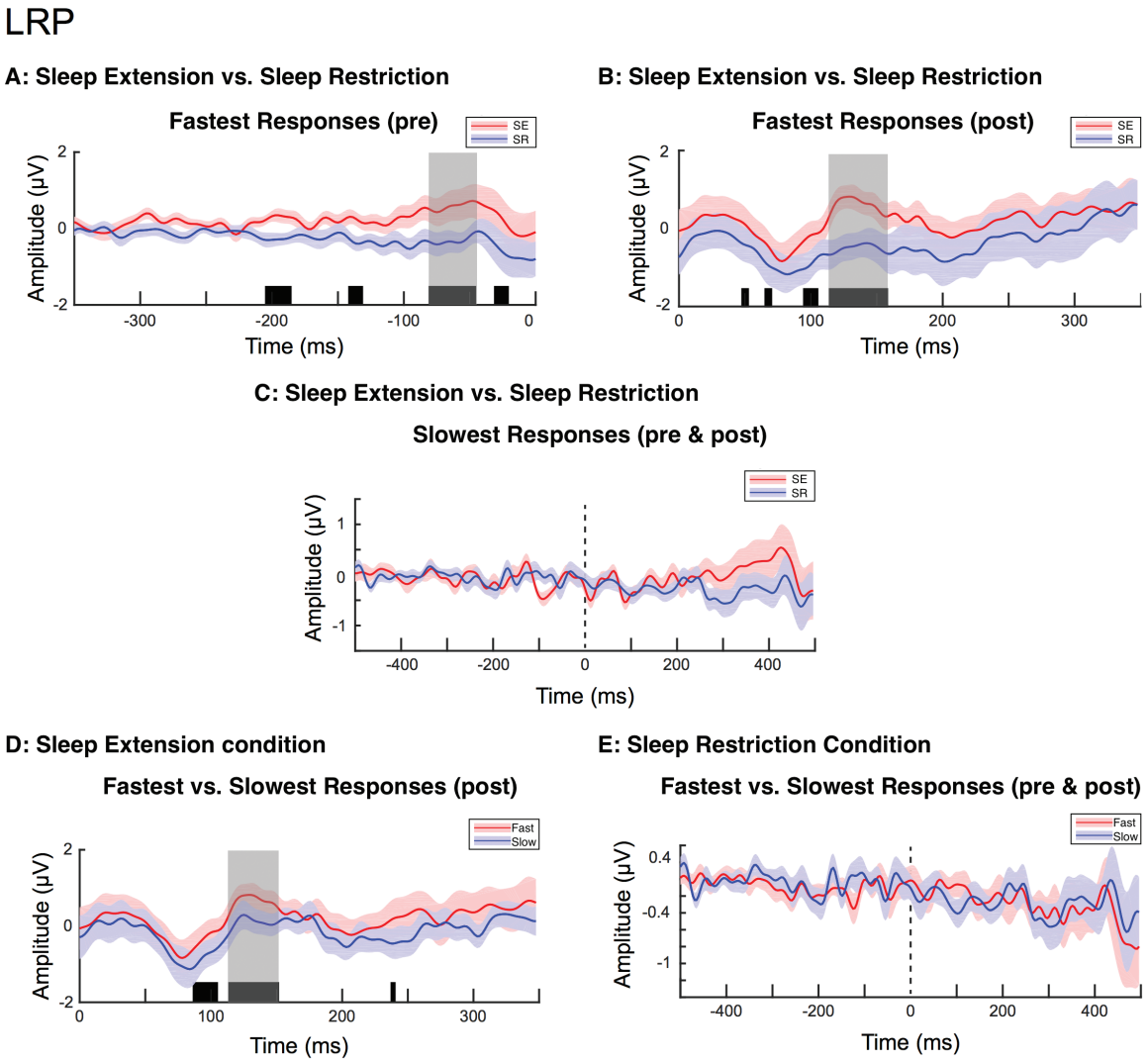

Figure 2. **(A)** Grand averaged LRPs comparing the fastest trials in the Sleep Extension (SE) condition (red) vs. Sleep Restriction (SR) condition (blue) prior to making a response (pre). Statistical significance (shaded gray area) was based on successive *t*-tests, where $p < 0.05$ for at least 12 consecutive time points (approximately 24 ms). (B) Differences in the grand averaged lateralized evoked potentials between the fastest trials in the Sleep Extension (red) vs. Sleep Restriction (blue) in the period after a response was a made (post). (C) Grand averaged lateralized readiness potentials for the slowest responses in the Sleep Extension (red) and Sleep Restriction condition (blue). We found no differences at any point either before or after the response (pre and post). (D) Grand averaged lateralized evoked potentials for the fastest (red) and slowest (blue) trials in Sleep Extension condition in the period after a response was a made (post). The time window when the two conditions differed based on successive *t*-tests are highlighted by the shaded gray region. (E) A comparison of the grand averaged lateralized readiness potentials for the fastest (red) vs. slowest (blue) response in the Sleep Restriction Condition revealed no differences before or after the response was made (pre and post). The LRP was measured in a difference wave calculated by subtracting activity at ipsilateral (e.g. right hemisphere) sites from that of contralateral (left hemisphere) sites by subtracting C3, F3, Fp1, O1, and P3 from C4, F4, Fp2, O2, and P4, respectively.

Discussion

Much is known about the behavioral and cognitive consequences of chronic sleep loss [5, 60–63]. Recent advancements have been made on the impact of severe, acute sleep loss on objective measures of vigilance and subjective measures of sleepiness, and also how this relates to EEG oscillations [42]. However, surprisingly, relatively little is known about the accompanying changes in brain activity associated with changes in vigilance (e.g. PVT performance) from mild (e.g. only a couple of hours) and acute (e.g. only a single night) of sleep loss.

