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Efficient and Regular Patterns of Nighttime Sleep are Related to Increased Vulnerability to Microsleeps Following a Single Night of Sleep Restriction

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Sleep-deprived people, or those performing extended monotonous tasks, can exhibit brief episodes in which they suspend performance and appear to fall asleep momentarily—behavioral microsleeps (''*microsleeps*''). In this study, microsleeps were identified using eye video and tracking response during a 20-min continuous tracking task undertaken by 16 healthy volunteers (mean age 24.9 yrs; 8 females, 8 males) in the early afternoon following a normally rested night and a night of restricted sleep (time-in-bed restricted to 4 h). Sessions were 1 wk apart and counterbalanced. Wrist actigraphy, self-reported sleepiness, and sleep quality were also recorded. We hypothesized that high microsleep rates when normally rested or after a night of sleep restriction would be related to poor sleep quality, sleep disturbance, circadian type, irregular sleep patterns, low daily sleep duration, or poor sleep efficiency. We also hypothesized that prior performance on a 10-min psychomotor vigilance task (PVT) (mean reaction time or number of PVT lapses) would be related to the number of microsleeps during the tracking task and that PVT performance could, therefore, be used as a fitness-for-duty indicator. The number of microsleeps during the tracking task increased following sleep restriction (mean 11.4 versus 27.9; $p = 0.03$). There were no correlations between the number of microsleeps in the normally rested session and any of the actigraphically measured or self-reported sleep measures. However, the number of microsleeps following sleep restriction was correlated with sleep efficiency ($r = 0.73$, $p = 0.001$), sleep onset latency ($r = -0.57$, $p = 0.02$), and sleep onset time-of-day standard deviation ($r = -0.54$, $p = 0.03$) over 11 normally rested nights. There was no correlation between PVT performance and the subsequent number of microsleeps during the tracking task in either session. Attributes usually associated with beneficial nighttime sleep patterns—going to sleep at a similar time each night, falling asleep quickly, and infrequent arousals—were related to greater vulnerability to microsleeps following sleep restriction. There were intercorrelations between all the sleep measures associated with microsleep rate following sleep restriction, indicating that the measures form a pattern of behaviors and are not independently related to microsleep rate. Perhaps some people maintain a regular sleep pattern because they experience sleepiness the following day when their pattern is disrupted. Conversely, people with more variation in their sleep pattern may do so because this does not substantially increase sleepiness the following day. We conclude that people with consistent sleep patterns and efficient sleep may be more prone to microsleeps than other people when their usual regular pattern is disrupted by sleep restriction.

Keywords: Actigraphy, healthy adults, interindividual differences, lapses, sleep habit flexibility, sleep parameters, sleepiness, tracking task

INTRODUCTION

Individuals who are sleep-deprived or performing an extended monotonous task can exhibit delayed or absent responses, including brief (0.5–15 s) episodes during which performance is suspended and they appear to fall asleep momentarily—behavioral microsleeps (''*microsleeps*'') (Chou et al., 2011; Davidson et al., 2007; Peiris et al., 2006; Poudel et al., 2008, 2013; Sommer et al., 2009). During an active task, these manifest as a complete failure to respond, accompanied by droopy eyes, slow-eye-closure, and head nodding (Peiris et al., 2006; Sommer et al., 2009; Torsvall & Akerstedt, 1988). Unintentional suspension of performance is of particular concern in occupations in which

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public safety depends on extended unimpaired functioning (e.g., drivers, pilots, air traffic controllers, health professionals, and process control workers) (Bourgeois-Bougrine et al., 2003; Horne & Reyner, 1999).

Large interindividual differences, but which are stable and trait-like within individuals, have been reported for subjective sleepiness, cognition, and vigilance following sleep deprivation (Berka et al., 2005; Chee & Tan, 2010; Chuah et al., 2006; Goel et al., 2010; Landolt, 2008; Leproult et al., 2003; Van Dongen et al., 2004, 2005), and in sleep electroencephalogram (EEG) architecture (Buckelmuller et al., 2006; Gander et al., 2010; Van Dongen, 2006). Microsleep rates have also been observed to vary greatly between normally rested people (Peiris et al., 2006; Poudel et al., 2010, 2013). Using conservative criteria, one study observed a range of 0–72 (mean 15.2) microsleeps per hour during a continuous monotonous tracking task undertaken in the early afternoon across 15 young healthy normally rested individuals: 7 had none, 6 had 15–30, and 2 had >60 over an hour. In a second study of 20 normally rested participants, we observed 14 had at least 36 microsleeps during a 50-min tracking task, 2 had 1–2, and 4 had none (Poudel et al., 2013). In a repeated-measures design study, interindividual differences in microsleep rates have shown substantial agreement across two sessions (single-rater intraclass correlation$_{(2,1)} = 0.72$, $p < 0.001$) (M. Peiris, personal communication).

In the current study, we identified microsleeps based on eye video and response characteristics during a continuous tracking task in the early afternoon following a normally rested night or a night of restricted sleep. Following our previous behavioral, electroencephalography (EEG), and functional magnetic resonance imaging (fMRI) studies of microsleeps in normally rested participants (Innes et al., 2010; Peiris et al., 2006, 2011; Poudel et al., 2013), we hypothesized that the high rates of microsleeps observed in some of our ostensibly non-sleep-deprived people would simply be due to poor sleep quality, sleep disturbance, circadian type, irregular sleep patterns, low daily sleep duration, or poor sleep efficiency. To test this hypothesis, we aimed (1) to determine the relationship between daytime microsleep rate when normally rested and when sleep-restricted, and (2) to determine the relationship between microsleep rate when normally rested or sleep-restricted and (a) actigraphic nighttime sleep parameters during 11 normally rested nights or one sleep-restricted night and (b) subjective self-reported measures of sleep propensity (Epworth Sleepiness Scale), sleep quality (Pittsburgh Sleep Quality Index), and circadian type (Horne-Ostberg Morning-Eveningness Questionnaire). In addition, we hypothesized that performance on a well-characterized 10-min vigilance task—the psychomotor vigilance task (PVT) (Dinges & Powell, 1985)—could be used as a ''fitness-for-duty'' tool to predict microsleep rate during the subsequent tracking task when normally rested or sleep-restricted.

