Physiological arousal and attention during a week of continuous sleep restriction

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Waking brain physiology underlying deficits from continuous sleep restriction (CSR) is not well understood. Fourteen good sleepers participated in a 21-day protocol where they slept their usual amount in a baseline week, had their time in bed restricted by 33% in a CSR week, and slept the desired amount in a recovery week. Participants slept at home, completing diaries and wearing activity monitors to verify compliance. Each day participants completed an RT task and mood and sleepiness ratings every 3 h. Laboratory assessment of electrophysiology and performance took place at the end of baseline, three times throughout the CSR week, and at the beginning of recovery. Participants reported less sleep during CSR which was confirmed by activity monitors. Correspondingly, well-being and neurobehavioural performance was impaired. Quantitative EEG analysis revealed significantly reduced arousal between the 1st and 7th days of restriction and linear effects at anterior sites (Fp2, Fz, F8, T8). At posterior sites (P4, P8), reductions occurred only later in the week between the 4th and 7th nights of restriction. Both the immediate linear decline in arousal and precipitous drop later in the week were apparent at central sites (C4, Cz). Thus, frontal regions were affected immediately, while parietal regions showed maintenance of function until restriction was more severe. The P300 ERP component showed evidence of reduced attention by the 7th day of restriction (at Pz, P4). EEG and ERPs deficits were more robust in the right-hemisphere, which may reflect greater vulnerability to sleep loss in the non-dominant hemisphere.

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Total sleep deprivation in humans (greater than 24 h of wakefulness) has been widely studied for more than a century [1]. The effects on mood and performance, as well as characteristics known to mask deficits, have been well described [2–6]; however, the nature of brain functioning underlying these behavioural changes remains poorly understood. It has been proposed that the deficits observed during sleep deprivation may involve frontal lobe impairment in particular [7]. Consistent with this notion, many brain imaging studies have shown reduced activity in prefrontal cortex following total sleep deprivation [8–12]. However, a number of imaging studies have also shown increased activity in the frontal regions [13,14]. Drummond and Brown suggested that these regional patterns of activation and deactivation may be dependent on the nature of the specific task performed, and that the brain may be capable of compensation during sleep loss [15]. EEG and event-related potentials (ERPs) are neurophysiological measures of arousal and information processing that may also be used to investigate the CNS nature of performance during sleepiness. They have greater time resolution than fMRI imaging techniques. Specifically, the timing of cognitive processes can be determined in the order of milliseconds using ERPs, while EEG provides a continuous measure of arousal over time. Much of the prior research using imaging, EEG, or ERP techniques to examine the neural basis of performance deficits during sleep loss has been done under extreme conditions of total sleep deprivation. The aim of the present study was to investigate waking brain function during continuous sleep restriction.

There are a number of reports on waking EEG characteristics following total sleep deprivation [16–24]. There are some inconsistencies in outcome, likely due to differences in methods (e.g., timing of recording, length of sleep deprivation, recording during eyes opened or closed) and analysis techniques (e.g., amount of EEG processed, reporting relative or absolute power, definition of frequency bands). For instance, some have reported that total sleep deprivation leads to increased power in lower frequencies (e.g., alpha and theta), when recorded at rest (eyes opened) or during task performance [16,19,20]. However, alpha is typically lower when sleepy if recorded with eyes closed [22], similar to what is seen during sleep onset. Given that alpha is normally blocked when the eyes are opened, recording it while eyes are closed is likely a more sensitive measure of changes in alpha and thus the best metric of sleepiness. The increases in theta have been shown to be correlated with reaction time (RT) slowing [22] and subjective sleepiness reports [16,21,24]. Alpha and theta EEG in wakefulness are thus markers of CNS arousal which are thought to reflect a homeostatic process similar to the rebound of slow wave sleep following sleep deprivation [17,25]. There have also been a number of reports of increased high frequency EEG in the beta range (e.g., typically 16–40 Hz) following total sleep deprivation which has...
been interpreted as increased effort [18–20,22]. A few studies have attempted to localize the scalp topography of these effects. Cajochen et al. reported that the increased power in theta band was predominant at frontalis sites [17]. Similarly, Forest and Godbout observed the increased theta at frontalis and temporal sites, as well as increased beta at frontalis sites, following one night of total sleep deprivation [26]. Ferreira et al. recently reported decreases in absolute power of alpha apparent in frontalis, temporal and occipital regions, decreases in absolute power of beta in the temporal region, and increases in relative power of theta and delta in the occipital and temporal areas following one night of sleep deprivation [27]. The authors proposed that the site-specific effects might reflect regional areas of brain adaptation.

ERPs have been investigated following total sleep deprivation using oddball paradigms to elicit long-latency ERPs such as N1-P2-P300 which reflect various stages of information processing. An early negative wave called N1 is considered mesogenous; that is, it is exogenous in nature because it is sensitive to changes in physical parameters of the stimulus (and thus reflects early sensory processing), yet it is endogenous in nature because it changes with manipulations to attention (i.e., N1 is reduced when attention is reduced due to the removal of a long-lasting negative wave, called Processing Negativity (PN), which overlaps in time and sums with N1) [28]. Delays in N1 latency have been reported following total sleep deprivation [29,30]; these changes may reflect slowed sensory encoding processes or delays in attentional processes due to PN. P300 is an endogenous ERP component that is altered by changes in attention [28]. Both delayed latency and reduced amplitude of the P300 component have been reported using auditory oddball paradigms [31–34]. These data indicate that total sleep deprivation also affects later, more complex, stages of information processing. Specifically, these alterations to P300 reflect delays in the time taken to discriminate target from non-target stimuli, and impairment in the degree of attention that is allocated to the task [35]. Corsi-Cabrera et al. reported reduced amplitudes in late components (ranging from 180 ms to 718 ms) following 40 h of total sleep deprivation using a visual ERP paradigm [36]. The reductions in amplitude were correlated with RT slowing and increased alpha/theta/beta EEG power in the pre-stimulus interval, suggesting a role for attentional mechanisms in the oft-reported deficits in performance and arousal. The aforementioned studies applied either a single electrode or three midline sites for analysis of ERPs. Based on a recording from five scalp sites, Kaneda et al. reported that P300 latency was delayed at fronto-central sites while amplitude was reduced centrally [37]. Recently, Gosselin et al. showed that 36 h of wakefulness affected the amplitude of a frontally driven ‘novel P3’ component [38]. These data show that a specific type of information processing which is localized to frontal brain regions, namely novelty processing, is compromised by sleep loss.