Although the PVT is undoubtedly the most frequently used cognitive task in sleep deprivation studies, very few studies have examined the actual neural correlates of information processing of the stimulus or related to the behavioral response. In one study, Hoedlmoser et al. examined the effect of total sleep deprivation on the PVT administering it at normal bedtime, and every hour thereafter over the course of a normal 8 hr sleep period [42]. They recorded ERPs following presentation of the stimulus. The authors noted that the early P1 amplitude became increasingly attenuated. The P1 is sensitive to manipulations of attention. By contrast, there was no modification of the N1 component. Unfortunately, the authors did not investigate processing related to the P3 component. Their results suggest that attenuation with total sleep deprivation may reflect an inability to sustain attention to the stimulus. By contrast, the present study examined the effects of only 2 hr of sleep restriction—an amount of sleep loss typically regarded as benign. A major aim of this study was to monitor the extent of information processing relative to the visual stimulus (the onset of the numerical timer) used in the PVT. Rather than studying the peaks and valleys elicited by the stimulus relative to baseline, we tested the effects of sleep loss at every point in time. Unlike the results of Hoedlmoser et al., the 2 hr of sleep loss and subsequent testing during the daytime did not significantly affect data points in the ~100 to 200 ms range corresponding to the traditional P1 or N1 components. Given that these studies are otherwise very comparable, taken together, it would appear that this early processing is only affected by sleep loss lasting longer than 2 hr, or when testing occurs during nonoptimal times (e.g. during the normal sleep period).

