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The dynamics of neurobehavioural recovery following sleep loss

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SUMMARY Rate of recovery of daytime performance and sleepiness following moderate and severe sleep deprivation (SD) was examined when recovery opportunity was either augmented or restricted. Thirty healthy non-smokers, aged 18–33 years, participated in one of three conditions: moderate SD with augmented (9-h) recovery opportunities, moderate SD with restricted (6-h) recovery opportunities, or severe SD with augmented recovery opportunities. Each participant attended the laboratory for 8–9 consecutive nights: an adaptation and baseline night (23:00–08:00 hours), one or two night(s) of wakefulness, and five consecutive recovery sleep opportunities (23:00–08:00 hours or 02:00–08:00 hours). On each experimental day, psychomotor vigilance performance (PVT) and subjective sleepiness (SSS) were assessed at two-hourly intervals, and MSLTs were performed at 1000h. PSG data was collected for each sleep period. For all groups, PVT performance significantly deteriorated during the period of wakefulness, and sleepiness significantly increased. Significant differences were observed between the groups during the recovery phase. Following moderate SD, response speed, lapses and SSS returned to baseline after one 9-h sleep opportunity, while sleep latencies required two 9-h opportunities. When the recovery opportunity was restricted to six hours, neither PVT performance nor sleepiness recovered, but stabilised at below-baseline levels. Following severe SD, sleepiness recovered after one (SSS) or two (physiological) 9-h sleep opportunities, however PVT performance remained significantly below baseline for the entire recovery period. These results suggest that the mechanisms underlying the recovery process may be more complicated than previously thought, and that we may have underestimated the impact of sleep loss and/or the restorative value of subsequent sleep.

KEYWORDS neurobehavioural recovery, performance, recovery opportunity, sleep deprivation, sleep restriction, sleepiness

INTRODUCTION

A significant body of research has investigated how fatigue accumulates during acute and chronic partial sleep loss. Studies typically indicate that once prior wake extends beyond approximately 16 h and/or sleep obtained in the prior 24-h period is reduced below 5–6 h, significant sleepiness and neurobehavioural impairment occurs (Belenky *et al*., 2003; Dinges *et al*., 1997; Carskadon and Dement, 1981; Rosenthal *et al*., 1993; Van Dongen *et al*., 2003). Specifically, individuals fall asleep more easily, take longer to respond, make more errors, have reduced situational awareness, communicate less effectively and have increased difficulty in making decisions and prioritizing relevant information (Dinges and Barone Kribbs, 1991; Harrison and Horne, 2000; Jewett *et al*., 1999; Lamond and Dawson, 1999).

While there has been a significant effort directed toward understanding the manner in which fatigue accumulates, few studies have systematically described the recovery of neurobehavioural function in response to sleep loss. Historically, studies investigating recovery from sleep loss have focussed on

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changes in sleep architecture. Using the electroencephalogram (EEG) as the primary method of measurement, 'recovery' has usually been defined as the point at which sleep architecture returns to normal. Often, this occurs after one night of unrestricted sleep. If, however, the duration of recovery sleep is restricted, continuing homeostatic effects may be observed for a second or third night (Bonnet, 1994).

It has usually been assumed that neurobehavioural function recovers at the same rate as sleep itself, as the few early studies of sleep loss that also included a short recovery phase reported recovery of alertness and performance after only 1–2 days. However, the neurobehavioural measures used in these studies were often confounded by learning effects and limited by a possible lack of sensitivity to fatigue, or the length of the recovery opportunity was not controlled (Bonnet and Rosa, 1987; Fenz and Graig, 1972; Lubin *et al.*, 1976; Webb and Agnew, 1973). Moreover, recent research challenges this assumption, and suggests that the amount of sleep required for recovery of neurobehavioural function may have previously been underestimated. For example, recent studies of chronic sleep restriction, that also included a short recovery phase, indicated that neither two 10-h nor three 8-h recovery sleep opportunities were sufficient to allow performance to recover to baseline (B) levels following 7–14 consecutive days of sleep restriction (Belenky *et al.*, 2003; Dinges *et al.*, 1997; Van Dongen *et al.*, 2003). These findings demonstrate that more than three nights may be necessary for recovery of neurobehavioural function. Yet, most studies that included recovery as a secondary outcome only collected 1–3 nights of recovery data.

Despite a lack of systematic studies, the dynamics of recovery are of equal theoretical importance, and potentially of greater practical importance than the accumulation of fatigue and neurobehavioural performance deficits. For example, a better understanding of how much sleep is needed to reverse the effects of fatigue has significant practical benefits for any organization required to manage fatigue, and more broadly amongst the regulators who formulate hours-of-work policy. The purpose of the present study was to empirically determine the rate of recovery following moderate and severe sleep loss of sleepiness and neurobehavioural performance over five consecutive days of recovery sleep, when recovery opportunity was either augmented or restricted.