Waking EEG, ERP, and imaging data [7,15] reveal that there are changes in CNS function that underlie deficits in performance following total sleep deprivation. It is also important to investigate the impact of a degree of sleep loss that is more commonly experienced in the real world. Partially restricting sleep time (e.g., to 4 or 6 h per night) has been shown to affect the EEG characteristics of sleep [39,40]. Specifically, Brunner et al. and Belenky et al. reported decreased amplitudes of Stages I, 2 and REM sleep throughout the course of sleep restriction. Brunner et al. further showed that the pattern of delta power (.75–4.5 Hz) during non-REM sleep increased over four consecutive nights of restriction, demonstrating that sleep pressure increases across continuous nights of sleep restriction. The behavioural effects of sleep restriction on waking function have been well studied in recent years. Dinges et al. demonstrated cumulative deficits on a range of behavioral tasks during continuous sleep restriction [41]. They reported that sleep restricted by 33% below habitual amounts over a one-week period led to a linear increase in sleepiness and fatigue, and deficits in mood and performance “lapses”.

More recently, two separate groups published the results of dose–response investigations of cumulative sleep restriction. Van Dongen et al. manipulated sleep time by reducing time in bed for three separate groups to 4, 6, and 8 h over 14 days [42], while Belenky et al. did so by reducing time in bed for four groups to 3, 5, 7, and 9 h over 7 days [39]. In both studies, sleep restriction was compared to baseline and recovery conditions within subjects. Van Dongen et al. reported cumulative deficits on behavioural tasks across the 14 days in the 4- and 6-hour sleep groups. Similarly, Belenky et al. reported continuous deficits in the most severely restricted group (3-hr sleepers). In the 5- and 7-hour groups, however, they reported an initial drop in performance followed by stable but reduced performance for 7 days of restriction. Belenky et al. therefore concluded that the brain may adapt to this level of sleep restriction over time. Although these studies are inconsistent with respect to the cumulative nature of impairment from continuous sleep restriction, they do provide support for dose-dependent effects of sleep restriction on performance of behavioural tasks. Neither study provided data on the associated waking brain physiology. Examining EEG/ERPs in wakefulness coincident with behavioural performance will lead to a better understanding of the nature of brain functioning during sleep loss. These techniques may also be used to determine if the brain is capable of adaptive change or compensation in order to maintain performance during sleep loss.

The study by Brunner and colleagues is unique in reporting waking EEG changes following continuous sleep restriction paradigm [40]. The authors reported decreased alpha (9.25–10 Hz) following the 2nd night of restriction and increased delta (3.75–4.5 Hz) after the 4th night of restriction in the eyes opened condition. In the eyes closed condition, alpha decreased and delta increased after the 3rd and 4th nights of restriction. These waking EEG data showed a pattern of brainwave synchronization similar to what was observed in the nocturnal sleep episode. The interpretation of the waking EEG data in this study is limited by the amount of data recorded (e.g., a single recording session at 07:15, for 3 min, from either the C3 or C4 site) and the high degree of inter-subject variability that resulted. In the dose–response study of sleep restriction by Van Dongen et al. [42], they found no progressive changes in alpha or theta EEG over the 2 week period of sleep restriction [43]. Again, the small amount of data collected from a single electrode during a 5-minute recording session in a subset of only 6 participants per group may explain the lack of sensitivity in the EEG measure.

Although there are no reports of ERPs following sleep restriction, two studies have examined information processing following experimentally induced sleep fragmentation [44,45]. Cote et al. reported that two nights of fragmentation led to impairment in arousal and early encoding processes reflected in the N1 component of the auditory ERP [44]. No changes were observed for the later P300 waveform. Using a visual ERP paradigm, however, Kingshott et al. found reductions in amplitude of the later P300 component that were apparent at widespread locations across the scalp [45]. Together, these studies show deficits in information processing following a subtle level of sleep disruption which were apparent in electrophysiological measures when no behavioural performance deficit could be observed.

In the present study, physiological arousal and attention were investigated using quantitative electrophysiological methods, EEG and ERPs, during a week of continuous sleep restriction. In a 21-day protocol (one week of baseline, one week of sleep restriction, one week of recovery), participants completed sleep diaries, wore activity monitors and performed regularly scheduled RT tasks while at home. Visits to the laboratory for assessment of electrophysiology and performance took place on the morning of Day7 (following 7th night of baseline), Day8 (following 1st night of restricted sleep), Day11 (following 4th night of restricted sleep), Day14 (following 7th night of restricted sleep), and Day15 (following 1st night of recovery). Sleep
was restricted at home in order to study individuals over a 3-week period under conditions more ecologically valid than the laboratory setting where long-term study is problematic. Given that the behavioural profile of sleep restriction has been well established in systematic laboratory studies, it is timely that researchers begin to examine the effects of sleep loss outside the laboratory. There is a paucity of studies that systematically compare home-based and laboratory studies of sleep restriction. In order to ensure the reliability of home data, careful instructions were given and compliance was verified. Based on previous behavioural research, it was hypothesized that this degree of sleep restriction would lead to deficits in mood and performance (e.g., on RT). In addition, it was hypothesized that systematic changes in physiological arousal and attention would be associated with sleep restriction. A multiple-channel EEG recording approach was taken in order to investigate how regional brain activity changed over the course of sleep restriction.

1. Methods

1.1. Participants

Participants were recruited via advertisements placed around the local university and in the community. Participants meeting the following criteria were invited to contact the Sleep Research Laboratory: 18 to 35 years old, healthy, good sleeper, right-handed, non-smoker, and free from medications. In a telephone interview, participants were further screened to ensure that they had a regular sleep/wake schedule, typically consumed less than 1–2 caffeinated beverages per day, were fluent in English, and had no history of depression, heart disease, neurological disease, or chronic pain. Appropriate candidates were invited to an orientation session and tour of the laboratory. After signing consent, they were given a hearing test to ensure that their hearing was within an acceptable level for auditory ERP tasks (i.e., below 15 dB at 500, 1000, 1500, and 2000 Hz in both ears), and completed questionnaires on sleep history and habits, morningness–eveningness chronotype, personality, mood, and health. Candidates then underwent a single night of standard polysomnographic assessment in the Sleep Research Laboratory (e.g., signals recorded from C3/C4 EEG sites, a left and right EOG, and a submental EMG to identify sleep stages). Measures also included abdominal and thoracic respiration with effort bands and leg EMG with electrodes placed over the anterior tibialis of each leg to screen for sleep-related respiratory and movement disorders. No participants were removed during the sleep restriction week; one withdrew because the protocol was “too difficult” and the other was removed by experimenters due to non-compliance for completing tasks and for failure to arrive to the laboratory on time.

The remaining 14 participants completed the 3-week protocol (2 males; mean age=20.86, SD=2.57). Participants were paid $150 for completion of the protocol. Participants who withdrew from the study or who were removed by the researchers due to non-compliance were paid a prorated amount. The Research Ethics Board of the local University approved all study procedures.