Two hours of sleep loss did however result in a significant attenuation of a parietal maximum positivity occurring between about 300 and 500 ms (n.b., although not all data points within this time interval were significantly different between conditions). This spatiotemporal pattern corresponds to the much-studied P3. The P3 is classically elicited by the detection of infrequently presented stimuli. The definition of "infrequently" can vary. In most P3 studies, an oddball task is employed in which participants are presented with rapidly occurring "standard" stimuli, and at rare, unpredictable times, a "non-standard" target is presented. Stimuli are usually presented rapidly, e.g. every 1 to 2 s. In the oddball task, the probability of target presentation is thus very low. In this sense, the target occurs infrequently. By contrast, in the PVT, only a single stimulus type is presented. Its probability of occurrence is thus 1.0. It however occurs infrequently in time, and also at an unpredictable interstimulus interval, and thus may share some common information-processing properties as classic P3 paradigms, and may be a sensitive metric of information processing affected by sleep loss. That said, the amplitude of the positivity that was elicited during the PVT task was much smaller than that usually observed in oddball tasks (often over 10 μV). This is probably because the PVT stimulus was presented on every trial, thereby attenuating the magnitude of the response to the presentation of the unpredictable and infrequent stimuli. Nevertheless, on both the fastest and slowest response trials, the amplitude of this "P3-like" positivity was significantly reduced in the sleep restriction condition. Thus, the differences appear to be present regardless of the speed of responding to the stimulus.

Even after only 2 hr of sleep loss, RT was delayed. A possible explanation for the deterioration in performance on the PVT is thus stimulus evaluation processes, normally reflected by the P3 that employ classic oddball paradigms. On the other hand, processes involved in the actual motoric response may also be implicated. In addition to stimulus-related information processing, a slowed RT may also be a result of inadequate motor-related preparation or execution. This was examined here by the LRP. Because the participants responded with the right hand, this preresponse motor readiness potential would be expected to be larger over the left hemisphere. Sleep restriction also resulted in an attenuation of this LRP. However, unlike the VEP differences, this effect was only observed for the fastest responses. Thus, an explanation for a reduction of very fast responses following sleep restriction may be related to an inadequate readiness to respond. Although responding rapidly may require an optimal readiness-to-respond, this may not be necessary for the very slow responses.

Here, our behavioral results suggest that even a small amount of sleep loss on only one night significantly reduced vigilance and increased sleepiness. Mild and acute sleep restriction also led to significant changes in brain activity. Specifically, we found that sleep loss negatively affected processing of visual stimuli requiring sustained vigilance, and also reduced motor-related responses following stimulus presentation, supported by changes in both visually evoked and motor-related electrophysiological brain potentials.

Previous studies have investigated deficits in information processing during acute, but severe sleep deprivation, reflected by ERPs. By contrast, in the current study, we employed acute and mild sleep restriction with subsequent testing during the day. We found that later components from ~300 to 500 ms were reduced in sleep restricted when compared with sleep-extended conditions for both the fastest and slowest response times, but no ERP difference in fastest vs. slowest within each condition, likely reflecting reduced processing capacity for decision making [29]. We have extended these findings by also investigating motor response-locked evoked potentials. These analyses revealed that the amplitude of the LRP was reduced in the sleep-restricted compared with sleep-extended condition, both before (–70 to –40 ms) and after (115 to 158 ms) the motor response, thus suggesting that motor response preparation and execution [30, 64] were likely impaired under conditions where sustained vigilance is required. Interestingly, the P3b component characterized by positivity over posterior electrodes, coinciding with the topography of our VEP late positivity findings, and have recently been suggested to reflect processing at the intersection between perception and decision making [65]. Unlike the P3 which reflects the response to a rare and unpredictable stimulus, in the present study, this reduction of amplitude of late positivity might instead reflect lethargy in making a response at all, to an unpredictable stimulus in the face of monotony and sleep

restriction. Together, these results suggest that our findings of changes in both visually evoked and response-evoked measures of brain activity linked to poorer performance on the PVT may reflect an electrophysiological index of drowsiness.

The behavioral, cognitive, and electrophysiological correlates of mild and acute sleep loss are important to understand, as this type of sleep loss is ubiquitous, and, perhaps even more importantly, is typically regarded to be innocuous. The results of the present study suggest that even a small amount of sleep loss can have deleterious consequences for visual attention and behavioral responding in the face of actively trying to sustain vigilance. Thus, this type of sleep restriction has high ecological validity when compared with more extreme forms of sleep deprivation. This has direct implications for scenarios such as the DST change, long-haul highway driving, academic performance, and in a variety of workplace settings that require sustained vigilance in the face of a monotonous task. Thus, understanding the cognitive processes and neural markers of sleep loss may lead to important advancements in identifying and mitigating lost productivity, and potentially dangerous lapses in vigilance in the workplace, classroom, and when loss of vigilance can be life threatening, e.g. when driving motor vehicle.