MATERIALS AND METHODS

Participants

Thirty healthy individuals (11 females, 19 males), aged 18–33 years (mean ± standard deviation = 23.3 ± 4.3 years), participated in the current study. Participants were non-smokers who did not regularly consume large doses of caffeine (< 200 mg day^{-1}). Those recruited had no current health problems and were not taking any medication other than an oral contraceptive. All were self-reported good sleepers who typically went to bed between 22:00 hours and midnight (average bedtime = 23:30 hours), and woke between 07:00 and 09:00 hours (average wake time = 07:45 hours). Participants did not habitually nap, were not shiftworkers and had not undertaken transmeridian travel in the past 3 months. For the week prior to the study, participants were instructed to keep to a regular sleep/wake schedule (confirmed by sleep diaries). Before the study commenced, the protocol was approved by the University of South Australia Human Research Ethics Committee, using guidelines established by the National Health and Medical Research Council of Australia. Prior to participation, all volunteers provided written informed consent.

Procedure

Participants spent eight or nine consecutive days in-residence in the laboratory (Fig. 1), in groups of three or four. For all participants, the initial days involved an adaptation and baseline night, and training. On the adaptation day, participants arrived at the laboratory at 18:00 hours and were assigned to their individual bedroom. After they were provided with verbal and written descriptions of study procedures and rules, participants completed a short training session to familiarize themselves with the various performance tasks. On both the adaptation and baseline night, participants were required to be in bed from 23:00 to

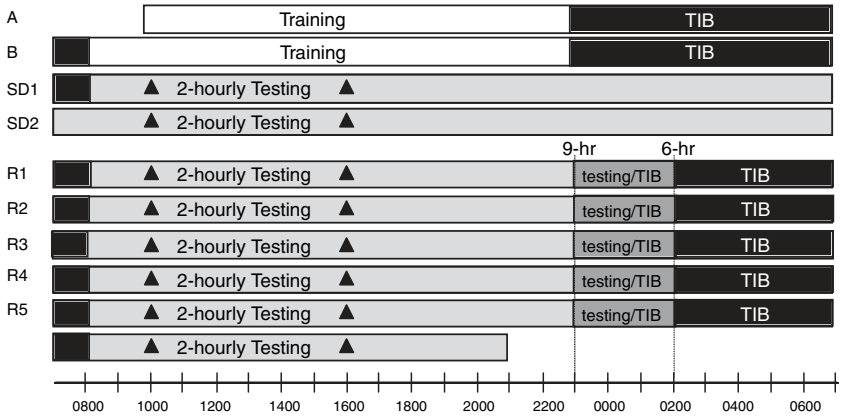

Figure 1. The experimental design indicating time in bed across days: adaptation (A), baseline (B), sleep deprivation (SD1/SD2) and recovery (R1–R5).

08:00 hours [9 h time in bed (TIB)]. Prior to retiring each night, a standard montage of electrodes was applied to each participants face and scalp. Following the adaptation night, electrodes were removed hence participants could shower and breakfast, later they completed further training to minimize learning effects.

Following the baseline night, baseline measures were taken. Participants were then deprived of sleep (SD) for either one or two nights, to experimentally induce moderate or severe levels of fatigue. Immediately following the period of extended wakefulness, participants were permitted five recovery sleep opportunities (R1–R5) that involved either 9-h TIB (23:00–08:00 hours; augmented sleep opportunity) or 6-h TIB (02:00–08:00 hours; restricted sleep opportunity). The severity of SD and the length of the recovery sleep opportunities depended on which of the three conditions participants were in: (i) moderate SD with 9-h recovery opportunity (moderate 9-h TIB), (ii) moderate SD with 6-h recovery opportunity (moderate 6-h TIB), or (iii) severe SD with 9-h recovery opportunity (severe 9-h TIB). Prior to each of the recovery sleep periods, a conventional montage of electrodes was attached to the face and scalp of each subject, and polysomnographic data was collected.

In all conditions, the room temperature was set at 25 °C. All the windows and curtains remained closed, and fluorescent lights remained on in each room (except during sleep periods). Throughout all phases of the study, lights-on time was 08:00 hours. Participants were not permitted any other TIB or opportunity for sleep except as required by the periodic sleep latency tests (SLT). Participants were permitted to consult timepieces but were not permitted to set alarms. Exercise and the use of baths or showers to enhance alertness were not permitted, and participants were required to abstain from caffeine and other stimulants for the entire study period. Regular balanced meals [breakfast, lunch, dinner] and snacks [morning, afternoon, evening] were provided. Between test sessions, participants were free to engage in quiet activities, such as reading, watching TV or conversing with other participants.