1.2. Procedure

The protocol lasted 21 consecutive days and nights (refer to Fig. 1 for an illustration of the protocol). Participants were instructed to maintain a regular sleep–wake schedule for the first week, were sleep restricted during the second week, and were allowed to return to their regular sleeping schedule for the third week. For the duration of the study, participants were instructed to refrain from napping, to minimize alcohol consumption, and to keep caffeine consumption constant at pre-study levels (note: participants were recruited to be minimal caffeine users). For example, participants were allowed to consume their usual morning coffee. The purpose of allowing minimal, but usual amounts of caffeine was to collect data from a more naturalistic sample, avoid caffeine withdrawal, and ensure that participation was not too onerous in the lengthy protocol. On days in which participants were scheduled to have in-laboratory testing of EEG/ERPs, they were instructed to refrain from drinking caffeine prior to the 9 am testing session. This ensured that electrophysiological measures would not be confounded by individual differences in caffeine consumption and sensitivity, or acute enhancement of arousal due to morning caffeine intake. Daily diaries of caffeine consumption showed no statistical difference between the 3-week study conditions, and verified the restriction of caffeine prior to in-laboratory testing.

1.2.1. Pre-study training

Participants came to the laboratory on the day preceding their first day in the 21-day study. They were given all equipment needed for the at-home procedures, including an actigraph (JM Systems Inc), reaction time device and charger, sleep-activity diary, and a study instruction log. Reaction time was collected using a hand-held electronic device.
that was custom built in the laboratory. Data was displayed to the participant on a screen, and responses were made using buttons with the right or left hand. A computer was used to pre-program the parameters of the RT task (e.g., task length, ISI, etc), response conditions, and visual analogue scales. Data was downloaded each week in the laboratory. Participants were trained on how to use the reaction time device, and then practiced each task in the performance assessment battery (PAB) that would be administered in laboratory sessions with the 2-back memory test being completed twice. Practice data was checked for accuracy in order to ensure participants understood task instructions. The importance of strict compliance with instructions and the timing of all aspects of the protocol were imparted to participants at this time.

1.2.2. Home assessment

Participants were instructed to wear an activity monitor on the wrist, which resembled a watch, for each of the 21 days and nights. They were told only to remove the monitor so that it did not get wet (e.g., when showering) or damaged (e.g., when playing contact sports). The activity monitors were preset to begin recording at 18:00 on the day of training. Participants were also asked to keep a sleep-activity diary, indicating their time in bed and sleep time, as well as any meals, snacks, caffeine intake, alcohol consumption, and exercise. Participants were instructed to begin this diary at 18:00 on the day of their training. They were also given a hand-held RT monitor; they were instructed to self-administer a 10-minute RT task, 5 times each of the 21 days (i.e., 09:00, 12:00, 15:00, 18:00, 21:00). They were to begin this task at 09:00 the following morning (i.e., Day 1 of the protocol). The hand-held RT monitor also delivered visual analogue scales (VAS) of sleepiness and mood prior to the RT task, where higher values on a 16-point scale reflected more alertness and happiness respectively. On their study instruction log, participants were required to sign their initials and log the time that each RT task was completed. The RT monitor also logged the exact time the task was completed so that the accuracy of participants’ logs could be verified.

For the second week, each participant’s sleep was restricted by 33% of their baseline. In order to calculate this sleep restriction, an average time in bed (less major wake time) was calculated as an estimate of habitual sleep time. Sleep was restricted both by delaying bedtime and by advancing rise time (i.e., 16.5% of total time in bed was truncated from both the beginning and end of the night). Specific lights-out and lights-on times were communicated to the participant. Actigraphy and diary data confirmed that participants complied with these instructions.

1.2.3. Laboratory assessment

Participants visited the laboratory from 09:00 until 11:00 a.m. according to the following schedule: on the morning of Day 7 (following 7th night of baseline), Day 8 (following 1st night of restricted sleep), Day 11 (following 4th night of restricted sleep), Day 14 (following 7th night of restricted sleep), and Day15 (following 1st night of recovery). On each occasion, the participants’ actigraph record and sleep-activity diary were examined to ensure that they had complied with instructions related to sleep/wake times, napping, and caffeine consumption. The data from the participants’ reaction time device were also examined to ensure that all tests had been completed at the appropriate times.

Electrophysiological signals were recorded at 200 Hz with Lamont digital amplifiers (analog filters preset at .05–70 Hz), and displayed and analyzed using Harmonie software (Stellate, Inc). Using an electrode cap, a 19-channel montage was recorded from frontal-parietal (FP1, FP2), frontal (F3, Fz, F4, F7, F8), central (C3, Cz, C4), temporal (T7, T8), parietal (P3, Pz, P4, P7, P8), and occipital (O1, O2) scalp sites. References were placed at A1 and A2 and ground at AFz. Vertical and horizontal EOG channels were recorded, as well as EMG, to allow for offline artifact rejection. Waking EEG and ERPs were recorded during a 45 min PAB that was administered on a computer located in the bedroom using E-Prime software (Psychology Software Tools Inc). Participants began the PAB at 10:00 and completed the tasks in a fixed order as follows:

Profile of Mood States (POMS): Participants were asked to rate their mood on a 5-point scale for 30 adjectives [46]. Analysis was done on a total mood score as well as six identifiable subscales including: Vigor–Activity (V), Fatigue–Inertia (F), Tension–Anxiety (T), Confusion–Bewildenment (C), Depression–Dejection (D), Anger–Hostility (A), and. The POMS was administered on paper.

Pitch ERP oddball task: Participants were asked to discriminate between two tones differing in pitch and respond to the higher-pitched ‘target’ tone (ISI=1–2 s at random; task duration=6 min). The standard tone (1000 Hz, 100 ms, 70 dB) was presented on 80% of trials and the target tone (1500 Hz, 100 ms, 70 dB) was presented on the remaining 20% of trials.

Visual RT task: A warning tone preceded the presentation of a circle on the screen. The participant was asked to respond as quickly as possible to the visual stimulus. While initially designed to pilot test a CNV task, it was used only as a filler task between auditory oddball tasks and was not analyzed further.

Duration ERP oddball task: Participants were asked to discriminate between tones of two different durations, and respond to the shorter duration ‘target’ tone (ISI=1–2 s at random; task duration=6 min). The standard tone (250 Hz, 200 ms, 70 dB) was presented on 80% of trials and the target tone (250 Hz, 100 ms, 70 dB) was presented on the remaining 20% of trials.

Break: 10-min.

Reaction time task: Participants were asked to respond to a 70 dB tone as quickly as possible using the “zero” key on the keypad for 10 min. The inter-stimulus interval varied from 2–10 s.

