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Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study

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SUMMARY Daytime performance changes were examined during chronic sleep restriction or augmentation and following subsequent recovery sleep. Sixty-six normal volunteers spent either 3 ($n = 18$), 5 ($n= 16$), 7 ($n = 16$), or 9 h ($n = 16$) daily time in bed (TIB) for 7 days (restriction/augmentation) followed by 3 days with 8 h daily TIB (recovery). In the 3-h group, speed (mean and fastest 10% of responses) on the psychomotor vigilance task (PVT) declined, and PVT lapses (reaction times greater than 500 ms) increased steadily across the 7 days of sleep restriction. In the 7- and 5-h groups speed initially declined, then appeared to stabilize at a reduced level; lapses were increased only in the 5-h group. In the 9-h group, speed and lapses remained at baseline levels. During recovery, PVT speed in the 7- and 5-h groups (and lapses in the 5-h group) remained at the stable, but reduced levels seen during the last days of the experimental phase, with no evidence of recovery. Speed and lapses in the 3-h group recovered rapidly following the first night of recovery sleep; however, recovery was incomplete with speed and lapses stabilizing at a level comparable with the 7- and 5-h groups. Performance in the 9-h group remained at baseline levels during the recovery phase. These results suggest that the brain adapts to chronic sleep restriction. In mild to moderate sleep restriction this adaptation is sufficient to stabilize performance, although at a reduced level. These adaptive changes are hypothesized to restrict brain operational capacity and to persist for several days after normal sleep duration is restored, delaying recovery.

KEYWORDS chronic sleep restriction, modeling, partial sleep deprivation, performance, recovery, sleep deprivation, sleep restriction, sleep

INTRODUCTION

Chronic sleep restriction is endemic in modern society. Americans are sleeping less (Bliwise *et al.* 1992; Webb and Agnew 1975) and, in absolute terms, a large fraction is reporting daily sleep substantially below the recommended optimum of 8 h per night (National Sleep Foundation 2002; Sleep in America Poll), although the true optimum may be higher (Coren 1997; Palinkas *et al.* 1995) or lower (Harrison and Horne 1995; Kripke *et al.* 2002). Sleep deprivation and chronic sleep restriction degrade health, safety, productivity (individual and societal) and quality of life (Bonnet and Arand 1995; Leger 1994; Mitler *et al.* 1988; Pilcher and Huffcutt 1996; Spiegel *et al.* 1999). In contrast to the apparent endemic nature of chronic sleep restriction, acute total sleep deprivation (TSD) occurs infrequently outside of the sleep laboratory.

Despite the prevalence of sleep restriction in modern society, few studies have evaluated the performance effects

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of chronic sleep restriction (e.g. Carskadon and Dement 1981; Dinges *et al.* 1997, 1999; Friedman *et al.* 1977; Horne and Wilkinson 1985; Kuo *et al.* 1998; Mullaney *et al.* 1977; Webb and Agnew 1974). Most studies to determine the behavioral effects of sleep loss have used acute TSD as the experimental manipulation. TSD studies are more efficient than chronic sleep restriction studies as the former produce larger behavioral decrements over a shorter period of time. Because of the paucity of well-controlled sleep restriction studies, findings from acute TSD studies, and specifically findings with respect to the accumulation and discharge of pressure for electroencephalographic (EEG) slow wave activity, have generally been used in the development of mathematical models to predict performance on the basis of prior sleep–wake history (Achermann *et al.* 1993; Akerstedt and Folkard 1995, 1996, 1997; Borbely 1982; Borbely *et al.* 1989; Folkard and Akerstedt 1991; Jewett and Kronauer 1999; Jewett *et al.* 1999). These models, both in terms of degradation and recovery functions, are anchored at the extremes of normal sleep on the one hand and TSD on the other with little objective data on what goes on in between (i.e. during restricted sleep). In sum, little is known about the effect of chronic sleep restriction on objectively measured alertness and performance and on the time course of subsequent recovery.

The purpose of the present study was to empirically determine the effects of several levels of restricted and one level of augmented sleep over seven consecutive days on objective and subjective alertness and objective performance, and to determine the extent to which 3 days of subsequent recovery sleep restored performance and alertness to baseline levels – a sleep dose–response study.