Test instruments and measures

Psychomotor vigilance task

A 10-min psychomotor vigilance task (PVT), which measures simple reaction time to a visual stimulus, was used to evaluate sustained attention (Dorrian *et al.* 2004). During this test, participants were required to attend to the LED timer display and press the response button with the thumb of their dominant hand as quickly as possible after the appearance of the visual stimuli. As per standard methodology, the inter-stimulus interval varied from 2000 to 10 000 ms. Dependent measures included mean speed (reciprocal of average response latency), number of lapses (lapse = response latency exceeding 500 ms) and mean speed for the fastest 10% of all responses.

Polysomnography

Polysomnographic (PSG) measures [EEG (C3-A2 and C4-A1); electro-oculogram (outer canthi of each eye); electromyogram] were recorded during each sleep period using the Compumedics 10–20 system (Melbourne, Australia) and Medilog MPA-2 sleep analysis system (Oxford Medical Ltd, Abingdon, UK). EEG signals were sampled within a 0.33–70 Hz bandwidth, digitized at 250 Hz and filtered with a 50 Hz notch filter. The PSG recordings for each sleep period and for SLTs were scored in 30-s epochs in accordance with standard criteria (Rechtschaffen and Kales, 1968).

Sleep latency test

A 20-min SLT was conducted at 10:00 and 16:00 hours to objectively assess sleepiness. For each SLT, subjects were allowed to be in bed in a quiet, temperature controlled, darkened room and instructed to close their eyes and not resist the urge to fall asleep. Two researchers monitored PSG signals in a control room, using Compumedics 10–20 system and Medilog MPA-2 sleep analysis system. The SLT was terminated after three consecutive epochs of stage 2 sleep or after any one epoch of stage 3, 4 or REM sleep (or after 20 min without sleep onset), and the subject was woken via an intercom system. When woken, subjects were asked to sit up and turn their lamp on, and wait until the test session was complete (i.e. when all subjects had fallen asleep or after 20 min).

Subjective sleepiness

At the beginning of each test session, subjective sleepiness was assessed using the Stanford Sleepiness Scale [SSS, (Hoddes *et al.*, 1973)], a single item scale ranging from 1 ('feeling active and vital, alert, wide awake') to 7 ('almost in reverie, sleep onset soon, losing struggle to remain awake').

Testing schedule

Across the period of imposed wakefulness and on each of the recovery days, the PVT and SSS were administered at 2-h intervals, commencing at 09:00 hours and ending at 21:00 hours (9-h TIB groups) or 23:00 hours (6-h TIB group). The test battery took approximately 30 min to complete and included, in addition to the tests described above, several short computer-based tasks from the Walter Reed Performance Assessment Battery (Thorne *et al.*, 1985) (e.g. Running memory, Stroop, Serial Add/Subtract, Matching to Sample, Six-letter Search). The results of these tasks are not reported here as considerable practice effects confounded the data. On each experimental day, the SLT was administered at 10:00 hours and 16:00 hours for each group. Following the 10:00 hours SLT, electrodes were removed, and participants could shower. They were then applied again at 14:30 hours for the afternoon SLT, and at 22:00 hours (9-h TIB) or at 01:00 hours (6-h TIB), prior to the recovery sleep periods.

Showers were not permitted on the second day of sleep deprivation.

Data analysis

Statistical analysis was performed with SPSS (version 11.0.2, SPSS Inc., Chicago, IL, USA). For the PVT and SSS data, analyses of time-of-day effects as a function of condition indicated that there were no significant time-of-day × condition interactions, or main effects of condition for any of the recovery days, or for the baseline day. Therefore, as the circadian profiles were parallel in each condition, and the focus of the study was on overall differences in recovery amongst the three conditions rather than on recovery across the day, daily averages were calculated for each measure for simplicity. All data (PVT, SSS and SLT) were analysed using separate repeated measures (ANOVA) with two within subject factors: group (moderate 9-h, moderate 6-h, or severe 9-h) and experimental day (B, SD, R1, R2, R3, R4 and R5). Simple main effects analyses were then applied to all significant Group Day-interactions and where relevant, planned comparisons were performed to specify differences amongst mean values (Kirk, 1995). Huynh–Feldt corrections were applied to all repeated measures effects. A significance level of $P \leq 0.05$ was used for all statistical analyses.

RESULTS

The demographic data for each group are reported in Table 1. Neither the distribution of females and males ($\chi^2 = 0.29$, $P = 0.866$) nor the mean age ($F_{2,27} = 1.5$, $P = 0.249$) differed amongst the groups.