2-back memory task: Participants identified cases in which the letter that appeared on the computer screen matched the letter that had appeared two before it, and responded to the second letter of the matching pair (Stimulus duration=250 ms, ISI=1750 ms). Three consecutive blocks were administered with a 30 s break in between blocks. Each block contained 18 targets and 42 standard trials presented in a pseudo-random fashion.

Alpha attenuation task: During this 3-min task, participants sat still with their eyes alternately opened or closed, for 30 s at a time. EEG was recorded from 19-sites during the task. Participants were instructed to remain still with eyes fixated on an ‘X’ appearing on the computer screen. This task provided a quantitative measure of physiological sleepiness and arousal [47].

Stanford Sleepiness Scale: Participants used a 7-point scale to rate their level of alertness or sleepiness, where ‘7’ indicated the greatest amount of sleepiness [48].

Subjective Performance Scale: Participants rated their subjective performance on a 5-point scale (1=very poor, 5=very good).

1.3. Data analysis

The screening night was sleep scored according to standard criteria [49] and checked for the presence of sleep-related breathing and movement disorders. Actigraphy, RT and diary data were checked for compliance and downloaded to computer upon each visit to the laboratory. A number of composite variables were calculated from the raw RT data collected at home and in-laboratory sessions. These variables included mean RT, 10% fastest RTs, 10% slowest RTs, and the number of lapses (RTs >500 ms). A lapse is a missed or slow response that is due to a “lapse” in attention.
Waking EEG data from the Alpha Attenuation Task was quantified using Fast Fourier Transform (FFT) analysis techniques. Artifact-free epochs were submitted to power spectral analysis (PSA) using a hanning window method (75% overlap; spectral record length=5.12 s; FFT length =2.56 s). Data were re-referenced offline to an average of A1 and A2 prior to spectral analysis. EEG data were quantified as the power (µV^2/Hz) of each of the following pre-defined frequency bands: theta, 4–8 Hz; low frequency alpha, 8–10 Hz; high frequency alpha, 10–12 Hz. Absolute power in each band was examined to characterize CNS arousal throughout sleep restriction. In addition, the ratio of theta to alpha power (lower values represent reduced arousal) and the Alpha Attenuation Coefficient (ratio of eyes closed to eyes open alpha; lower values reflect reduced arousal) were calculated. EEG data were log transformed prior to statistical analysis due to the skewed nature of the data.

ERPs were obtained from electrophysiological data recorded during both the pitch and duration auditory oddball tasks, and analyzed using ERPScore [50]. Target and non-target trials were averaged separately. A software-driven automatic artifact rejection routine was used to identify and remove any trials on which the EEG or EOG exceeded +/-100 µV in order to remove artifact from eye movements. A grand average ERP was determined for each study condition. After artifact rejection, the average number of trials in grand averages for each PAB session was 194 targets and 807 standards for the pitch oddball and 195 targets and 733 standards for the duration oddball task. Based on the peaks apparent in the grand averages, N1 and P2 amplitude and latency were then measured following standard stimuli in the within-subject averages. The amplitude and latency of N1 was measured as the most negative peak between 80 and 150 ms and P2 was the most positive peak between 150 and 250 ms. To measure the long-latency P300, the average amplitude value was measured in the within-subject averages from the latency range where the peak was apparent in the grand average for the pitch (300–450 ms) and the duration (400–650 ms) target stimuli separately. N1 and P2 appear as sharp peaks which are easily identifiable in the individual averages and thus precise measurement of the peak amplitude is possible; however, the P300 peak is a relatively slow wave lasting about 200 ms, so its amplitude is more accurately measured by taking the average amplitude values across a wider range.

Validation data (sleep time and activity level) and behavioural data from home (RT, mood, sleepiness) were analyzed using one-way repeated measures ANOVAs (baseline, restriction, recovery weeks). Significant F-tests were followed up with planned Least Significant Differences (LSD) comparisons. All behavioural and physiological data collected in the laboratory were analyzed using one-way repeated measures ANOVAs (baseline, 1st day of restriction, 4th day of restriction, 7th day of restriction, recovery), and followed up with LSD planned comparisons. Given the design of the study, significant quadratic polynomials were also predicted to show decreases in performance from baseline to restriction, followed by a return to baseline levels during recovery. In addition, linear changes across the sleep restriction week were expected. Boxplots were used to identify outliers on all variables; outliers more than 3 standard deviations beyond the mean at baseline were removed from analysis. Violations to assumptions of sphericity were corrected using Greenhouse–Geisser corrections as noted in the adjusted degrees of freedom for each analysis if appropriate. Missing data are indicated in the results section for each variable.

2. Results

Examination of polysomnographic data on the screening night confirmed that all participants were good sleepers. Specifically, participants slept an average of 6.8 h (M=408.07 min; SD=46.02), fell asleep in an average of 9.39 min (SD=8.03), and had an overall sleep efficiency of 91 percent (SD=6.53).

2.1. Actigraphy and diaries

To validate compliance with instructions to curtail sleep, diary estimates of total sleep time and the level of inactivity from activity monitors were compared between baseline, restriction, and recovery weeks using one-way ANOVAs. Participants reported sleeping significantly less during the CSR week compared to baseline (p<.001) and recovery weeks (p<.001), F(2,22)=79.79, p<.001, eta^2=.88. Data were missing from two participants due to ambiguous diary entries at some point during the 21-day protocol. Activity monitors showed that there was significantly less “inactivity” (i.e., sleep) during the CSR week compared to the recovery week (p<.001), and a trend for less activity during the CSR week compared to baseline (p=.061), F(2,22)=6.63, p<.01, eta^2=.38. Data were missing from two participants because of partial missing data from the activity monitor due to technical problems. See Table 1 for means and standard deviations of the home data.

2.2. Home data

Data collected every 3-h while at home (i.e., RT, mood, sleepiness) was collapsed across time-of-day for a daily average value. The daily average accurately reflected data from each time-of-day, except for mood and sleepiness variables for which the morning data (at 09:00) were relatively lower (presumably due to sleep inertia). Therefore, ANOVAs were run for sleepiness and mood with 09:00 data excluded from the daily average. Subjective sleepiness differed significantly across study condition, F(1.36, 17.71)=8.10, p<.01, eta^2=.38. Specifically, sleepiness was lower on baseline compared to both the CSR (p<.01) and recovery weeks (p<.05). Although sleepiness did not return to baseline levels, significant quadratic polynomials provided evidence of some improvement during recovery, F(1,13)=7.88, p<.05, eta^2=.38. Subjective mood reports did not differ significantly by condition, however, there was a linear decline in mood, F(1,13)=5.10, p<.05, eta^2=.28, which showed that mood deteriorated from the baseline to the CSR week, and remained low during recovery.