The data reported here are a subset of data collected in a larger study analysed and published as a US Department of Transportation Report (Balkin *et al.* 2000). In the present paper we report findings for psychomotor vigilance task performance (PVT), sleep latency, and subjective sleepiness. Of the measures taken in the larger study, the PVT was chosen for this paper because it was the most sensitive to the effects of sleep restriction and was the least subject to learning effects (Balkin *et al.* 2000).

METHODS

Subjects

Sixty-six volunteers (16 women, age 24–55, mean = 43 years; and 50 men, age 24–62, mean = 37 years) participated. All subjects held valid Commercial Motor Vehicle (CMV) drivers' licenses. Subjects were in good general health as determined by medical history and medical examination and were free of neurological diseases, psychiatric disorders, sleep disorders, and drug or alcohol addiction. They did not use nicotine in any form and reported consuming no more than 300–400 mg caffeine per day. Subjects were medication-free (including over-the-counter medications) beginning 48 h prior to the study, with the exception that female subjects could continue birth control medications.

Design

Volunteers spent 14 days in-residence in the laboratory (Fig. 1); a 'day' is defined as the time from the beginning of one scheduled sleep period to the beginning of the next. The first 3 days (T1, T2 and B) were adaptation and training (T1 and T2) and baseline (B) and subjects were required to be in bed from 23:00 to 07:00 h [8 h required time in bed (TIB)]. On the third day (B), baseline measures were taken. Beginning on the fourth day and continuing for a total of 7 days (E1–E7) subjects were in one of four sleep conditions [9 h required TIB (22:00–07:00 h), 7 h required TIB (24:00–07:00 h), 5 h required TIB (02:00–07:00 h), or 3 h required TIB (04:00–07:00 h)], effectively one sleep augmentation condition, and three sleep restriction conditions. The purpose of these conditions was to produce differing levels of sleep restriction/augmentation relative to the adaptation and training and baseline days. Beginning on the eleventh day and continuing for a total of 3 days (R1–R3) subjects were again required to be in bed from 23:00 to 07:00 h (8 h required TIB). These last 3 days constituted recovery. Subjects obtained a final night of 8 h TIB prior to release from the study; however, no testing occurred following this fourth night of recovery sleep. Throughout all phases of the study, lights on for all subjects was 07:00 h. Subjects were not permitted any other time in bed

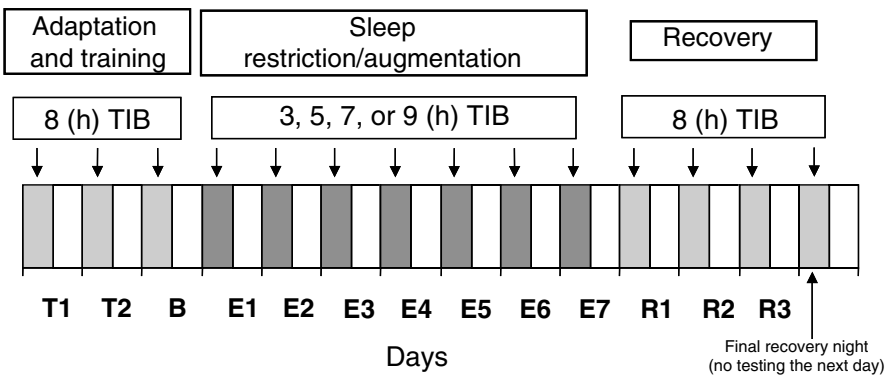

Figure 1. Study experimental design, showing nightly time in bed across days (adaptation/training, baseline, experimental phase, recovery phase).

or opportunity for sleep except as required by the periodic sleep latency tests (described below).

Test instruments and measures

Psychomotor vigilance test

The PVT measures simple reaction time to a visual stimulus, presented approximately 10 times/minute (interstimulus interval varied from 2 to 10 s in 2-s increments) for 10 min and implemented in a thumb-operated, hand-held device (Dinges and Powell 1985). Subjects attended to the LED timer display on the device and pressed the response button with the preferred thumb as quickly as possible after the appearance of the visual stimulus. The visual stimulus was the LED timer turning on and incrementing from 0 at 1-ms intervals. In response to the subject's button press, the LED timer display stopped incrementing and displayed the subject's response latency for 0.5 s, providing trial-by-trial performance feedback. At the end of this 0.5-s interval the display turned off for the remainder of the foreperiod preceding the next stimulus. Foreperiods varied randomly from 2 to 10 s. Dependent measures, averaged or summed across the 10-min PVT session, 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 and C4); EOG (outer canthi of each eye), EMG (mental/submental)], and EKG (from just below left and right clavicle) were recorded continuously throughout the study using Medilog 9000-II magnetic cassette recorders (Oxford Instruments, Largo, FL, USA). Raw data were digitized, and both night-time sleep and sleep latency tests (described below) were scored in accordance with Rechtschaffen and Kales (1968) criteria using Eclipse software (Stellate Systems, Westmont, Quebec, Canada).