Future research combining vigilance testing and electrophysiological recording in more ecologically valid test conditions (e.g. using driving simulators) may help us to uncover how sleep loss can impair performance, and to identify the neural markers of reduced vigilance. In addition, the interaction of sleep pressure and circadian rhythmicity on mild acute sleep loss would be interesting to disentangle, in terms of understanding when vigilance is maximally or minimally affected. Here, we chose the "mid-afternoon dip" for the time of the testing sessions in order to maximize the chance of detecting the effects of sleep loss on related behavior, information processing, and the EEG. Finally, future studies could also employ combined neuroimaging to better understand the functional and neuroanatomical substrates which are affected by sleep loss.

Funding

This research was supported by an Natural Sciences and Engineering Research Council (NSERC) of Canada Engage Grant (EGP 485531-15) and an NSERC Engage Plus grant (EGP2 503228-16) (to S.F. and A.C.S.).

Conflict of interest statement. None declared.

References

1. Tjepkema M. Insomnia. *Heal Reports*. 2005;**17**(1):9–25.
2. National Heart, Lung and BI (NHLBI). *National Sleep Disorders Research Plan*. Bethesda, MD: U.S. Department of Health; 2003.
3. Hossain JL, *et al*. The prevalence, cost implications, and management of sleep disorders: an overview. *Sleep Breath*. 2002;**6**(2):85–102.
4. Roth T. An overview of the report of the national commission on sleep disorders research. *Eur Psychiatry*. 1995;**10**:109s–113s.
5. Hafner M, *et al*. *Why Sleep Matters – the Economic Costs of Insufficient Sleep*. Santa Monica, CA: RAND Corporation; 2016.
6. Foundation NS. *2013 International Bedroom Poll*. Arlington, VA: National sleep foundation; 2013.
7. Swaen GMH, *et al*. Fatigue as a risk factor for being injured in an occupational accident: results from the Maastricht cohort study. *Occup Environ Med*. 2003;**60**(Suppl 1):i88–i92.
8. Léger D, *et al*. Medical and socio-professional impact of insomnia. *Sleep*. 2002;**25**(6):625–629.
9. Akerstedt T, *et al*. A prospective study of fatal occupational accidents – relationship to sleeping difficulties and occupational factors. *J Sleep Res*. 2002;**11**(1):69–71.
10. Barnes CM, *et al*. Changing to daylight saving time cuts into sleep and increases workplace injuries. *J Appl Psychol*. 2009;**94**(5):1305–1317.
11. Smith ME, *et al*. The impact of moderate sleep loss on neurophysiologic signals during working-memory task performance. *Sleep*. 2002;**25**(7):784–794.
12. Barkan R. Using a signal detection safety model to simulate managerial expectations and supervisory feedback. *Organ Behav Hum Decis Process*. 2002;**89**:1005–1031.