Night-time sleep

Mean total sleep time (TST) for each group across B and R1–R5 is displayed in Fig. 2. TST for the night-time baseline sleep did not differ amongst the three groups ($F_{2,27} = 0.9$, $P = 0.431$). On the baseline night, average TST was 7.96 ± 0.34 h across all groups. TST increased significantly in the moderate 9-h TIB group (R1–R2) and the severe 9-h TIB group (R1) initially, but returned to baseline for the remainder of the recovery period, and decreased significantly in the moderate 6-h TIB group (R1–R5) compared with baseline (Day, $F_{5,135} = 26.6$, $P < 0.0001$; Group, $F_{2,27} = 144.3$, $P < 0.0001$; Group × Day, $F_{10,135} = 21.7$, $P < 0.0001$). Average TST over the five recovery sleep periods was 8.0 ± 0.4 h for the moderate 9-h TIB group, 5.7 ± 0.1 h for the moderate 6-h TIB group, and 8.0 ± 0.3 h for the severe 9-h TIB group.

Table 1 Demographic data for each group

Group	*No. females : males*	*Mean (± standard deviation) age*
Moderate 9-h TIB	4 : 6	25.1 ± 4.5 years
Moderate 6-h TIB	4 : 6	22.0 ± 3.4 years
Severe 9-h TIB	3 : 7	22.7 ± 4.7 years

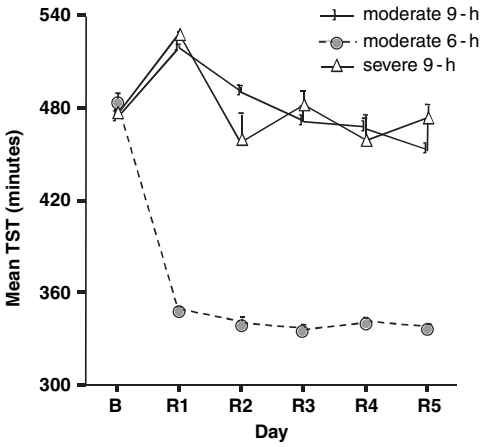

Figure 2. Mean (± SEM) total sleep time during the baseline (B) and recovery (R1–R5) nights for each group.

Psychomotor vigilance task

Mean response speed

Figure 3 displays mean PVT response speed [(1/mean response time) × 1000] as a function of group and day (collapsed across time of day). Response speed significantly varied across experimental days for each group ($F_{6,162} = 38.9$, $P < 0.0001$). A significant Group × Day interaction ($F_{12,162} = 4.4$, $P < 0.0001$) was also found (Table 2 displays simple effects across days for each group). For each group, response speed significantly deteriorated during the period of wakefulness (SD). For the moderate 9-h TIB group, response speed was significantly faster on the

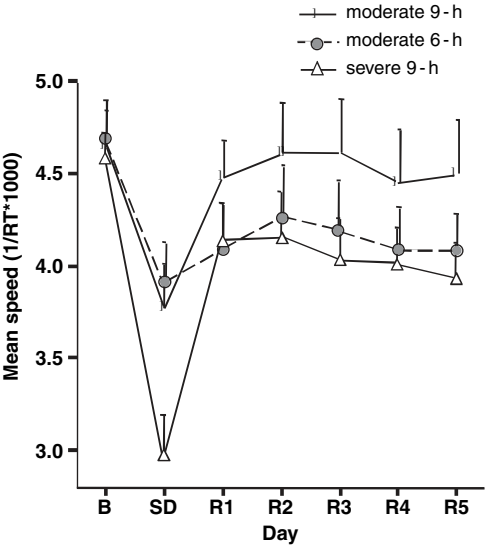

Figure 3. Mean (± SEM) psychomotor vigilance task response speed across days for each group.

Table 2 Analysis of variance results of simple effects of day for each group for each psychomotor vigilance task metric, sleep latency and subjective sleepiness ratings

Group	$F_{6,54}$	*P*-value
Mean speed		
Moderate 9-h TIB	12.5	0.000
Moderate 6-h TIB	15.7	0.000
Severe 9-h TIB	17.9	0.000
Number of lapses		
Moderate 9-h TIB	6.2	0.005
Moderate 6-h TIB	3.4	0.071
Severe 9-h TIB	16.9	0.000
Mean fastest 10% responses		
Moderate 9-h TIB	3.4	0.049
Moderate 6-h TIB	5.0	0.015
Severe 9-h TIB	11.0	0.000
Daytime sleep latency		
Moderate 9-h TIB	39.3	0.000
Moderate 6-h TIB	11.4*	0.000
Severe 9-h TIB	41.1	0.000
Stanford Sleepiness Scale		
Moderate 9-h TIB	52.7	0.000
Moderate 6-h TIB	31.5	0.000
Severe 9-h TIB	32.5	0.000

*sleep data lost for one subject d.f. = 6,48.

first recovery day (R1) than SD, and did not significantly vary from baseline levels across the recovery period (R1–R5). For the moderate 6-h TIB group, response speed failed to recover and remained significantly slower than B for the entire recovery period (R1–R5). For the severe 9-h TIB group, response speed during SD decreased in a dose–dependent manner compared with the moderate SD groups. While response speed increased from SD to R1, it failed to recover to baseline levels and remained significantly impaired during recovery.