METHODS

Participants

Sixteen right-handed volunteers (8 males and 8 females, aged 20–37 yrs, mean age 24.9 yrs) with no history of neurological, psychiatric, or sleep disorder participated in the study. For inclusion in the study, participants had to report a usual time to bed between 22:00 h and midnight and a usual time-in-bed of between 7.0 and 8.5 h. Ethical approval for the study was obtained from the New Zealand Upper South B Regional Ethics Committee. Written consent was obtained from participants. The experimental protocol used in this study conforms to international ethical standard as outlined in Portaluppi et al. (2010).

Study Procedure

All participants visited the laboratory three times. On the first visit, they were briefed on the experimental protocol and provided with an Actiwatch (Respironics, Murrysville, PA, USA) to record their sleep habits for 7 days and 6 nights prior to the two experimental sessions. Measures included in the actigraphy analysis were (1) time-in-bed (length of time from first attempt to sleep at the beginning of the sleep period to final wake time at the end of the sleep period), (2) sleep onset latency (length of time taken from first attempt to sleep to actual sleep onset), (3) assumed sleep (time-in-bed less sleep onset latency), (4) wake time during assumed sleep, (5) actual sleep (assumed sleep less wake time), (6) sleep efficiency (actual sleep/time-in-bed), (7) sleep onset time-of-day (time of sleep onset), and (8) sleep onset time-of-day standard deviation.

Participants also completed a detailed sleep diary to record their estimated time-to-bed, time of sleep onset, periods of wake time (>10 min) during the sleep period, and time to wake at the end of the sleep period for 7 days and 6 nights prior to the two experimental sessions. They also recorded time of intake of stimulants (e.g., caffeine) and depressants (e.g., alcohol) and food in the diary.

Participants also completed a set of questionnaires to assess their self-reported everyday sleep propensity (Epworth Sleepiness Scale [ESS]), sleep quality (Pittsburgh Sleep Quality Index [PSQI]), and circadian type (Horne-Ostberg Morning-Eveningness Questionnaire [MEQ]).

The ESS is a validated sleep questionnaire measuring daytime sleep propensity by means of the participant subjectively assessing the likelihood of falling asleep in eight situations commonly encountered in daily life (Johns, 1991). Possible scores range from 0 (no chance of dozing in any of the eight situations) to 24 (high chance of dozing in all eight situations).

The PSQI subjectively assesses sleep quality and disturbances during the past month (Buysse et al., 1989). Nineteen individual items generate seven scores: subjective sleep quality, sleep latency, sleep duration,

habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The sum of the seven scores yields a global score with a range of 0–21, with higher scores indicating worse sleep quality and greater sleep disturbance.

The Horne-Ostberg MEQ assesses a person's circadian type, by asking questions on preferential sleep timing and activities (Horne & Ostberg, 1976). The majority of questions are 4-choice items, which lead to an overall score between 16 and 86. The overall score corresponds to definite evening type (16–30), moderate evening type (31–41), neither type (42–58), moderate morning type (59–69), and definite morning type (70–86).

The second and third visits involved sleep-restricted and normally rested sessions, the order of which was counterbalanced across the participants. The sessions were 1 wk apart to minimize residual effects of sleep restriction in participants who were sleep-restricted during the first session.

The participants were asked to sleep normal hours during the week prior to the normally rested session (e.g., to go to bed between 22:00 h and midnight and have a time-in-bed of 7.0–8.5 h). They were also asked to do likewise for the sleep-restricted session except for the immediately preceding night in which their time-in-bed was restricted to 4 h (3:00–7:00 h). Participants were requested not to engage in any safety-sensitive tasks (such as driving) following the sleep restriction. They were also asked not to consume any stimulants or depressants, such as alcohol, caffeine, and nicotine, on the day of either experimental session.

Sleep habits recorded by the Actiwatch and in the sleep diary were inspected prior to experimental sessions to confirm compliance with the sleep schedule required for inclusion in the study.

Experimental sessions commenced at either 13:30 or 14:30 h. Just before the start of the experimental sessions, participants were provided with a lunch of hot noodles and asked to rate their current subjective sleepiness using the Karolinska Sleepiness Scale (KSS) (Åkerstedt & Gillberg, 1990) and Stanford Sleepiness Scale (SSS) (Hoddes et al., 1972). The KSS requires the participant to rate their current sleepiness on a 9-point scale with descriptions at every second point: 1 = extremely alert; 3 = Alert, 5 = Neither alert nor sleepy; 7 = Sleepy—but no difficulty remaining awake; and 9 = Extremely sleepy—fighting sleep. The SSS requires the participant to rate their current sleepiness on an 7-point scale with descriptions at every point: 1 = Feeling active, vital, alert, or wide awake; 2 = Functioning at high levels, but not at peak; able to concentrate; 3 = Awake, but relaxed; responsive but not fully alert; 4 = Somewhat foggy, let down; 5 = Foggy; losing interest in remaining awake; slowed down; 6 = Sleepy, woozy, fighting sleep; prefer to lie down; 7 = No longer fighting sleep, sleep onset soon; having dream-like thoughts; or X = Asleep (with X scored by the assessor).

Participants also undertook a 10-min computerized psychomotor vigilance task (PVT) (Dinges & Powell, 1985) in which they were asked to press a button as soon as they saw a counter start counting up in the center of a screen. Pressing the button stopped the counter and this number was displayed on the screen for 1.0 s to indicate the participant's reaction time. Reaction times longer than 500 ms were considered to be ''PVT lapses'' (Dinges et al., 1997).

Experimental Task and Behavioral Recordings

Participants performed a 20-min tracking task while lying supine in an MRI scanner that was used in a concomitant study of the effect of sleep restriction on resting cerebral blood flow (Poudel et al., 2012) and microsleep-related functional MRI blood oxygen level–dependent (fMRI BOLD) activity. Joystick response and eye video were recorded synchronously. Video of the right eye was captured using a Visible Eye system (Avotec, Stuart, FL, USA) mounted on the head coil of the MRI scanner. The video was recorded on a personal computer at 25 frames per second (fps) (350×280 pixels) using a video-capture card and custom-built video recording software.