13. Barkan, Z. Erev. accidents and decision making under uncertainty: a comparison of four models. *Organ Behav Hum Decis Process*. 1998;**74**(2):118–144.
14. Smith AC. Spring forward at your own risk: daylight saving time and fatal vehicle crashes. *Am Econ J Appl Econ*. 2016;**8**(2):65–91.
15. MacLean AW, *et al*. Psychometric evaluation of the Stanford sleepiness scale. *J Sleep Res*. 1992;**1**(1):35–39.
16. Dinges DF, *et al*. Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. *Behav Res Methods*. 1985;**17**(6):652–655.
17. Jewett ME, *et al*. Dose-response relationship between sleep duration and human psychomotor vigilance and subjective alertness. *Sleep*. 1999;**22**(2):171–179.
18. Basner M, *et al*. Maximizing sensitivity of the psychomotor vigilance test (PVT) to sleep loss. *Sleep*. 2011;**34**(5):581–591.
19. Dustman RE, *et al*. Beta brain waves as an index of alertness. *Science*. 1962;**137**(3529):533–534.
20. Klimesch W. EEG alpha and theta oscillations reflect cognitive and memory performance: a review and analysis. *Brain Res Brain Res Rev*. 1999;**29**(2-3):169–195.
21. Klimesch W, *et al*. Induced alpha band power changes in the human EEG and attention. *Neurosci Lett*. 1998;**244**(2):73–76.
22. Ogilvie RD. The process of falling asleep. *Sleep Med Rev*. 2001;**5**(3):247–270.
23. Blake H, *et al*. Factors influencing brain potentials during sleep. *J Neurophysiol*. 1939;**2**(1):48–60.
24. Aeschbach D, *et al*. Two circadian rhythms in the human electroencephalogram during wakefulness. *Am J Physiol*. 1999;**277**(6):R1771–R1779.
25. Ogilvie RD, *et al*. Behavioral, event-related potential, and EEG/FFT changes at sleep onset. *Psychophysiology*. 1991;**28**(1):54–64.
26. Cajochen C, *et al*. Dose-response relationship for light intensity and ocular and electroencephalographic correlates of human alertness. *Behav Brain Res*. 2000;**115**(1):75–83.
27. Cote KA, *et al*. Changes in the scalp topography of event-related potentials and behavioral responses during the sleep onset period. *Psychophysiology*. 2002;**39**(1):29–37.
28. Campbell K, *et al*. Evoked potential measures of information processing during natural sleep.In: Broughton R, Ogilvie RD, eds. *Sleep Arousal Perform*. Berlin: Birkhäuser - De Gruyter; 1992: 86–116.
29. Kok A. On the utility of P3 amplitude as a measure of processing capacity. *Psychophysiology*. 2001;**38**(3):557–577.
30. Lutzenberger W, *et al*. Asymmetry of brain potentials related to sensorimotor tasks. *Int J Psychophysiol*. 1985;**2**(4):281–291.
31. Houlihan M, *et al*. Extraversion and motor response initiation. *J Individ Differ*. 2011;**32**(2):103–109.