Night-time sleep

Six technicians, whose inter-rater reliabilities were at least 85% compared with the scoring of a diplomate of the American Board of Sleep Medicine (TJB), scored night-time sleep periods (defined as lights out to lights on). Dependent measures included minutes of individual sleep stages [1, 2, slow wave sleep (SWS) and REM] and minutes of total sleep time (TST) (sum of minutes spent in all sleep stages).

Sleep latency test

Subjects were placed in bed in a quiet, darkened room and instructed to close their eyes and not resist the urge to fall asleep. Staff monitored PSG signals from outside of the bedroom using Oxford Mentor systems. To decrease the likelihood of premature test termination during ambiguous stage 1, the sleep latency test (SLT) was terminated immediately after the onset of stage 2 sleep (or after 20 min without sleep onset) by a staff member opening the bedroom door, turning on the lights, and announcing that the test was over. As stage 1 sleep does not appear to confer recuperative benefit in otherwise normal, healthy adults (Wesensten *et al.* 1999), the potential accumulation of up to 40 min stage 1 sleep daily (20 min per SLT × 2 SLTs per day) was not expected to affect performance. SLT schedules were staggered by 25 min for subject roommates, so that each could be tested in the bedrooms individually. For purposes of analyses, sleep latency was re-scored off-line from lights out to the first 30 s of stage 1 sleep.

Subjective alertness/sleepiness

The Stanford Sleepiness Scale (SSS; Hoddes *et al.* 1973) assessed subjective sleepiness on 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'). The dependent measure was the subject's sleepiness rating.

Procedure

Subjects reported to the Division of Neuropsychiatry, Walter Reed Army Institute of Research at 10:00 h on the day prior to T1. After being provided with verbal and written descriptions of study procedures and rules, subjects were individually informed of the sleep schedule to which their group (of two to four subjects) was being assigned and electrodes for ambulatory PSG (Oxford Medilog 9200-II), including EOG, EMG, C3, C4, O1, O2, and EKG were applied. Subjects then underwent training on the various performance tasks. At 18:00 h, they were transported to the Johns Hopkins Bayview General Clinical Research Center (GCRC, Baltimore, MD, USA) where they resided until the end of the study. Throughout the study, meals were served at 08:30, 12:30, and 17:30 h, with snacks and beverages available *ad libitum* between performance tests. Vital signs (blood pressure, pulse, and tympanic temperature) were recorded periodically for purposes of checking general health status. Subjects did not use/consume nicotine or caffeine-containing products during the study; random urine drug screens verified compliance. Use of medications during the study (e.g. acetaminophen for headache) was allowed at the discretion of the attending physician. For all women enrolled in the study, serum pregnancy tests performed at the beginning of the study were negative.

Same-sex subject pairs were assigned to share 2-person hospital-style bedrooms. T1 and T2 were devoted to training on the performance tests and familiarization with study procedures. Baseline testing commenced on the morning of the third day (B) and testing continued for the duration of the study (E1–E7, R1–R3). On the morning of R4 electrodes were removed shortly after awakening, and subjects were debriefed and released from the study. No testing occurred on R4.

Test schedule

The PVT and SSS were administered together as a battery four times per day (09:00, 12:00, 15:00, and 21:00 h); the battery included other tests not reported here (see Balkin *et al*. 2000). The sleep latency test was administered at 09:40 and 15:30 h for all groups.

Subjects in the 3- and 5-h TIB groups performed an additional battery at 00:00 h and 02:00 h to occupy their additional time awake. The PVT and SSS were administered in this battery; however, as data from the 00:00 and 02:00 h sessions were not common to all TIB groups, these data were not included in the statistical analyses reported below.