Number of lapses

Figure 4 displays mean number of lapses across the days as a function of group. Lapses significantly varied across the experimental days ($F_{6,162} = 22.9$, $P < 0.0001$). A significant Group × Day interaction ($F_{12,162} = 5.6$, $P < 0.0001$) was also found (Table 2 displays simple effects across days for each group). Mean number of lapses significantly increased during the period of wakefulness for the moderate 9-h TIB and severe 9-h TIB groups. Lapses in the moderate 9-h TIB group decreased from SD to R1, and did not significantly differ from B for any of the recovery days (R1–R5). For the severe 9-h TIB group, the increase in lapses from B to SD was approximately three times the increase that was observed from B to SD in the moderate SD groups. During the recovery phase, significantly more lapses occurred on R3, R4 and R5 compared with B, however, the difference did not reach statistical significance for R1 and R2 ($P = 0.059$). Although the number of lapses during the period of wakefulness increased in the moderate 6-h TIB group (planned comparison indicated a significant difference between B and SD; $F_{1,9} = 8.2$, $P = 0.019$), once the Huynh–Feldt correction was applied, analysis indicated that number of lapses did not significantly vary across the experimental period. Thus, while more lapses occurred on R1–R5 compared with baseline, the difference was not significant.

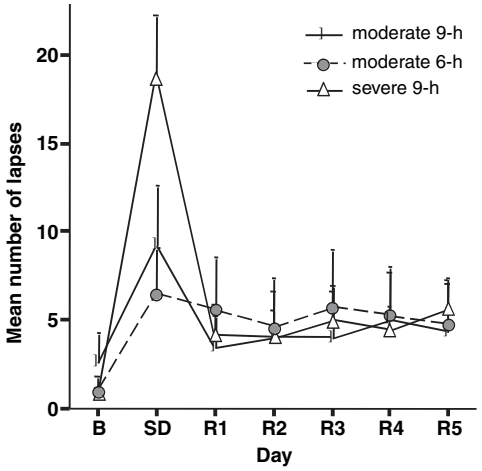

Figure 4. Mean (± SEM) number of lapses on the psychomotor vigilance task across days for each group.

Mean fastest 10% of responses

Figure 5 displays the mean speed for the fastest 10% of responses as a function of group and day. Fastest 10% of responses significantly varied across experimental days for each group ($F_{6,162} = 14.2$, $P < 0.0001$). A significant Group × Day interaction ($F_{12,162} = 2.4$, $P = 0.0280$) was also found (Table 2 displays simple effects across days for each group). The pattern for the fastest 10% of responses for the moderate 9-h TIB group and moderate 6-h TIB group was similar to that found for mean speed. Response speed for the fastest 10% of responses significantly deteriorated during the period of wakefulness (SD) for both groups. For the moderate

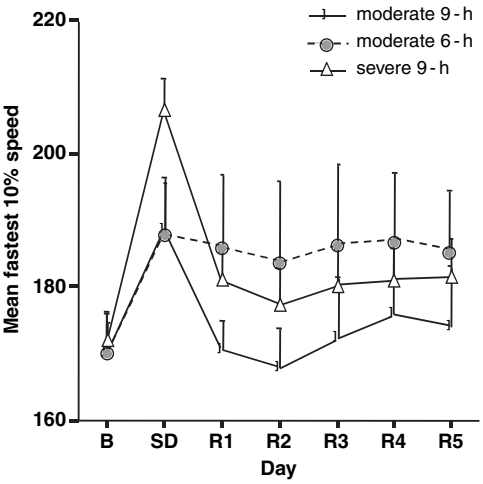

Figure 5. Mean (± SEM) psychomotor vigilance task speed for the fastest 10% of responses across days for each group.

9-h TIB group, speed was significantly faster on the first recovery day (R1) than SD, and did not significantly vary from baseline levels across the recovery period (R1–R5). For the moderate 6-h TIB group, speed failed to recover and remained significantly slower than B for most of the recovery period (R1, R3–R5). For the severe 9-h TIB group, the pattern differed somewhat to that of response speed. The fastest 10% of responses decreased in a dose–dependent manner during SD, compared with the moderate SD groups, and increased significantly from SD to R1. Speed across R1–R5 did not significantly differ from B.

Objective and subjective sleepiness

Daytime sleep latency

On the baseline day, average sleep latency was 17.4 ± 4.2 min across all groups. Mean sleep latency did not differ amongst groups on B ($F_{2,26} = 2.6$ $P > 0.05$). As shown in Fig. 6, latency significantly varied across experimental days for each group ($F_{6,156} = 72.5$, $P < 0.0001$). A significant Group × Day interaction ($F_{12,156} = 6.4$, $P < 0.0001$) was also found (Table 2 displays simple effects across days for each group). Sleep latency significantly decreased during the period of wakefulness (average sleep latency for SD was 2.4 ± 1.7 min across all groups). For the moderate 9-h TIB group, sleep latency on R1 was significantly shorter than baseline. Latency on R2–R5 did not significantly differ from baseline. For the moderate 6-h TIB group, latency failed to recover and remained significantly shorter than B for the entire recovery period (R1–R5). Sleep latency for the severe 9-h TIB group was significantly shorter than B for R1 and R2. On the third recovery day, latency recovered, and did not significantly differ from B for R3–R5.