During the visuomotor tracking task, participants maneuvered an MR-compatible finger-based joystick (Current Designs, Philadelphia, PA, USA), sampled at 60 Hz, to pursue a two-dimensional (2D) random target moving continuously on a computer screen (Figure 1a). The horizontal and vertical components of the target were produced by a sum-of-sines ($n = 7$) with frequencies evenly spaced from 0.033 to 0.231 Hz. The target amplitude was scaled to fit 80% of the 1024×768-pixel resolution screen. This produced a 2D periodic target (T = 30 s) with a velocity range of 63–285 pixels/s. The target (yellow disc, d = 23 pixels) and the joystick response (red disc, d = 20 pixels), generated by a custom-designed software, were presented via MRI-compatible goggles (Avotec) with a field of view of $30^\circ \times 23^\circ$. The background pattern was gray. Participants were familiarized with the tracking task and instructed to control the joystick position so that the response disc was as close as possible to the center of the moving target at all times. Foam support was placed below the right elbow for subject comfort and to minimize hand movement during tracking.

Data Analysis

To identify individual microsleeps, a custom-built SyncPlayer program was used to replay synchronized eye video, and tracking target (x and y), response (x and y), speed, and tracking error. The tracking error was defined as the Euclidean distance between the centers of the target and response discs. Any episodes of flat tracking (zero response speed) of 0.5–15-s duration accompanied by behavioral signs of drowsiness and full

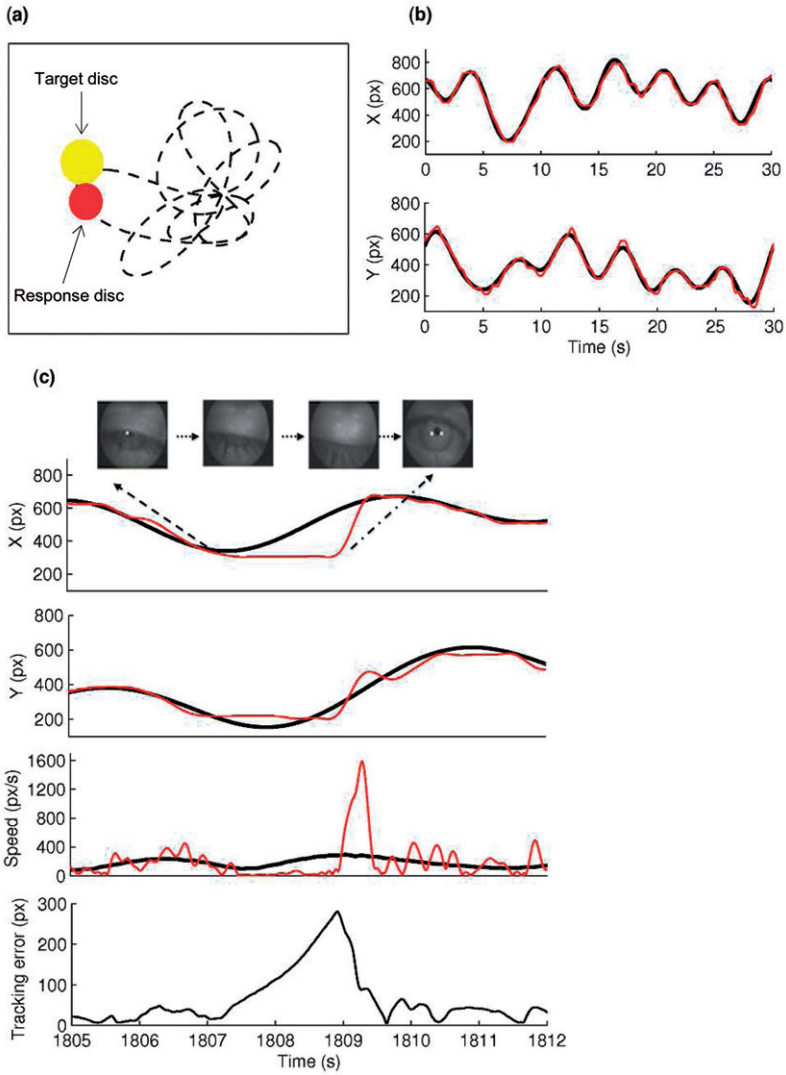

FIGURE 1. Tracking task and eyelid and response behavior associated with a typical microsleep during the tracking task. (a) Participants used a finger-based joystick to track the displayed yellow target disc moving in a quasi-random trajectory (dotted line) with a red response disc. (b) Accurate tracking led to the movement of the response disc along the same trajectory as the target disc as displayed in the target (smooth black line) and response (jerky line) position for one cycle (30 s) of tracking. (c) Tracking response (jerky line) is flat in both directions (leading to an increase in tracking error and zero speed) and eyes slowly close during a typical microsleep. The speed of the tracking response (jerky line) shows a typical fast corrective movement at the end of the microsleep. The units are in pixels (px).

or partial (>80%) slow-eye-closures were marked as microsleeps. The response position, speed, and error signals were used to mark the onset and end of flat tracking responses (Figure 1c). Eye video was used as a cue to mark the onset and end of eye closure.

A *t* test for dependent samples was used to determine whether there was an increase in the number of daytime microsleeps during the tracking task following sleep restriction compared with baseline. A Pearson's correlation was then used to investigate any relationship between the number of microsleeps in the normally rested and sleep-restricted sessions. Pearson's correlations were also used to investigate the relationship between the number of daytime microsleeps observed following normal rest or sleep restriction and actigraphic sleep measures or ESS, PSQI, MEQ, SSS, or KSS scores. Linear regression analyses were conducted to determine the relative contribution made by any measures related to number of microsleeps in either session to the overall prediction of microsleep number. Finally, a Pearson's correlation was used to determine whether there was a relationship between PVT performance (mean reaction time or PVT lapses) and the number of subsequent microsleeps during the following normal rest or sleep restriction.

To ensure there were no significant differences in sleep prior to the two experimental sessions (other than the single night of sleep restriction), a *t* test for dependent samples was used to examine actigraphic sleep measures in the week prior to the normally rested and sleep-restricted sessions (excluding the final night).