Statistical analyses

The PVT, SLT, and SSS data were analysed using a mixed analysis of variance (ANOVA) for TIB group (3-, 5-, 7-, or 9-h), day (B, E1, E2, E3, E4, E5, E6, E7, R1, R2, and R3), and time of day [09:00, 12:00, 15:00, and 21:00 h for PVT and SSS; 09:30 and 15:40 h for multiple sleep latency test (MSLT)] with repeated measures on the latter two factors. The time of day factor was included to help determine whether sleep group-specific changes across days were mediated by phase shifts in the circadian rhythm of performance, and to help determine whether sleep restriction effects were primarily manifested at particular times of day. These possibilities would be suggested by a three-way Sleep Group × Day × Time of Day interaction. Therefore, in results reported below, only significant three-way interactions are reported (two-way interactions and main effects for time of day are not reported). Night-time sleep data were analysed using a two-way mixed ANOVA for TIB group and day, with repeated measures on the day factor. Procedures outlined by Kirk (1995) were followed for interpretation of interactions – first, simple main effects analyses were applied to all significant Sleep Group–Day interactions (i.e. simple main effects of day separately for each sleep group, and simple main effects of sleep group separately for each day). Next, post hoc comparisons (Tukey's Honestly Significant Difference procedure or 'HSD' – Kirk 1995) were conducted for each significant simple main effect to specify differences among mean values. Greenhouse–Geisser corrections (Kirk 1995) were applied to all repeated measures effects. Unless otherwise noted, a significance level of $P \leq 0.05$ was used for all statistical analyses.

RESULTS

Due to technical difficulties, one or more sessions of PVT performance data were lost for some subjects from each sleep group. The final *n* for each sleep group and demographic data are reported in Table 1. Neither the distribution of females and males (chi-square analyses) nor the mean age (one-way analyses of variance) differed among TIB groups (both analyses $P > 0.05$).

Table 1 Demographic data for each time in bed (TIB) group

TIB group	*No. of females/ mean age (year)*	*No. of males/ mean age (year)*
3 h ($n = 13$)	5/39.2	8/39.6
5 h ($n = 13$)	2/45.5	11/32.5
7 h ($n = 14$)	4/43.8	10/38.6
9 h ($n = 16$)	4/47.0	12/37.4

Night-time sleep

Total sleep time

Figure 2 shows mean TST (TST = sum of stages 1, 2, SWS and REM) for each group across B, E1–E7, and R1–R3. TST increased significantly in the 9-h group and decreased significantly in the 3-, 5- and 7-h groups across the sleep restriction/augmentation phase (E1–E7) compared with baseline (B) (group, $F_{3,61} = 486.53$, $P = 0.0000$; night, $F_{10,610} = 281.10$, $P = 0.0000$; Group × Night, $F_{(30,\ 610)} = 141.83$, $P = 0.0000$). Average TST over the 7 days of sleep restriction/augmentation were 7.93 h for the 9-h TIB group, 6.28 h for the 7-h TIB group, 4.66 h for the 5-h TIB group, and 2.87 h for the 3-h TIB group.

Table 2 lists *F*-values and corresponding Tukey HSD comparisons for group and day simple effects. TST significantly differed among all TIB groups on nights E1–E7 but not on B or R1–R3. Within each TIB group, nightly sleep amounts did not change across E1–E7. For all groups, TST amounts appeared to return to baseline values during recovery, i.e. B was not different from R1–R3 for any group.

Sleep stages

Amounts of stage 1, 2 and REM sleep changed in a dose-dependent fashion during the experimental phase (E1–E7); stages 1, 2, and REM significantly decreased in the 3- and 5-h TIB groups while stages REM and 1 significantly increased in the 9-h TIB group (group simple effects, $P < 0.05$). Amounts of stages 1 and 2 also decreased in the 7-h TIB group, albeit non-significantly ($P > 0.05$). Sleep stage amounts returned to baseline values during recovery for all groups (B not different from R1–R3; Tukey HSD, $P > 0.05$). There were no group differences on B or R1–R3 (group simple effects, $P > 0.05$). The SWS amounts failed to vary significantly across experimental days as a function of TIB group ($P > 0.05$).