32. Van Dongen HP, *et al*. Caffeine eliminates psychomotor vigilance deficits from sleep inertia. *Sleep*. 2001;**24**(7):813–819.
33. Belenky G, *et al*. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. *J Sleep Res*. 2003;**12**(1):1–12.
34. Lustig KA, *et al*. Sex hormones play a role in vulnerability to sleep loss on emotion processing tasks. *Neurobiol Sleep Circadian Rhythm*. 2018;**5**:94–104.
35. Cote KA, *et al*. CNS arousal and neurobehavioral performance in a short-term sleep restriction paradigm. *J Sleep Res*. 2009;**18**(3):291–303.
36. Corsi-Cabrera M, *et al*. Time course of reaction time and EEG while performing a vigilance task during total sleep deprivation. *Sleep*. 1996;**19**(7):563–569.
37. Caldwell JA, *et al*. Body posture affects electroencephalographic activity and psychomotor vigilance task performance in sleep-deprived subjects. *Clin Neurophysiol*. 2003;**114**(1):23–31.
38. Van Dongen HPA, *et al*. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep*. 2003;**26**(2):117–126.
39. Schulze C, *et al*. Can cognitive deficits facilitate differential diagnosis between at-risk mental state for psychosis and depressive disorders? *Early Interv Psychiatry*. 2013;**7**(4):381–390.
40. Banks S, *et al*. Behavioral and physiological consequences of sleep restriction. *J Clin Sleep Med*. 2007;**3**(5):519–528.
41. Cote KA, *et al*. Physiological arousal and attention during a week of continuous sleep restriction. *Physiol Behav*. 2008;**95**(3):353–364.
42. Hoedlmoser K, *et al*. Event-related activity and phase locking during a psychomotor vigilance task over the course of sleep deprivation. *J Sleep Res*. 2011;**20**(3):377–385.
43. Dinges DF, *et al*. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4-5 hours per night. *Sleep*. 1997;**20**(4):267–277.
44. Mollicone DJ, *et al*. Time of day effects on neurobehavioral performance during chronic sleep restriction. *Aviat Space Environ Med*. 2010;**81**(8):735–744.
45. Russo M, *et al*. Oculomotor impairment during chronic partial sleep deprivation. *Clin Neurophysiol*. 2003;**114**(4):723–736.
46. Vgontzas AN, *et al*. Adverse effects of modest sleep restriction on sleepiness, performance, and inflammatory cytokines. *J Clin Endocrinol Metab*. 2004;**89**(5):2119–2126.
47. Douglass AB, *et al*. The sleep disorders questionnaire. I: creation and multivariate structure of SDQ. *Sleep*. 1994;**17**(2):160–167.
48. Beck AT, *et al*. Psychometric properties of the beck depression inventory: twenty-five years of evaluation. *Clin Psychol Rev*. 1988;**8**(1):77–100.
49. Beck AT, *et al*. An inventory for measuring clinical anxiety: psychometric properties. *J Consult Clin Psychol*. 1988;**56**(6):893–897.
50. Yun CH, *et al*. Daytime sleepiness associated with poor sustained attention in middle and late adulthood. *Sleep Med*. 2015;**16**(1):143–151.
51. Jongen S, *et al*. Sensitivity and validity of psychometric tests for assessing driving impairment: effects of sleep deprivation. *PLoS One*. 2015;**10**(2):e0117045.
52. Lim J, *et al*. Sleep deprivation and vigilant attention. *Ann N Y Acad Sci*. 2008;**1129**:305–322.
53. Basner M, *et al*. Maximizing sensitivity of the psychomotor vigilance test (PVT) to sleep loss. *Sleep*. 2011;**34**(5):581–591.
54. Jones K, *et al*. Frontal lobe function, sleep loss and fragmented sleep. *Sleep Med Rev*. 2001;**5**(6):463–475.
55. Iber C, *et al*. *The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specifications*. 1st ed. Westchester, IL: American Academy of Sleep Medicine; 2007.
56. Delorme A, *et al*. EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics including independent component analysis. *J Neurosci Methods*. 2004;**134**(1):9–21.
57. Maris E, *et al*. Nonparametric statistical testing of EEG- and MEG-data. *J Neurosci Methods*. 2007;**164**(1):177–190.
58. Oostenveld R, *et al*. FieldTrip: open source software for advanced analysis of MEG, EEG, and invasive electrophysiological data. *Comput Intell Neurosci*. 2011;**2011**:156869.
59. Guthrie D, *et al*. Significance testing of difference potentials. *Psychophysiology*. 1991;**28**(2):240–244.
60. Krause AJ, *et al*. The sleep-deprived human brain. *Nat Rev Neurosci*. 2017;**18**(7):404–418.
61. Killgore WDS. Effects of sleep deprivation on cognition. In: Kerkhof GA, van Dongen HPA, eds. *Progress in Brain Research*. Vol. **185**. Elsevier; 2010: 105–129.
62. Alhola P, *et al*. Sleep deprivation: impact on cognitive performance. *Neuropsychiatr Dis Treat*. 2007;**3**(5):553–567.
63. Goel N, *et al*. Neurocognitive consequences of sleep deprivation. *Semin Neurol*. 2009;**29**(4):320–339.
64. Houlihan M, *et al*. Reaction time and movement time as measures of stimulus evaluation and response processes. *Intelligence*. 1994;**18**(3):289–307.
65. Verleger R, *et al*. Evidence for an integrative role of P3b in linking reaction to perception. *J Psychophysiol*. 2005;**19**(3):165–181.