One-way ANOVAs were run to compare RT across the three study weeks. There was a significant condition effect for mean RT, F(1,27, 16,51)=5.06, p<.05, eta^2=.28. Mean RT during baseline was faster compared to the recovery week (p<.05), with a trend for baseline also being faster than the CSR week (p=.054). The fastest 10% RTs did not reach significance for the main effect, however, there was a trend for a quadratic effect, F(1,13)=3.65, p=.078, illustrating the predicted direction of change in performance from baseline to sleep restriction and to recovery. The slowest 10% RTs significantly differed across condition, F(2,26)=21.56, p<.001, eta^2=.62. RT was slower during CSR (p<.001) and recovery (p<.001) compared to baseline. A significant quadratic effect indicated there was a slight return to baseline in recovery, F(1,13)=6.48, p<.05, eta^2=.68. There was also a significant condition effect for the number of RT lapses, F(1,19, 15.40)=12.02,
2.3. Laboratory data

2.3.1. Behavioural data

See Table 2 for means and standard deviations. Subjective sleepiness, perception of performance, mood, and RT changed as predicted during sleep restriction relative to baseline and recovery conditions. Significant condition effects and quadratic polynomials depicted the following changes during CSR:

Subjective sleepiness. One participant was excluded from this analysis due to missing data. There was a significant main effect, $F(4,48)=5.84, p<.001$, eta$^2=.33$, and quadratic polynomial, $F(1,12)=23.27, p<.001$, eta$^2=.66$. Participants were sleepier on the 4th (p<.001) and 7th days of restriction (p<.01) compared to baseline, sleepier on the 4th compared to the 1st day of restriction (p<.05), and sleepier on the 4th (p<.005) and 7th days of restriction (p<.05) compared to recovery.

Perception of performance. One participant had missing data. There was a significant main effect, $F(4,48)=4.46, p<.005$, eta$^2=.27$, and quadratic polynomial $F(1,12)=18.45, p<.001$, eta$^2=.61$. Participants reported that they were performing better on baseline compared to the 4th day of sleep restriction (p<.01), and better on recovery compared to the 4th (p<.005) and 7th days (p<.005) of sleep restriction. There were also trends for the perception of better performance on baseline compared to the 7th day of restriction (p<.006), and for better performance on recovery compared to the 1st day of sleep restriction (p<.053).

POMS mood. The total mood score, and the Vigor–Activity (V), Fatigue–Inertia (F), and Confusion–Bewilderment (C) subscales of the POMS showed the expected effects of sleep restriction. There was one missing participant from this analysis due to incomplete data entry on the paper form. For total mood, there was a main effect, $F(4,48)=6.60, p<.001$, eta$^2=.36$. Mood was significantly better on baseline than on the 4th (p<.001) and 7th (p<.05) days of sleep restriction. Mood on the 1st day of sleep restriction was also significantly better than the 4th (p<.001) and 7th (p<.05) days of sleep restriction, as well as recovery (p<.05). A significant quadratic effect, $F(1,12)=11.83, p<.005$, eta$^2=.50$, showed that mood was returning toward baseline during recovery. For the V-subscale, there was a main effect for condition, $F(2.17, 26.01)=9.59, p<.001$, eta$^2=.44$; Vigor was higher on baseline compared to the 4th (p<.001) and 7th (p<.005) days of sleep restriction and recovery (p<.01). Similarly, Vigor was higher on the 1st day of sleep restriction compared to the 4th (p<.005) and 7th (p<.01) days of sleep restriction and recovery (p<.05).

RT. Reaction time. Mean RT, median RT, standard deviation of RTs, slowest 10% RTs, fastest 10% RTs, and RT lapses were all calculated from the RT task administered in the laboratory. Three participants had missing data from at least one of the study conditions. There was a significant main effect, $F(1.86,18.64)=3.86, p<.05$, eta$^2=.28$, and quadratic polynomial, $F(1,10)=11.21, p<.01$, eta$^2=.53$, for mean RT. Participants were faster at baseline compared to the 4th (p<.01) day of sleep restriction [including a trend to also be faster at baseline than the 7th day of restriction, p=.051], and faster following recovery compared to the 4th (p<.001) and 7th (p<.05) days of sleep restriction. There was also a trend for slowed RT performance from the 1st to the 4th days of sleep restriction (p=.089). The results for median RT were similar, $F(1.58, 15.85)=3.99, p<.05$, eta$^2=.29$: participants were significantly faster at baseline compared to the 4th (p<.05) and 7th (p<.05) days of sleep restriction, and faster at recovery compared to the 4th (p<.005) and 7th (p<.05) days of sleep restriction. There were trends indicating that following the 1st day of sleep restriction, RT was faster still compared to the 4th (p=.056) and 7th (p=.070) days of sleep restriction. The fastest and slowest 10% RTs, as well as the number of RT lapses showed trend level main effects but significant quadratic effects as predicted. Specifically, the fastest 10% RTs showed a trend for the main effect, $F(4,40)=2.40, p=.066$, but a significant quadratic effect, $F(1,10)=5.93, p<.05$, eta$^2=.37$; RT was faster at baseline (p<.05) and recovery (p<.01) compared to the 4th day of restriction. The slowest 10% RTs showed a trend for the main effect, $F(2.13, 21.26)=3.24, p=.057$, but a robust quadratic effect, $F(1,10)=12.64, p<.01$, eta$^2=.56$; RT was slower following the 4th

### Table 2

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Sleep restriction (Day 1)</th>
<th>Sleep restriction (Day 4)</th>
<th>Sleep restriction (Day 7)</th>
<th>Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjective variables</td>
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<tr>
<td>Subjective sleepiness</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
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<td>Total mood score</td>
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<td>14.92</td>
<td>11.91</td>
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<td>7.47</td>
<td>2.52</td>
<td>2.62</td>
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<tr>
<td>F-scale</td>
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<td>6.85</td>
<td>4.08</td>
<td>11.69</td>
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<td>3.08</td>
</tr>
<tr>
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<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Mean RT</td>
<td>337.52</td>
<td>70.28</td>
<td>348.83</td>
<td>66.47</td>
<td>377.92</td>
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<tr>
<td>Median RT</td>
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<td>69.15</td>
<td>327.09</td>
<td>65.46</td>
<td>356.68</td>
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<td>Slowest 10% RTs</td>
<td>83.06</td>
<td>20.16</td>
<td>94.06</td>
<td>28.36</td>
<td>105.14</td>
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<tr>
<td>Fastest 10% RTs</td>
<td>499.81</td>
<td>99.21</td>
<td>532.29</td>
<td>134.60</td>
<td>586.00</td>
</tr>
<tr>
<td>No. of lapses</td>
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<td>58.04</td>
<td>263.71</td>
<td>60.45</td>
<td>276.18</td>
</tr>
<tr>
<td>% Accuracy</td>
<td>91.36</td>
<td>11.09</td>
<td>93.67</td>
<td>6.43</td>
<td>89.51</td>
</tr>
</tbody>
</table>