RESULTS

Actigraphic Sleep Measures

In line with our sleep-restriction protocol, there was a decrease in actual sleep during the sleep-restricted night compared with the night prior to the normally rested session (mean 3.5 versus 8.0 h; $t(15) = 21.51$, $p < 0.001$, one-tailed). There were no other differences in sleep in the night or week prior to the two experimental sessions (see Table 1).

Self-reported Sleep Measures

Participants reported a mean ESS score of 5.0 ± 2.8 (range: 2–11), mean global PSI score of 4.7 ± 2.2

TABLE 1. Actigraphic sleep measures in the week or night before each experimental session.

Sleep measures	Prior to normally rested session	Prior to sleep-restricted session	*t* Test for dependent samples: *t* Statistic	*t* Test for dependent samples: *p* Value
Prior week†				
Mean actual sleep duration (h)	7.24 ± 0.7	6.99 ± 0.4	1.56	0.14
Mean sleep onset latency (min)	16.6 ± 13.4	16.9 ± 10.1	−0.08	0.93
Mean sleep efficiency (%)	86.7 ± 5.3	86.1 ± 4.1	0.56	0.59
Prior night				
Actual sleep duration (h)	8.02 ± 0.9	3.51 ± 0.2	21.51	0.00*
Sleep onset latency (min)	12.4 ± 10.2	8.2 ± 7.6	1.30	0.21
Sleep efficiency (%)	86.5 ± 4.9	87.0 ± 6.0	−0.36	0.73

†Excluding the night immediately preceding the experimental session.
*$p < 0.05$.

(range: 2–10), and mean MEQ score of 57.1 ± 7.1 (range: 38–67). Females reported lower ESS scores than males (mean 3.6 versus 6.4; $t(14) = -2.17$, $p = 0.05$, two-tailed). Using the 85th percentile of ESS scores in a normal population (≥10 in females and ≥12 in males) as an indication of ''excessive daytime sleepiness'' (Whitney et al., 1998), none of the participants in the study met the criterion for excessive sleepiness. On the MEQ, no participants were scored as a Definite Morning or Evening type, whereas 7 were Moderately Morning types, 8 were Neither type, and 1 was a Moderately Evening type.

There was an increase in self-rated sleepiness between the normally rested and sleep-restricted sessions as rated by SSS (mean 1.9 versus 3.0; $t(15) = -6.07$, $p < 0.001$, one-tailed) and KSS (mean 3.1 versus 5.2; $t(15) = -6.12$, $p < 0.001$, one-tailed). Females reported higher subjective sleepiness than males during the normally rested session as rated by SSS (mean 2.5 versus 1.3; $t(14) = 5.00$, $p < 0.001$, two-tailed) and KSS (mean 3.6 versus 2.5; $t(14) = 3.00$, $p < 0.01$, two-tailed). However, there was no difference in subjective sleepiness between females and males during the sleep-restricted session as rated by SSS (mean 3.4 versus 2.6; $t(14) = 1.79$, $p = 0.10$, two-tailed) or KSS (mean 5.6 versus 4.8; $t(14) = 1.04$, $p = 0.32$, two-tailed).

Psychomotor Vigilance Task Performance

Between the normally rested and sleep-restricted sessions, there was an increase in PVT reaction time (mean 266 versus 295 ms; $t(15) = -4.86$, $p < 0.001$, one-tailed), number of PVT lapses (mean 0.7 versus 1.3; $t(15) = -1.84$, $p = 0.04$, one-tailed), PVT reaction time standard deviation (mean 48 versus 63 ms; $t(15) = -2.65$, $p = 0.01$, one-tailed), fastest reaction time (mean 198 versus 225 ms; $t(15) = -4.81$, $p < 0.001$, one-tailed), and slowest reaction time (mean 496 versus 645 ms; $t(15) = -2.14$, $p = 0.02$, one-tailed). The number of PVT lapses (responses >500 ms) and mean PVT reaction time in each session across the 16 participants are provided in Table 2.

Behavioral Microsleeps

There was a considerable intersubject variability in the rate of microsleeps across participants within both 20-min tracking task sessions. The total number and mean duration of microsleeps and sleep episodes across the 16 participants during the 20-min sessions are provided in Table 2. There was an increase in the mean number of microsleeps in the session following sleep restriction (mean 11.4 versus 27.9; $t(15) = -2.10$, $p = 0.03$, one-tailed). There was no correlation between the number of microsleeps when normally rested and the number following sleep restriction ($r = 0.31$, $p = 0.25$).

The order of presentation of the sleep-restricted and normally rested sessions was counterbalanced across participants. There was no difference in the number of microsleeps recorded in either the first or second session (mean 19.75 versus 19.63; $t(15) = 0.01$, $p = 0.99$, two-tailed), indicating that there was no presentation order effect.

There was no correlation between number of microsleeps in the normally rested session and ESS ($r = 0.07$, $p = 0.80$), PSQI ($r = -0.10$, $p = 0.72$), MEQ ($r = 0.34$, $p = 0.19$), SSS ($r = -0.05$, $p = 0.87$), or KSS ($r = -0.17$, $p = 0.54$). There was also no correlation between number of microsleeps in the sleep-restricted session and ESS ($r = 0.10$, $p = 0.71$), PSQI ($r = -0.04$, $p = 0.89$), MEQ ($r = 0.19$, $p = 0.49$), SSS ($r = 0.30$, $p = 0.26$), or KSS ($r = 0.11$, $p = 0.68$). Age was not correlated with number of microsleeps in the normally rested session ($r = -0.13$, $p = 0.64$), but there was a trend for number of microsleeps to increase with increasing age in the sleep-restricted session ($r = 0.48$, $p = 0.06$).

There were no correlations between number of microsleeps in the normally rested session and any of the actigraphically measured sleep measures recorded during the prior night or week. However, there were correlations between number of microsleeps following sleep restriction and mean sleep efficiency ($r = 0.73$, $p = 0.001$), mean sleep onset latency ($r = -0.57$, $p = 0.02$), and mean sleep onset time-of-day standard deviation ($r = -0.54$, $p = 0.03$) during normally rested nights—with increased microsleep rate following sleep

TABLE 2. PVT performance, and number and mean microsleeps duration during the tracking task (ordered by number of microsleeps in the sleep-restricted session).