Psychomotor vigilance test

Mean speed

Figure 3 shows mean PVT response speed {[1/mean reaction time (RT)] × 1000} as a function of TIB group and day (collapsed across time of day), and Table 3 lists *F*-values and corresponding Tukey HSD comparisons for TIB group and day simple effects. Table 4 lists standard deviations for mean speed as a function of day and TIB group. Response speed

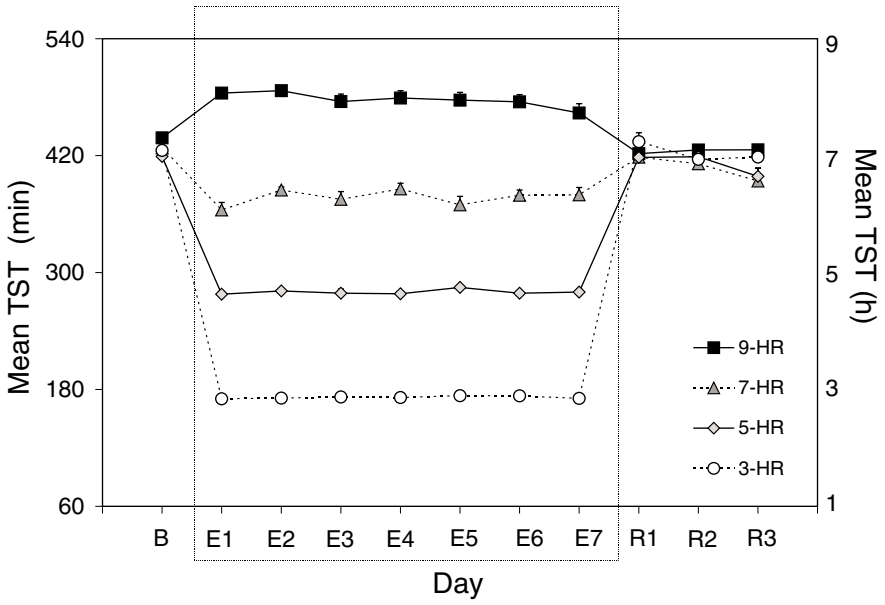

Figure 2. Mean total daily sleep durations in minutes (and hours – right-hand ordinate) as a function of time in bed group. Error bars (standard error of the mean) are included but are obscured by data points.

Table 2 Analyses of variance results of simple effects (TIB group, day) for total sleep time (TST). (a) Simple effects of TIB group for each day, (b) Simple effects of day for each TIB group

(a) *Day*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
B	1.07	3	61	0.3675	*
E1	826.44	3	61	0.0000	18.32
E2	2239.87	3	61	0.0000	11.35
E3	523.54	3	61	0.0000	22.50
E4	691.37	3	61	0.0000	20.05
E5	537.50	3	61	0.0000	22.00
E6	829.63	3	61	0.0000	17.87
E7	434.82	3	61	0.0000	24.07
R1	0.84	3	61	0.4766	*
R2	0.55	3	61	0.6506	*
R3	2.22	3	61	0.0944	*
(b) *TIB group*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
3 h	566.72	10	610	0.0000	24.30
5 h	143.19	10	610	0.0000	25.77
7 h	12.60	10	610	0.0000	26.62
9 h	20.80	10	610	0.0000	25.77

TIB, time in bed; TST, total sleep time, HSD, honestly significant difference.
*HSD values are not applicable to non-significant simple effects.

decreased across experimental days in a TIB dose-dependent manner for the 3-, 5-, and 7-h TIB groups (group $F_{3,52} = 5.72$, $P = 0.0018$; day $F_{10,520} = 19.01$, $P = 0.0000$); Group × Day $F_{30,520} = 6.64$, $P = 0.0000$; simple effects for day at each group are given in Table 3. Speed for the 9-h TIB group did not vary across days. For the 3-h TIB group, speed on days E2 through E7 was lower than B; within the experimental phase, E3–E7 were lower than E1 and E2; E6 was lower than E1–E3; and E7 was lower than E1–E5. Speed increased significantly in the 3-h TIB group from E7 to R1, but no further speed increases were evident during recovery (R1 not different from R2 and R3); and speed failed to recover to baseline levels in the 3-h TIB group (R1–R3 < B). For the 5-h TIB group, speed was significantly impaired on E3–E7 compared with B; within the experimental phase E5–E7 were significantly lower than E1 and E2. Although speed appeared to increase from E7 to recovery days, the difference was not significant (E7 not different from R1–R3). Speed in the 5-h TIB group failed to recover (R1–R3 < B). For the 7-h group, speed was lower on R2 and R3 compared with B and E1. Within days, during the experimental phase speed for the 3-h TIB group was significantly lower than speed for 9-h group on E2–E7, significantly lower than speed in the 7-h group on E3–E7, and significantly lower than speed in the 5-h group on E7. Speed for the 5-h group was significantly lower than that for the 9-h group on E5. Speed did not differ between the 7- and 9-h TIB groups during the experimental phase. During the recovery phase, speed for the 3-h TIB group was significantly lower than speed for the 9-h TIB group on R1 and R3. Speed did not differ among the 5-, 7-, and 9-h TIB groups on any recovery day.