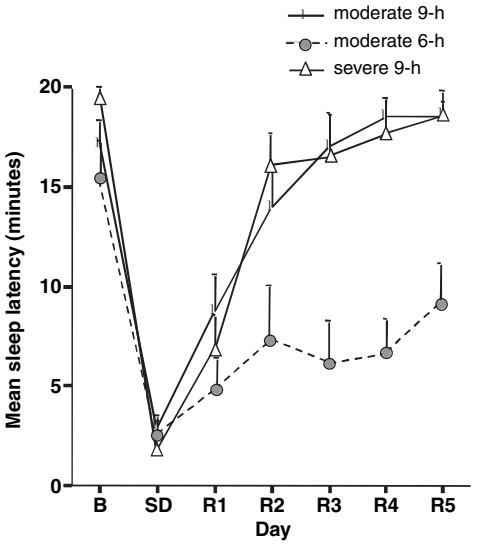

Figure 6. Mean (± SEM) sleep onset latency to the first 30 s of three consecutive epochs of stage 2 sleep across days for each group.

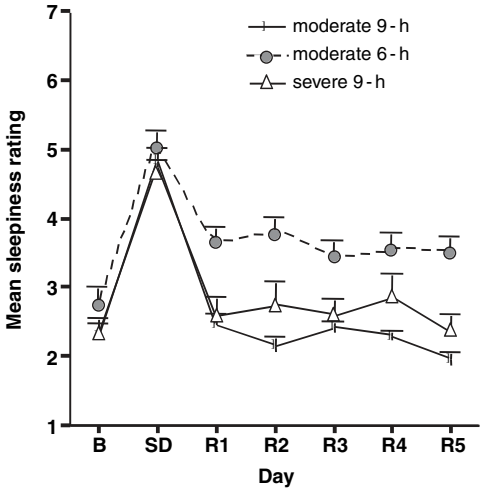

Figure 7. Mean (± SEM) Stanford Sleepiness Scale scores across days for each group.

Stanford sleepiness scale

Ratings of sleepiness significantly varied across experimental days for each group ($F_{6,162} = 108.6$, $P < 0.0001$), as shown in Fig. 7. A significant Group × Day interaction ($F_{12,162} = 4.0$, $P < 0.0001$) was also found (Table 2 displays simple effects across days for each group). For each group, ratings of sleepiness significantly increased during the period of wakefulness (SD). For both the moderate 9-h TIB and severe 9-h TIB group, ratings of sleepiness significantly decreased following R1, and did not significantly differ from B across the recovery period (R1–R5). For the moderate 6-h TIB group, ratings of sleepiness remained significantly higher than B for all of the recovery period (R1–R5).

DISCUSSION

As a result of a paucity of studies, the link between recovery and performance has not been clearly elucidated. Research has typically been limited to studies primarily designed to investigate the accumulation of performance deficits and sleepiness following sleep loss, with a short recovery phase reported as a secondary outcome (Belenky *et al.*, 2003; Dinges *et al.*, 1997; Caldwell and Caldwell, 1997; Fenz and Graig, 1972; Lubin *et al.*, 1976; Van Dongen *et al.*, 2003; Webb and Agnew, 1973; Williams and Lubin, 1967; Williams and Williams, 1966; Williams *et al.*, 1962; Williams *et al.*, 1965), or to studies which did not control the length of the recovery opportunity and/or disrupted sleep continuity (Bonnet and Rosa, 1987; Lubin *et al.*, 1974; Rosa *et al.*, 1983). As a consequence, it is possible that the impact of sleep loss and/or the restorative value of subsequent sleep have been underestimated. Certainly, several of the recent studies have noted a discrepancy between their data and current theoretical models of recovery, and a recent review article suggested that our understanding of the dynamics of recovery from sleep loss is lacking (Dawson and McCulloch, 2005). The aim of the current study was to systematically investigate the rate of recovery of

neurobehavioural function and sleepiness following moderate and severe sleep loss when recovery opportunity was either augmented or restricted.