	Normally rested session				Sleep-restricted session			
Subject	PVT mean reaction time (ms)	Number of PVT lapses	Number of microsleeps	Mean microsleep duration (s)	PVT mean reaction time (ms)	Number of PVT lapses	Number of microsleeps	Mean microsleep duration (s)
312	240	0	7	2.81	288	2	100	3.40
302	279	2	0	–	323	1	76	2.39
311	246	1	85	4.22	256	0	60	3.24
301	291	1	10	1.54	298	1	47	4.38
315	247	0	2	1.90	297	2	39	1.89
310	262	1	28	2.96	301	1	27	4.44
322	331	1	47	3.10	320	3	25	4.66
318	251	1	0	–	270	2	21	2.42
316	251	1	1	1.76	280	2	13	1.53
306	314	2	0	–	305	0	11	1.87
324	242	0	0	–	266	0	10	2.28
308	268	0	0	–	324	1	7	1.35
309	241	0	0	–	298	2	6	2.18
323	260	0	0	–	324	3	3	1.93
319	276	1	3	3.83	283	1	2	4.30
303	258	0	0	–	281	0	0	–
Mean	266	0.7	11.4	2.77	295	1.3	27.9	2.82
SD	26	0.7	23.5	0.98	21	1.0	29.3	1.15
Median	259	1.0	0.5	2.89	298	1.0	17.0	2.39

PVT = psychomotor vigilance task.

restriction associated with higher sleep efficiency, shorter sleep onset latency, and less variation in sleep onset time-of-day. There was also a correlation between number of microsleeps following sleep restriction and sleep efficiency during the sleep-restricted night ($r = 0.53$, $p = 0.03$) and a trend for a relationship with sleep onset latency during the sleep-restricted night ($r = -0.46$, $p = 0.07$).

There were correlations between sleep onset latency and sleep efficiency during both normally rested nights ($r = -0.63$, $p = 0.009$) and the sleep-restricted night ($r = -0.58$, $p = 0.019$), with shorter latencies associated with higher efficiency. There was also a correlation between mean sleep onset latency and mean sleep onset time-of-day standard deviation during normally rested nights ($r = 0.51$, $p = 0.04$), with less variation in sleep onset time-of-day associated with shorter sleep onset latencies.

There was no evidence of a relationship between the number of microsleeps following sleep restriction and actual sleep time during normally rested nights ($r = 0.13$, $p = 0.64$) or the sleep-restricted night ($r = 0.38$, $p = 0.15$).

There was no correlation between the number of microsleeps and number of PVT lapses in either the normally rested ($r = 0.19$, $p = 0.47$) or sleep-restricted ($r = 0.03$, $p = 0.91$) sessions. There was also no correlation between the number of microsleeps and PVT mean reaction time in either the normally rested ($r = 0.13$, $p = 0.62$) or sleep-restricted ($r = -0.23$, $p = 0.39$) sessions.

The four variables related to the number of microsleeps during the sleep-restricted session—sleep efficiency during normally rested nights, sleep efficiency during the sleep-restricted night, sleep onset latency during normally rested nights, and sleep onset time-of-day standard deviation during normally rested nights—were included in a linear regression analysis with microsleep rate as the dependent variable. Following backward stepwise elimination of predictor variables, the linear regression retained only sleep efficiency during the normally rested nights in the model (adjusted $R^2 = 0.41$, $F(1, 14) = 11.66$, $p < 0.01$).

DISCUSSION

Contrary to our hypothesis, we found no evidence that high rates of microsleeps observed in some of our ostensibly non-sleep-deprived people were due to poor sleep quality, sleep disturbance, circadian type, irregular sleep patterns, low daily sleep duration, or poor sleep efficiency. Conversely, we found that a higher number of microsleeps following a single night of sleep restriction is related to higher sleep efficiency, shorter sleep latency, and less variation in sleep onset time during normally rested nights. This indicates that people who usually have less variability in the time of night that they go to sleep, fall asleep quickly, and have nonfragmented sleep are more vulnerable to the effect of limited time-in-bed and/or delayed sleep onset time on microsleep rate the following afternoon. These nighttime sleep measures did not relate to the number of microsleeps observed following normally rested nights.

It might be considered that the relationship between the number of microsleeps following sleep restriction

and shorter sleep onset latency during normally rested nights indicates that the people with a higher microsleep rates usually fall asleep quickly and sleep solidly throughout the night because they are compensating for underlying chronic partial sleep deprivation. However, when participants were asked to maintain their usual pattern of sleep duration and timing, there was no relationship between microsleep rates and actual sleep hours during the previous week or night. This does not eliminate the possibility that some participants were chronically partially sleep-deprived due to the fact that they require more sleep than other people or due to an undiagnosed sleep disorder that was not picked up by the actigraphy analysis. To meet study inclusion criteria, participants needed to report a usual time-to-bed time between 22:00 h and midnight and usual estimated time-in-bed of 7.0–8.5 h. However, although participants were asked to maintain their usual pattern of sleep timing and duration during the normally rested nights, sleep during normally rested nights was not ''unrestricted,'' as timing and duration would likely have been restricted by social and work demands. Thus, given the opportunity, some of our participants may have chosen to sleep longer than they did during the normally rested nights. Although undiagnosed sleep pathologies in participants with high rates of microsleeps cannot be ruled out without full polysomnographic investigation, it is important to note that people with higher sleep efficiency and shorter sleep latencies during normally rested nights did not show a higher microsleep rates when normally rested and nor did they report excessive daytime sleepiness (as self-rated via ESS). One explanation for the increased microsleep rates following sleep restriction, which is not evident when normally rested, is that the people who fall asleep quickly, wake infrequently during the night, and have a regular sleep onset time may be more susceptible to disruption of their usual regular pattern and this leads to greater vulnerability to microsleeps following sleep restriction.