The three-way Group × Day × Time of Day interaction was not significant ($P = 0.1018$).

Number of lapses

Figure 4 shows mean number of lapses across days as a function of TIB group, and Table 5 lists *F*-values and corresponding Tukey HSD comparisons for TIB group and day simple effects. Lapses increased across the experimental phase in the 3- and 5-h groups but not in the 7- and 9-h groups (group $F_{3,52} = 7.29$, $P = 0.0004$; day $F_{10,520} = 15.84$, $P = 0.0000$; Group × Day $F_{30,520} = 6.90$, $P = 0.0000$). For the 3-h group, more lapses occurred on E3–E7 compared with B; on E3–E7 compared with E1 and E2; on E5–E7 compared

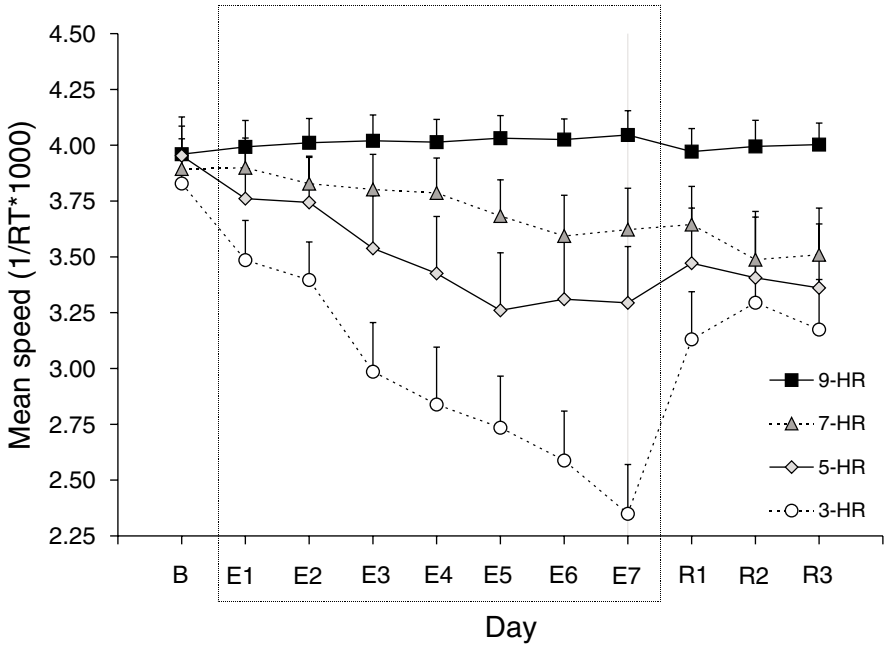

Figure 3. Mean psychomotor vigilance task speed (and standard error) across days as a function of time in bed group.

Table 3 Analyses of variance results of simple effects (TIB group, day) for PVT mean speed. (a) Simple effects of TIB group for each day, (b) Simple effects of day for each TIB group

(a) *Day*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
B	0.18	3	52	0.9079	*
E1	1.87	3	52	0.1457	*
E2	2.95	3	52	0.0412	0.5909
E3	6.05	3	52	0.0013	0.7127
E4	6.95	3	52	0.0005	0.7634
E5	8.89	3	52	0.0001	0.7417
E6	9.77	3	52	0.0000	0.7633
E7	14.20	3	52	0.0000	0.7524
R1	3.74	3	52	0.0165	0.7216
R2	2.62	3	52	0.0602	0.7790
R3	3.15	3	52	0.0325	0.8091
(b) *TIB group*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
3 h	26.11	10	520	0.0000	0.3824
5 h	7.14	10	520	0.0001	0.3824
7 h	3.25	10	520	0.0177	0.3685
9 h	0.11	10	520	0.9669	*

TIB, time in bed; TST, total sleep time; HSD, honestly significant difference; PVT, psychomotor vigilance task.
*HSD values are not applicable to non-significant simple effects.