POMS subscales: Vigor–Activity (V), Fatigue–Inertia (F), Tension–Anxiety (T), Confusion–Bewilderment (C), Depression–Dejection (D), Anger–Hostility (A).
Fig. 2. Dynamic topography of alpha EEG during sleep restriction. At frontal sites, a linear decline in EEG arousal is seen over the week. At posterior sites, changes occur only at the end of sleep restriction week. Note: values represent the mean log power (with standard error indicated).
day of sleep restriction compared to both baseline (p < .05) and recovery (p < .01); RT at recovery was also faster than the 7th (p < .01) day of sleep restriction. Similarly, there was a trend for the main effect of lapses, F(1,59, 15.89) = 3.57, p = .061, with a significant quadratic effect, F(1,10) = 7.16, p < .05, eta^2 = .42; there were a greater number of lapses in response time following the 4th day of sleep restriction compared to both baseline (p < .05) and recovery (p < .01); there was also a trend for more lapses following the 7th day of sleep restriction compared to recovery (p = .050). The standard deviation of RTs did not yield significant differences.

2-back memory task. Mean RT, the standard deviation of RTs, and percent accuracy on the 2-back memory task were assessed. One participant was removed because accuracy data indicated a failure to understand the task, and two others were excluded because of missing data in at least one condition. There was a trend for a condition effect for mean RT, F(4,40) = 2.24, p = .08, and a quadratic effect, F(1,10) = 12.04, p < .01, eta^2 = .55, which depicted the expected effects of sleep restriction (see Table 2 for means). Mean RT was faster following one night of recovery sleep compared to the 4th (p < .001) day of restriction, with similar trends compared to the 1st (p < .06) and 7th (p < .09). The standard deviation of RTs and accuracy variables did not yield significant differences.

Physiological data. EEG. Absolute power in pre-defined EEG frequency bands (theta, low frequency alpha, high frequency alpha) was examined during eyes opened and closed recording sessions. No outliers were identified in EEG power. Only low frequency alpha (8–10 Hz) during eyes closed recording sessions varied across study condition: alpha was higher at baseline (p < .05) compared to all other conditions at posterior sites (P3, Pz, P4, P7, P8, O1, O2), and higher after the 1st night of sleep restriction (p < .05) compared to the 7th night of sleep restriction restriction compared to baseline, 1st, 4th, 7th nights of restriction, and recovery). One-way ANOVA to compare across the five laboratory testing sessions (baseline, 1st, 4th, 7th nights of restriction, and recovery). Only the AAC calculated with low frequency alpha yielded significance. Results showed greater physiological sleepiness following the 7th night of sleep restriction compared to baseline, 1st night of restriction, 4th night of restriction, and recovery at Fz, Cz, and C4 (p < .05), and a trend for the same outcome at Pz. In addition, at Fz, there was greater physiological sleepiness following the 4th night of restriction (p < .05) compared to baseline. No other sites were significant. To further investigate the scalp topography of EEG slowing during the sleep restriction, follow-up one-way ANOVAs were run to compare the AAC across the week of sleep restriction. Changes in physiological alertness emerged as follows (refer to Fig. 2 for illustration of topography): the 1st day differed from the 7th day of restriction at right-hemisphere anterior sites (Fp2, Fz, F8, T8), and significant linear polynomials also depicted this continuous decline in arousal; the 4th day differed from the 7th day of restriction at right-hemisphere posterior sites (P4, Pz, with trends at P7/P8); and both the linear decline from the 1st to the 7th night of sleep restriction as well as the sharp decline from the 4th to the 7th night of sleep restriction were seen at right-hemisphere central sites (Cz, C4). There were trends for similar effects at left-hemisphere sites (see Table 3), which shows that although the decrease in physiological alertness was widespread across the scalp, the effects in the right-hemisphere were more robust.

Event-related potentials. Behavioural performance on the pitch and duration oddball tasks was largely unaffected by sleep restriction. Percent accuracy to both target stimuli (i.e., providing

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Results of 3-way ANOVAs on the Alpha Attenuation Coefficient (AAC)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Region</td>
<td>Site</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal</td>
<td>Fp1</td>
</tr>
<tr>
<td></td>
<td>Fp2</td>
</tr>
<tr>
<td></td>
<td>F3</td>
</tr>
<tr>
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</tr>
<tr>
<td></td>
<td>F7</td>
</tr>
<tr>
<td></td>
<td>F8</td>
</tr>
<tr>
<td>Central</td>
<td>C3</td>
</tr>
<tr>
<td></td>
<td>C4</td>
</tr>
<tr>
<td></td>
<td>C5</td>
</tr>
<tr>
<td>Temporal</td>
<td>T7</td>
</tr>
<tr>
<td>Parietal</td>
<td>T8</td>
</tr>
<tr>
<td></td>
<td>P3</td>
</tr>
<tr>
<td></td>
<td>P4</td>
</tr>
<tr>
<td></td>
<td>P7</td>
</tr>
<tr>
<td>Occipital</td>
<td>P8</td>
</tr>
<tr>
<td></td>
<td>O1</td>
</tr>
<tr>
<td></td>
<td>O2</td>
</tr>
</tbody>
</table>

Note: SR1, SR4, and SR7 represent the 1st, 4th, and 7th nights of sleep restriction. Pairwise comparisons are reported for trends to show topographic pattern of EEG changes. Significance where p < .05 appears in bold.
response) and standard stimuli (i.e., inhibiting response) did not vary significantly by condition. Similarly, the one-way ANOVAs comparing across baseline, 1st, 4th, 7th nights of restriction, and recovery, did not yield significance for mean RT to target stimuli for either the pitch or duration oddball tasks. However, closer examination of the sleep restriction week alone showed that mean RT to pitch targets was significantly slower following the 7th night compared to 4th night of restriction \((p < .05), F(2,20) = 3.66, p < .05, \eta^2 = .27\). There was a trend for mean RT to duration targets, \(F(2,20) = 3.11, p = .067\), driven by RTs being slower following 7th night compared to 4th night of restriction \((p < .01)\). N1 and P2 amplitude and latency were measured following standard stimuli. For both oddball paradigms, the predicted effects of sleep restriction were not apparent at any site for N1 and P2 latency or amplitude. For the pitch oddball task, there were significant condition effects and a linear increase in P2 amplitude over the five study conditions that were apparent at all frontal sites \((p s < .05)\). There were similar effects found for P2 amplitude for the duration task, however, the effects were not as statistically robust.