There were intercorrelations between all the sleep measures associated with microsleep rates following sleep restriction, which indicates that the measures are part of the same pattern of behaviors and are not independently related to microsleep rates. This is supported by the multiple regression analysis, which found that other sleep measures did not add significantly to model microsleep rates following sleep restriction over-and-above the variation explained by sleep efficiency during normally rested nights. One possible explanation is that going to sleep at a very similar time each night leads to shorter sleep onset latencies and higher sleep efficiency due to the coordination of circadian rhythm, homeostatic pressure, body temperature, and melatonin levels. Perhaps participants who go to sleep approximately the same time every night do so because they experience sleepiness the following day when their regular pattern is disrupted. Conversely, perhaps participants who have more variation in their sleep onset time do so because, for whatever reason, this does not lead to increased sleepiness the following day.

People in our study who exhibited low variation in their sleep onset time would likely be rated as low in sleep habit flexibility as assessed by the Circadian Type Inventory (CTI) (Folkard et al., 1979). The CTI was developed to rate an individual's capability to adapt to shiftwork and assesses two factors that influence a person's ability to alter their sleeping rhythms: flexibility of sleeping habits and ability to overcome drowsiness. Our finding that low sleep habit flexibility is related to increased microsleep rates following sleep restriction is consistent with previous studies, which have found that low sleep habit flexibility is associated with poorer shiftwork tolerance and higher reports of fatigue in shiftworkers (Costa et al., 1989; Kaliterna et al., 1995). Notwithstanding, research has failed to find a prospective relationship between the sleep habit flexibility reported by people entering shiftwork and their subsequent reported shiftwork tolerance (Kaliterna et al., 1995). This may be due to some people actually improving their sleep habit flexibility with exposure to shiftwork and this leading to improved shiftwork tolerance.

Some previous literature has indicated that chronotype (i.e., morningness-eveningness) is an important factor mediating PVT vigilance across difference shifts in shiftworkers (Vetter et al., 2012) and that morning types are less tolerant to shiftwork (Bohle & Tilley, 1989; Harma et al., 1988). However, other research has failed to find a relationship between chronotype and shiftwork tolerance (Costa et al., 1989). Our study also failed to find a relationship between morningness-eveningness score and microsleeps following normal rest or sleep restriction. Previous literature has indicated that morning types may exhibit less flexible sleep habits (Costa et al., 1989; Harma et al., 1988). However, when we investigated this relationship post hoc, we found no relationship between morningness-eveningness and sleep habit flexibility (i.e., sleep onset time standard deviation) in our study group ($r = -0.18$, $p = 0.49$). However, as we had no definite morning or evening types in our participant group, the influence of chronotype on microsleeps or sleep habit flexibility may have been suppressed.

There was no correlation between number of microsleeps when normally rested and number following sleep restriction, indicating that the factors underlying microsleep rates when normally rested are different from those underlying rates following sleep restriction. This is consistent with previous research, which observed substantial interindividual variability in lapse propensity following sleep restriction or deprivation even after controlling for baseline lapse rates (Rupp et al., 2012; Van Dongen et al., 2004).

Contrary to our second hypothesis, no relationship was observed between PVT performance (PVT lapses or mean reaction time) and the subsequent number of

microsleeps during the tracking task in either the normally rested or sleep-restricted session. At least in terms of predicting brief intrusions of sleep during our monotonous task, unexpectedly the PVT did not prove to be a useful fitness-for-duty indicator. A number of factors may have led to this finding, including the PVT being the first task participants undertook when they arrived in the laboratory and were likely to have been most alert. However, if used as a fitness-for-duty assessment at, say, the beginning of a work shift, this would be the most likely time that the PVT would be completed and it would have been very useful if performance had given an indication of likely microsleep propensity during the subsequent work period. There were a number of differences between the test environment during the PVT versus the tracking task. The PVT was undertaken while sitting upright in front of a computer and was only 10 min in duration, whereas the tracking task was undertaken while lying supine and was 20 min in duration; this positional and duration difference may have led to the tracking task being more soporific. Conversely, the PVT was undertaken in a quiet room, whereas the tracking task was undertaken in a relatively uncomfortable, very noisy, and claustrophobic MRI environment. Again, if PVT performance was to be used as a fitness-for-duty purpose, then it is likely that the test would be undertaken in a quiet location separate to the work environment and the duration would need to be relatively brief.

A limitation of our study is that it is based on sleep measures recorded via actigraphy, which involves the use of an unobtrusive wristwatch-style activity and light monitor. Although actigraphy has been shown to have good reliability and validity for measuring sleep-wake patterns in normal populations (e.g., Ancoli-Israel et al., 2003; Sadeh, 2011), full polysomnography (PSG) allows for a much more comprehensive assessment of sleep involving the recording of brain, eye, and cardiac activities, respiration, and peripheral blood oxygenation. PSG would have allowed measurement of sleep architecture and allowed identification of any sleep disorders such as sleep apnea or periodic limb movements. Previous research has reported that the correlation between actigraphically and PSG-measured sleep efficiency can be quite variable, with some people showing good consistency between the two recording methods but others (especially in older populations) showing poor consistency (Reid & Dawson, 1999). Although the limitations of actigraphy, especially in clinical populations, should not be underestimated, the fact that we did find a pattern of intercorrelated sleep measures related to increased microsleep rates following sleep restriction leads us to believe that there was at least consistency in the actigraphic measurements in our participant group. PSG is also not without its disadvantages. It is time-consuming both to set up for the participant and to score by a sleep technologist. In addition, PSG electrodes and recording devices are obtrusive and thus can affect sleep parameters. Studies have observed substantial intrasubject variability across nights for all sleep parameters measured with PSG (Edinger et al., 1991; Newell et al., 2012). Overall, we believe that the incorporation of polysomnographic sleep measures would not alter the essence of our current findings. Notwithstanding, PSG would provide additional features of sleep associated with increased microsleep rates following sleep restriction in people with low sleep habit flexibility.