Restriction of the recovery sleep opportunity

Restricting the recovery sleep opportunity clearly impacted on recovery. Given that the week of sleep restriction was preceded by a night of acute sleep loss, it was considered possible that performance would decline continuously across the recovery week as the sleep debt continued to accumulate. Certainly, this pattern of response would be predicted by recent studies indicating that chronic restriction of sleep opportunity for two weeks is associated with a near-linear accumulation of performance impairment (Van Dongen *et al*., 2003). Indeed, it has recently been speculated that the manner in which the wakefulness is accumulated (i.e. chronic restriction versus acute sleep loss) does not affect the net performance impairment, and that the level of impairment associated with two weeks of sleep restriction is equivalent to the impairment following one night of wakefulness (Van Dongen *et al*., 2003). This would suggest that the net impairment observed at the end of the week in the restricted condition should have been equivalent to the impairment associated with three weeks of chronic sleep restriction (i.e. one night sleep loss is equal to two weeks restriction, plus an additional week during the recovery period).

However, this was clearly not the case. While neurobehavioural function failed to recover when recovery opportunity was restricted, both psychomotor vigilance performance and sleepiness did stabilize at lower than baseline levels. These findings are more in line with the suggestion that the brain undergoes adaptive changes in response to chronic sleep restriction, that serve to sustain a stable, albeit reduced, level of performance (Belenky *et al*., 2003). Moreover, the current findings suggest that this occurs even when the performance is initially reduced below baseline, because of a period of acute sleep loss.

The observation of stabilized, lowered function during restricted recovery sleep, rather than continuing decrements with accumulating partial sleep loss, also lends weight to the extensive body of research conducted by Taub and colleagues (Taub, 1978, 1980; Taub and Berger, 1973; Taub and Berger, 1974, 1976b; Taub and Berger, 1976a). Specifically, this series of studies postulated that sleep occupies an integral position in the 24-h cycle, and that optimal levels of certain behavioural functions are highly dependent upon maintenance of an established temporal rhythm of sleep and wake. Thus, altering the placement of the sleep period, whether by shifting, restricting, or extending sleep, can result in decrements in waking function, independent of sleep length. While delaying bedtime in the current study disrupted participants' established diurnal rhythm and therefore potentially contributed to the performance decrements (or lack of recovery), the regular temporal placement of sleep during the recovery week may explain, at least in part, the adaptive changes observed. Certainly, research that varies the timing of the recovery sleep is warranted, and may provide important information about waking function following restricted recovery sleep,

While the number of lapses increased following sleep loss and remained elevated, the difference was not statistically significant. In contrast, fastest 10% of responses, which are typically not impacted by occasional lapses in performance, significantly slowed and failed to recover. In line with previous studies, the finding that fastest 10% responses (i.e. responses other than lapses) as well as mean speed were affected and failed to recover suggests that the sleep-loss induced performance deficits were because of persistent, pervasive change in brain function, rather than simply lapses in performance because of brief episodes of sleep. The fact that the increase in lapses for the moderate 6-h group was non-significant was likely because of a small sample size ($n = 10$) and inter-individual differences. Certainly, lapses were significantly affected in the moderate condition, which involved the same period sleep loss.

Augmentation of the recovery sleep opportunity

The negative effects of one night of sleep loss on neurobehavioural function were reversed by one 9-h recovery sleep opportunity. This observation is in line with the findings of earlier studies of acute sleep loss that also attempted to characterize recovery (Fenz and Graig, 1972; Lubin *et al*., 1976; Rosa *et al*., 1983; Webb and Agnew, 1973). Yet, this contrasts with the rate of recovery reported in recent sleep restriction studies, which have observed that three 8-h recovery sleep opportunities were insufficient to restore PVT performance to baseline levels, even if the sleep restriction experienced during the week was only mild (i.e. 7-h TIB per night). It is likely that this discrepancy is because of the fact that acute and chronic sleep restriction causes differential performance effects that are most salient during the recovery period. Indeed, it is apparent from our findings that while the mode of sleep deprivation may not affect the net performance impairment (Van Dongen *et al*., 2003), it does affect rate of neurobehavioural recovery.

Another equally important factor that appears to impact on rate of neurobehavioural recovery is the length of the recovery sleep opportunity, or more specifically, whether it is terminated prematurely. In a recent study of one night of sleep restricted to 2-h (less detrimental than a full night awake), while multitask performance returned to near baseline level, complete recovery was not reported (Sallinen *et al*., 2004). Most notably, the recovery sleep opportunity in this study was only 8 h in duration. In contrast, our current study augmented the recovery opportunity and allowed participants 9 h in bed each night, which appears to have facilitated recovery. The suggestion that baseline and/or recovery sleep opportunities should be > 8 h is also supported by recent research indicating that the amount of sleep needed per day to prevent cumulative neurobehavioural deficits is 8.16 h (Van Dongen *et al*., 2003).

The SLT data in the current study further emphasizes the importance of allowing more than 8-h TIB for baseline and

recovery sleeps. Previous studies have noted that when baseline sleep opportunities are only 8 h in length, the mean sleep onset latencies observed on the baseline day are often near the pathological ranges (e.g. 6.3 min) (Belenky *et al.*, 2003; Harrison and Horne, 1996). In the current study, however, baseline sleep latency scores were not in clinical range. Rather, individuals had a mean sleep onset latency of 17.4 min on the baseline day, suggesting they were well-rested from the 9-h sleep opportunities on the previous two nights, and further highlighting the recuperative value of an additional hour of sleep opportunity.