The average amplitude of the long-latency P300 was measured from 300–450 ms for the pitch stimuli and from 400–650 ms for the duration stimuli. For both analyses, one outlier was removed.

![Figure 3](image_url)

**Fig. 3.** A. Means plot of P300 amplitude at Pz following pitch targets. Note that the P300 waveform becomes smaller (i.e., less positive) during sleep restriction, and returns to baseline following recovery. B. ERPs to pitch targets at parietal sites. P300 is noted as a large upward deflection occurring from 200–500 ms. Baseline=thin solid, SR1=thin, dashed, SR4=thin, broken line, SR7=thick solid, and Recovery=thick, dashed line.
because of extreme values at parietal sites during baseline (i.e., negative amplitude). For the pitch oddball task, one-way ANOVAs were run at each site to compare P300 amplitude across the five study conditions. There was a trend for the expected reduction in P300 amplitude at Pz, $F(4,28)=2.59, p=0.058$, with a significant quadratic effect, $F(1,7)=6.40, p<0.05, \eta^2=.48$. P300 was smaller (less positive) on the 7th day of sleep restriction compared to both 1st day of sleep restriction ($p<0.05$) and recovery ($p<0.05$), with a trend for also being smaller than baseline ($p=0.069$). See Fig. 3 for the means plots of P300 amplitude changes across condition at Pz and the grand average ERP waveforms at parietal sites. There was also a significant quadratic effect for the right-hemisphere parietal site, P4, $F(1,7)=7.69, p<0.05, \eta^2=.52$, although the condition effect was not significant. There were no significant effects or trends at the left-parietal lead, P3, nor at any other sites recorded. P300 amplitude to the target stimuli did not vary reliably across condition for the duration oddball task.

3. Discussion

Data from weekly averages of sleep diaries and actigraph monitors showed that participants complied with the instruction to curtail sleep at home. Behavioural data collected from home showed some effects of sleep restriction with subjective sleepiness and mean RT being reduced during the week of sleep restriction compared to baseline. There was no evidence of improvement during the recovery week for data collected from home, and moreover there were linear declines over the study period seen in mood and RT lapses suggesting problems with participants’ motivation when data were collected at home. Measurement taken during the laboratory sessions revealed more robust effects in the expected direction for sleepiness, mood, and RT. These behavioural findings were consistent with previous studies of continuous sleep restriction [39,41,42], further validating that sleep was successfully manipulated at home. Electrophysiological measures of EEG and ERPs showed dynamic changes in arousal and attention as a result of sleep restriction. Specifically, EEG data indicated that anterior brain regions showed immediate and continuous reductions in arousal, whereas posterior regions did not show changes until later in the sleep restriction week. Impairment in attention also emerged toward the end of the sleep restriction period, as seen in the P300 component of the ERP which was reduced in amplitude at parietal sites where the component is typically largest. Both EEG and ERP data revealed that the effects of sleep restriction were more robust in the right-hemisphere, perhaps demonstrating greater resiliency of the dominant hemisphere. In summary, a number of different variables converge to show that there are deficits in performance and brain function which are apparent after four and seven nights of continuous sleep restriction.

One of the main goals of the present study was to apply physiological measures of arousal and attention using EEG and ERP techniques to investigate the CNS nature of performance deficits during sleep restriction. EEG topography showed that the brain regions affected by sleep loss were dynamic over the course of sleep restriction. Specifically, quantitative analysis of the EEG showed that there were significant differences between the 1st and 7th days of sleep restriction with significant linear decreases in arousal at frontal sites, depicting an immediate and continuous reduction in physiological alertness over the sleep restriction week. However, at parietal sites, there was relative stability between the 1st and 4th days of restriction, with deficits becoming apparent only later in the week, with a sharp decline between the 4th and 7th days of restriction. Both the linear decline in arousal over the week and the more precipitous drop at the end of the week was apparent at central sites. These data provide support for CNS changes in arousal that underlie the oft-reported deficits in performance following sleep restriction. The immediate and continuous deficits apparent at frontal EEG sites are consistent with the hypothesis that frontal lobe impairment is responsible for the behavioural deficits seen during sleep loss [7]. Furthermore, the localization of arousal changes to frontal regions is consistent with EEG studies following total sleep deprivation [17,26,27], and consistent with some imaging data showing reduced activity in frontal brain regions following total sleep deprivation [8–11]. At least initially, CNS function in the parietal regions seemed to be maintained, providing evidence for theories of brain compensation that have been proposed [15]. The parietal brain regions may simply be able to function longer during the challenge or stress of sleep deprivation. As the duration of sleep restriction continued, deficits also became apparent in parietal brain regions. Presumably, either compensation fails at this point, or the extent of brain regions compromised by sleep restriction becomes more widespread. These data suggest that the length or duration of continuous sleep restriction may be a more important indicator of the magnitude of deficits than the degree of sleep loss. In other words, the brain may be capable of adaptation or compensation for short periods even when the sleep loss is extreme (e.g., 24 h); however, the brain may not be adaptable when there is a chronic lack of sufficient sleep.

ERPs to the target stimuli in the pitch oddball task revealed reduced P300 amplitude at parietal sites during sleep restriction. These findings are consistent with previous studies that found reductions in P300 amplitude following total sleep deprivation [31–34,36,37]. Thus, sleep restriction, a more subtle degree of sleep loss that is commonly experienced, led to impairment in the ability to allocate attention to target discrimination or possibly deficits in memory updating [35,51]. Earlier components, N1 and P2, were measured from the frequently occurring standard stimuli. It was expected that N1 would be smaller and P2 would be larger in amplitude during sleep restriction representing reduced attention toward the less meaningful non-target stimuli [52]. There were no effects of sleep restriction for the amplitude or latency of these components that index early stages of information processing (e.g., stimulus identification and encoding). However, P2 amplitude increased linearly over all study conditions (baseline, restriction, and recovery). It is possible that the linear increase in P2 amplitude over the study protocol reflects reduced motivation or effort on the mundane task over repeated measures. Lee et al. noted a similar increase in P2 amplitude over 2 days of total sleep deprivation which they related to mood and anxiety [33]. Given the linear changes in mood reported here, it may be that the systematic increase in P2 amplitude over time can be explained by emotion dysregulation as a result of both the sleep loss and the lengthy paradigm combined. ERPs in the duration oddball task did not vary with sleep restriction. This outcome was surprising because we expected that this relatively more difficult stimulus discrimination task would be more sensitive to the effects of sleep loss. Indeed there is evidence that the duration task was more difficult: accuracy was greater, RT was faster, and P300 amplitude was higher at baseline for the pitch targets (90.72%, 376.82 ms, 8.80 µV) compared to the duration targets (88.50%, 542.73 ms, 7.24 µV). However, because the duration task was more difficult or challenging, participants may have exerted more effort on the task. This increased attention to or interest in the task might have increased P300 amplitude, masking any decreases due to sleep loss.