Our use of behavioral measures to define microsleeps may be considered by some researchers to be rather subjective. There have been attempts in the literature to identify objective methods to define microsleeps such as through the use of EEG measures. During the transition to sleep during eyes-closed resting, a shift from predominantly alpha-band (8–13 Hz) activity to theta-band (4–7 Hz) activity has been observed (Hori et al., 1994; Rechtschaffen & Kales, 1968). Thus, there have been attempts to use EEG spectral power changes to define microsleeps during an eyes-open active task (e.g., Boyle et al., 2008; Harrison & Horne, 1996; Paul et al., 2005; Peiris et al., 2006). Correlations have been observed between EEG theta-band activity and performance on a continuous task when drowsy, with theta activity increasing as performance decreases (Boyle et al., 2008; Huang et al., 2008; Lin et al., 2005; Makeig et al., 2000; Poudel et al., 2010, 2013). However, there is a poor direct relationship between EEG spectral power changes and the behavioral features of microsleeps (Peiris et al., 2006), and theta-burst activity can occur without noticeable performance changes or the behavioral features of microsleep such as eye closure and nodding off (Boyle et al., 2008; Torsvall & Akerstedt, 1988). Thus, although theta power increases are likely to relate to decreased arousal during an active task, they do not define the characteristic behavioral microsleep features of task nonresponsiveness, slow-eye-closures, and head nodding.

In contrast to a poor direct relationship with EEG spectral changes, we have observed a consistent time-linked relationship between behaviorally defined microsleeps and blood oxygen level–dependent (BOLD) changes in the brain as measured by fMRI. In an fMRI study in normally rested participants, we observed a phasic decrease in thalamic activity and increase in activity in frontoparietal cortical brain regions responsible for visuomotor and executive control time-linked to microsleeps (Poudel et al., 2013). It is likely that the changes in thalamic activity observed in the fMRI analysis were sufficiently deep in the brain for the activity to not be observable in the averaged surface EEG data. We, like others, seek to find a consistent EEG-based method for detecting microsleeps and are currently investigating the use of beamformers as spatial filters to attenuate signals from other brain regions in order to extract neuroelectric activity information from

specified regions of interest such as the thalamus (Mohamadi et al., 2012).

Overall, this study indicates that people who go to sleep at a regular time, fall asleep easily, and sleep efficiently are most likely to be adversely affected by sleep restriction, as evidenced by frequent brief intrusions of sleep and task nonresponsiveness while undertaking a monotonous task in the afternoon. People with regular sleep patterns and efficient sleep may be the people who need to be most aware of involuntarily falling asleep following disruption to their usual pattern of sleep onset and duration and may be the people least able to manage work situations that vary their sleep onset time such as shiftwork. Additionally, surprisingly, it was observed that a 10-min PVT was not useful as a predictor of subsequent microsleep rates or ''fitness-for-duty'' indicator, at least under our study conditions.

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REFERENCES

Åkerstedt T, Gillberg M. (1990). Subjective and objective sleepiness in the active individual. Int J Neurosci, 52, 29–37.

Ancoli-Israel S, Cole R, Alessi C, et al. (2003). The role of actigraphy in the study of sleep and circadian rhythms. Sleep, 26, 342–92.

Berka C, Levendowski DJ, Westbrook P, et al. (2005). EEG quantification of alertness: methods for early identification of individuals most susceptible to sleep deprivation. Paper presented at the SPIE Defense and Security Symposium, Orlando, Florida, 5797, 78–89.

Bohle P, Tilley AJ. (1989). The impact of night work on psychological well-being. Ergonomics, 32, 1089–99.

Bourgeois-Bougrine S, Carbon P, Gounelle C, et al. (2003). Perceived fatigue for short- and long-haul flights: a survey of 739 airline pilots. Aviat Space Environ Med, 74, 1072–7.

Boyle LN, Tippin J, Paul A, Rizzo M. (2008). Driver performance in the moments surrounding a microsleep. Transport Res F Traff Psychol Behav, 11, 126–36.

Buckelmuller J, Landolt HP, Stassen HH, Achermann P. (2006). Trait-like individual differences in the human sleep electroencephalogram. Neuroscience, 138, 351–6.

Buysse DJ, Reynolds 3rd CF, Monk TH, et al. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res, 28, 193–213.

Chee MW, Tan JC. (2010). Lapsing when sleep deprived: neural activation characteristics of resistant and vulnerable individuals. NeuroImage, 51, 835–43.

Chou Y-H, Chuang C-C, Zao JK, et al. (2011). An fMRI study of abrupt-awake episodes during behavioral microsleeps. Proc Int Conf IEEE Eng Med Biol Soc, 33, 5060–3.

Chuah YM, Venkatraman V, Dinges DF, Chee MW. (2006). The neural basis of interindividual variability in inhibitory efficiency after sleep deprivation. J Neurosci, 26, 7156–62.

Costa G, Lievore F, Casaletti G, et al. (1989). Circadian characteristics influencing interindividual differences in tolerance and adjustment to shiftwork. Ergonomics, 32, 373–85.

Davidson PR, Jones RD, Peiris MT. (2007). EEG-based lapse detection with high temporal resolution. IEEE Trans Biomed Eng, 54, 832–9.

Dinges DF, Powell JP. (1985). Microcomputer analysis of performance on a portable, simple visual RT task during sustained operation. Behav Res Methods Instrum Comput, 17, 652–5.

Dinges DF, Pack F, Williams K, et al. (1997). Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. Sleep, 20, 267–77.

Edinger JD, Marsh GR, McCall WV, et al. (1991). Sleep variability across consecutive nights of home monitoring in older mixed DIMS patients. Sleep, 14, 13–7.

Folkard S, Monk TH, Lobban MC. (1979). Towards a predictive test of adjustment to shift work. Ergonomics, 22, 79–91.

Gander PH, Signal TL, Van Dongen HPA, et al. (2010). Stable interindividual difference in slow-wave sleep during nocturnal sleep and naps. Sleep Biol Rhythms, 8, 239–44.

Goel N, Banks S, Mignot E, Dinges DF. (2010). DQB1*0602 predicts interindividual differences in physiologic sleep, sleepiness, and fatigue. Neurology, 75, 1509–19.

Harma M, Ilmarinen J, Knauth P. (1988). Physical fitness and other individual factors relating to the shiftwork tolerance of women. Chronobiol Int, 5, 417–24.