These findings emphasize the importance of not restricting the first recovery sleep opportunity. It is apparent that allowing individuals just one extra hour can significantly increase the rate of recovery. This has important implications for shiftworkers, particularly those with quick turn around times between shifts, and minimal time to recover. To expedite recovery and increase the quality of their time off, shiftworkers should extend their first recovery sleep period and wake naturally, rather than setting an alarm and restricting their TIB.

Increasing the severity of the sleep loss

Increasing the severity of sleep loss resulted in a dose–dependent decrease in psychomotor vigilance performance. Specifically, response speed was twice as slow, while the number of lapses increased three-fold. Most notably, in contrast to the moderate 9-h sleep loss condition, psychomotor vigilance performance failed to recover, even following five 9-h recovery sleep opportunities. Interestingly, this is not in line with findings of previous studies which reported recovery of performance following 60 h of wakefulness after only one night of recovery sleep (Dinges *et al.*, 1997; Caldwell and Caldwell, 1997; Fenz and Graig, 1972; Webb and Agnew, 1973; Williams *et al.*, 1959). Further, it does not support the widely held belief that the amount of sleep taken to recover the performance deficit associated with acute sleep loss is typically less than the amount of total sleep lost. As two nights of sleep were lost (i.e. approximately 16 h sleep based on the baseline data), this theory would suggest that recovery should occur after two nights of recovery sleep. It is clear, however, that this did not occur. Rather, recovery was only approximately 70% complete – even after five 9-h sleep opportunities.

It is possible that the lack of recovery was an indication of boredom because of repeated testing. However, it is unlikely that this is the case given that a similar number of testing sessions occurred in the moderate 9-h TIB group, yet recovery still occurred. Moreover, performance remained at baseline levels for the entire period, rather than decreasing because of over-testing. Rather, we believe this data indicates that the mechanisms underlying the recovery process may be more complicated than previously thought. It has typically been assumed that the system is elastic, with neurobehavioural performance returning to baseline levels reasonably quickly. In the current study, however, a residual deficit was observed, with individuals not recovering despite an extended recovery period. This suggests that either recovery rate is very slow (i.e. recovery may have occurred if the recovery period was extended further) or that there is a fundamental change to the system (i.e. a reset process) following severe sleep loss, such that the baseline level of performance is shifted. Further research, including studies that increase the number of recovery nights or the length of the recovery sleep opportunity, is needed to investigate these theories.

Sleepiness and psychomotor vigilance performance

Finally, another interesting finding was that psychomotor vigilance performance and physiological sleepiness (as measured by the tendency to initiate sleep), recovered differently in two of the conditions. While PVT performance following moderate sleep loss recovered after one 9-h recovery opportunity, two 9-h recovery opportunities were required before sleep latencies did not significantly differ from baseline levels. Despite the fact that PVT performance did not recover following severe sleep loss, sleepiness followed a similar pattern following severe sleep loss as it did following moderate sleep loss (i.e. recovery after two 9-h opportunities). Similarly, unlike PVT performance, ratings of sleepiness also recovered to baseline levels following severe sleep loss. Notably, subjective recovery occurred after only one 9-h sleep opportunity, independent of the severity of the preceding sleep loss.

These findings emphasize that human performance cannot be reliably inferred from measures of sleepiness. While sleepiness is often assumed to be the intervening variable that mediates sleep-loss induced performance deficit, it is apparent that impairment can still be evident, despite recovery of physiological and subjective sleepiness. This also supports the argument that sleepiness and fatigue (also because of non-sleep-related factors) are both conceptually and operationally different. Thus, the fact that an individual no longer feels overtly sleepy, does not necessarily mean that they are recovered.

CONCLUSIONS

The current study suggests that following one night of sleep loss, individuals require two recovery sleep opportunities that are at least 9 h in length for both vigilance performance and sleepiness to recover. When recovery sleep opportunity is restricted to 6 h, even five consecutive recovery sleep opportunities are insufficient to reverse the effects of moderate sleep loss and restore waking function. However, rather than continuous decline, waking function stabilizes at below-baseline levels. Interestingly, when the severity of the sleep loss is increased, vigilance performance fails to recover, even after five augmented recovery sleep opportunities, yet sleepiness is restored to baseline levels after only one (subjective) or two (physiological) augmented recovery sleep opportunities. This data suggests that the mechanisms underlying the recovery process may be more complicated than previously thought, and that we may have underestimated the impact of sleep loss and/or overestimated the restorative value of subsequent sleep.

There is clearly a need for further research in this area, including studies that also focus on the impact of factors such as age, shiftwork experience and the timing of the recovery sleep opportunity on the rate of recovery.

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