Both EEG and ERPs showed that deficits were more apparent at right-hemisphere sites. Although the pattern of physiological change in arousal and attention was similar for left-hemisphere sites, the deficits were more statistically robust in the right-hemisphere. This effect cannot be explained by the choice of reference for EEG recordings because analysis of the EEG and ERPs was carried out on data that was re-referenced to the average of A1 and A2 sites, effectively created a balanced, central reference. All participants in the study were right-handed. Thus, one possibility is that the EEG laterality observed might be due to movement time or use of the preferred right hand. However, no response or movement occurred during the AAT task when EEG data was recorded, and responses to the target tones in the ERP tasks were given with a hand-held button
response (while this response involved use of the right hand, there was minimal movement needed to make the response). Given that all participants were right-handed, and therefore likely left-hemisphere dominant, the laterality of the EEG/ERP effects may reflect the functional superiority of the dominant hemisphere during a sleep loss challenge, i.e., the left-hemisphere was less vulnerable to the effects of sleep loss. Phrased in a way that is consistent with a compensation hypothesis of brain functioning during sleepiness, the dominant hemisphere may work harder to maintain function (or have more energy directed toward it), while the non-dominant hemisphere is compromised during a challenge.

There were differences in outcome between the home and laboratory behavioural data. The behavioural data assessed in the laboratory showed the predicted effects of sleep restriction which have been reported in previous studies [39,41,42]. Specifically, there were quadratic effects depicting a change from baseline to restriction and improvement in recovery for subjective sleepiness, mood, perception of performance, and reaction time. Behavioural data collected while participants were at home, however, was not as statistically robust and failed to show the recovery seen in the laboratory data. Moreover, linear declines in data for mood and RT lapses that were collected from home suggest that there was a problem with motivation or effort during the lengthy 3-week protocol when data was assessed in the home environment. Based on the results of the validation data (actigraph and diaries), and the outcome of the behavioural and electrophysiological data collected in the laboratory, it is clear that the manipulation of sleep was generally successfully at home. There are a number of possible explanations for the lack sensitivity in the behavioural data collected from home. Participants may have taken the assessments in the laboratory more seriously and exerted a more sincere effort because of the presence of research assistants or physiological recordings. Similarly, motivation for home assessments may have been reduced in the final week of data collection because participants were not required to return for laboratory assessment after the 1st night of recovery. In other words, had there been an additional laboratory assessment at the end of the recovery week, participants may have maintained their effort and motivation for the behavioural data collected from home. Another issue is that the home data was measured five times across the day; the number and frequency of data collection sessions may have made the task too demanding or intrusive for participants. Although participants were instructed to perform the RT and well-being assessments in a secluded quiet area, it is likely that this was not always possible, and therefore the data may be confounded by a variety of diversions (e.g., noise from TV or conversation, needing to leave class or stop driving in order to perform the task). Another possible explanation for the home versus laboratory differences may be that the mode of presentation for the RT tasks and the well-being scales were different in the two settings. For example, the RT task administered in the laboratory was done on a computer and responses were made using the keypad with the index finger of the preferred hand, while the RT task at home was done using a hand-held device that required a response with the thumb. In addition, the assessment of mood and sleepiness in the laboratory was done on paper using the extensive POMS questionnaire and a 7-point sleepiness scale, while the home assessment of mood and sleepiness was done on the hand-held electronic device using visual analog scales (i.e., sad–happy; sleepy–alert). Although the constructs were intended to be the same, these variations in presentation may have contributed to differences in the sensitivity between the laboratory and home measures. In summary, in the present study, while sleep could be successfully manipulated in the home environment with the aid of compliance checks like diaries and actigraphy monitors, the validity of the behavioural data collected at home is questionable in comparison to the laboratory environment. It would be beneficial to further investigate the conditions under which reliable home data can be collected. Home assessment of sleep and performance data increases the external validity achieved in the research design, permits long-term monitoring, and allows for easier study of applied populations. Moreover, home monitoring allows researchers to work with larger samples, thereby permitting more systematic evaluation of circadian factors and performance instability over time.

There are a number of design issues which should be considered in the interpretation of the data and will aid in design of future research. Over the long 21-day protocol, we decided to investigate electrophysiology and performance in the laboratory on 5 different occasions, at the end of baseline, three times throughout restriction, and following the 1st day of recovery. In the current study, participants as a group did not appear to significantly curtail their sleep on the first night of restriction (see Fig. 1). This unsuccessful manipulation may explain why deficits were not always apparent after the first night of sleep restriction. Thus, data from days 11 and 14 in the current study (after 4 and 7 nights of restriction) are more representative of the effects of sleep restriction. In addition, this behavioural data showed that participants as a group did not successfully obtain a greater amount of sleep after a single night of recovery sleep. Therefore, it would have been better to observe recovery later in the week. The schedule of the laboratory recording sessions in the present study likely accounts for the lack of improvement on many variables during recovery sleep. Future studies may monitor the daily sleep of individuals more closely either at home or in the laboratory, removing participants who do not meet the nightly reductions in sleep as instructed.

The present study contributes to the body of knowledge about the effects of sleep loss on waking function in a number of ways. First, the data demonstrate that one week of moderate sleep restriction is associated with cumulative changes in physiological measures of arousal and attention, in addition to the oft-reported behavioural performance deficits. These deficits are consistent with what has been seen following total sleep deprivation. Second, the EEG and ERP techniques revealed dynamic effects of sleep restriction over time which illustrates that various brain regions are affected differentially depending on the length of sleep restriction. Moreover, hemispheric effects showed that the dominant hemisphere is more resilient to the effects of sleep restriction. Lastly, there was successful manipulation of sleep using a home-based study, however, the lack of recovery in home data and the discrepancy with the laboratory data raises concern as to the participants’ motivation. Further studies are needed to assess the degree of control that can be achieved during home assessment of sleepiness and performance. The effects of sleep restriction can be quite subtle, yet this insidious level of sleepiness can lead to dangerous consequences. More studies are needed to map the CNS and performance deficits that follow levels of sleepiness that are commonly experienced (e.g., as in continuous sleep restriction, sleep fragmentation, and shift work). Examining changes in arousal and attention that occur over time and the associated brain topography will inform us about regional brain functioning and adaptation during sleepiness.

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References