Harrison Y, Horne JA. (1996). Occurrence of 'microsleeps' during daytime sleep onset in normal subjects. Electroencephalogr Clin Neurophysiol, 98, 411–6.

Hoddes E, Dement W, Zarcone V. (1972). The development and use of the Stanford sleepiness scale. Psychophysiology, 9, 150p.

Hori T, Hayashi M, Morikawa T. (1994). Topographical EEG changes and the hypnagogic experience. In: Ogilvie RD, Harsh JR, eds. Sleep onset: normal and abnormal processes. Washington, DC: American Psychological Association, 237–53.

Horne J, Reyner L. (1999). Vehicle accidents related to sleep: a review. Occup Environ Med, 56, 289–94.

Horne JA, Ostberg O. (1976). A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. Int J Chronobiol, 4, 97–110.

Huang RS, Jung TP, Delorme A, Makeig S. (2008). Tonic and phasic electroencephalographic dynamics during continuous compensatory tracking. NeuroImage, 39, 1896–909.

Innes CRH, Poudel GR, Signal TL, Jones RD. (2010). Behavioural microsleeps in normally-rested people. Proc Int Conf IEEE Eng Med Biol Soc, 32, 4448–51.

Johns MW. (1991). A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep, 14, 540–5.

Kaliterna I, Vidaček S, Prizmic Z, et al. (1995). Is tolerance to shiftwork predictable from individual difference measures? Work Stress, 9, 140–7.

Landolt HP. (2008). Genotype-dependent differences in sleep, vigilance, and response to stimulants. Curr Pharm Des, 14, 3396–407.

Leproult R, Colecchia EF, Berardi AM, et al. (2003). Individual differences in subjective and objective alertness during sleep deprivation are stable and unrelated. Am J Physiol Regul Integr Comp Physiol, 284, R280–90.

Lin C-T, Wu R-C, Liang S-F, et al. (2005). EEG-based drowsiness estimation for safety driving using independent component analysis. IEEE Trans Circuits Sys, 52, 2726–38.

Makeig S, Jung TP, Sejnowski TJ. (2000). Awareness during drowsiness: dynamics and electrophysiological correlates. Can J Exp Psychol, 54, 266–73.

Mohamadi YJ, Poudel GR, Innes CRH, Jones RD. (2012). Performance of beamformers on EEG source reconstruction. Proc Int Conf IEEE Eng Med Biol Soc, 34, 2517–21.

Newell J, Mairesse O, Verbanck P, Neu D. (2012). Is a one-night stay in the lab really enough to conclude? First-night effect and night-to-night variability in polysomnographic recordings among different clinical population samples. Psychiatry Res, 200, 795–801.

Paul A, Boyle LN, Tippin J, Rizzo M. (2005). Variability of driving performance during microsleeps. Paper presented at the International Driving Symposium on Human Factors in Driver Assessment, Training and Vehicle Design; 2005 Jun 27–30; Maine, USA, 3, 18–24.

Peiris MT, Davidson PR, Bones PJ, Jones RD. (2011). Detection of lapses in responsiveness from the EEG. J Neural Eng, 8, 1–15.

Peiris MTR, Jones RD, Davidson PR, et al. (2006). Frequent lapses of responsiveness during an extended visuomotor tracking task in non-sleep deprived subjects. J Sleep Res, 15, 291–300.

Portaluppi F, Smolensky MH, Touitou Y. (2010). Ethics and methods for biological rhythm research on animals and human beings. Chronobiol Int, 27, 1911–29.

Poudel GR, Jones RD, Innes CRH, Bones PJ. (2008). Characteristics and EEG spectral dynamics of behavioural microsleeps in a mock-MRI scanner. NeuroImage, 41, S59.

Poudel GR, Innes CRH, Bones PJ, Jones RD. (2010). The relationship between behavioural microsleeps, visuomotor performance and EEG theta. Proc Int Conf IEEE Eng Med Biol Soc, 32, 4452–5.

Poudel GR, Innes CRH, Jones RD. (2012). Cerebral perfusion differences between drowsy and non-drowsy individuals following acute sleep restriction. Sleep, 35, 1085–96.

Poudel GR, Innes CRH, Bones PJ, et al. (2013). Losing the struggle to stay awake: divergent thalamic and cortical activity during microsleeps. Hum Brain Mapp, In press.

Rechtschaffen A, Kales A. (1968). A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects. Los Angeles, CA: Brain Research Institute, University of California, 94p.

Reid K, Dawson D. (1999). Correlation between wrist activity monitor and electrophysiological measures of sleep in a simulated shiftwork environment for younger and older subjects. Sleep, 22, 378–85.

Rupp TL, Wesensten NJ, Balkin TJ. (2012). Trait-like vulnerability to total and partial sleep loss. Sleep, 35, 1163–72.

Sadeh A. (2011). The role and validity of actigraphy in sleep medicine: an update. Sleep Med Rev, 15, 259–67.

Sommer D, Golz M, Schnupp T, et al. (2009). A measure of strong driver fatigue. Proc Int Driving Symp Hum Factors Driver Assess Training, 5, 9–15.

Torsvall L, Akerstedt T. (1988). Extreme sleepiness: quantification of EOG and spectral EEG parameters. Int J Neurosci, 38, 435–41.

Van Dongen HP. (2006). Shift work and inter-individual differences in sleep and sleepiness. Chronobiol Int, 23, 1139–47.

Van Dongen HP, Baynard MD, Maislin G, Dinges DF. (2004). Systematic interindividual differences in neurobehavioral impairment from sleep loss: evidence of trait-like differential vulnerability. Sleep, 27, 423–33.

Van Dongen HP, Vitellaro KM, Dinges DF. (2005). Individual differences in adult human sleep and wakefulness: leitmotif for a research agenda. Sleep, 28, 479–96.

Vetter C, Juda M, Roenneberg T. (2012). The influence of internal time, time awake, and sleep duration on cognitive performance in shiftworkers. Chronobiol Int, 29, 1127–38.

Whitney CW, Enright PL, Newman AB, et al. (1998). Correlates of daytime sleepiness in 4578 elderly persons: the Cardiovascular Health Study. Sleep, 21, 27–36.