Table 4 Standard deviations of mean speed for each day and time in bed (TIB) group

	TIB Group			
Day	*3-HR*	*5-HR*	*7-HR*	*9-HR*
B	0.5032	0.7173	0.5711	0.528
E1	0.6972	0.8667	0.5335	0.5001
E2	0.7412	0.8144	0.5275	0.4632
E3	0.9471	0.8925	0.6275	0.5028
E4	1.0648	0.9835	0.7213	0.4626
E5	0.9543	1.1022	0.6469	0.4431
E6	0.9205	1.0715	0.7728	0.4119
E7	1.0131	1.0149	0.7619	0.4723
R1	0.8825	0.9543	0.7575	0.4673
R2	0.7227	1.0193	0.8799	0.5169
R3	0.8296	1.1119	0.8544	0.4492

with E3; and on E7 compared with E3–E5. Lapses decreased from E7 to recovery (E7 > R1–R3) but failed to return to baseline levels (R1 and R3 > B). For the 5-h TIB group, more lapses occurred on E6 and E7 compared with B; lapses failed to recover (R2 and R3 > B). During the experimental phase more lapses occurred in the 3-h TIB group than (a) the 9-h group on E3–E7; (b) the 7-h group on E4–E7; and (c) the 5-h group on E6 and E7. Mean number of lapses did not differ among the 5-, 7- and 9-h TIB groups during the experimental phase. During the recovery phase, more lapses occurred in the 3-h TIB group compared with the 9- h group on R1. Lapses did not differ among the 5-, 7-, and 9-h TIB groups on any recovery day.

A significant three-way Group × Day × Time of Day interaction ($F_{(30,520)} = 1.67$, $P = 0.0235$) indicated that the time of day effect (more lapses at 09:00 h vs. 21:00 h) was greatest in the 3-h group, and that these time of day differences in the 3-h group increased across the experimental phase. There was no evidence that the *timing* of the peak or trough in number of lapses shifted across experimental days for any TIB group.

Mean speed – fastest 10% of responses

To verify that sleep dose-dependent effects on mean speed were not due solely to an increased number of lapses, mean speed for the fastest 10% of responses also was analysed. Figure 5 shows mean speed for the fastest 10% of response as a

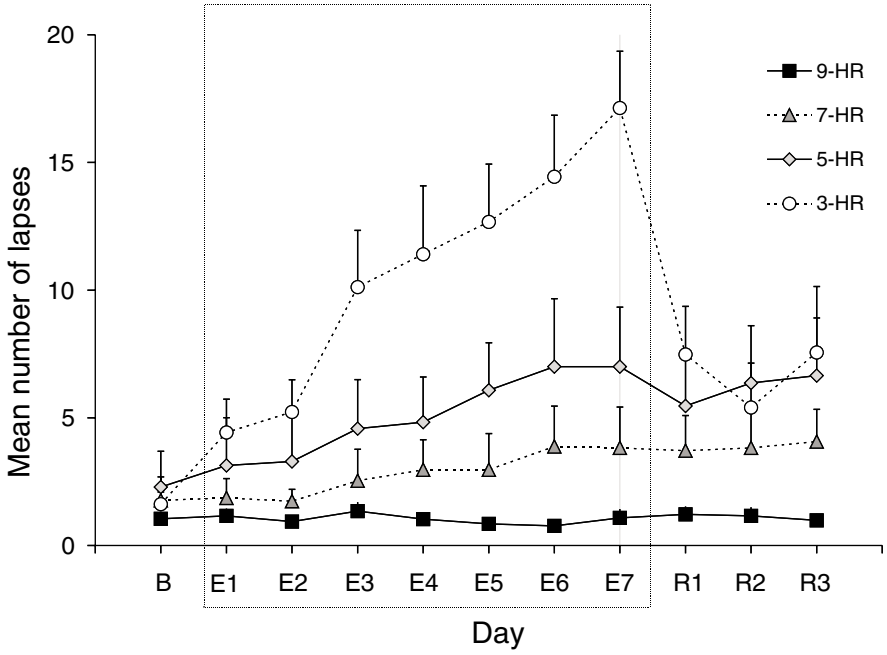

Figure 4. Mean number of lapses on the psychomotor vigilance task (and standard error) across days as a function of time in bed group.

Table 5 Analyses of variance results of simple effects (TIB group, day) for PVT number of lapses. (a) Simple effects of TIB group for each day, (b) Simple effects of day for each TIB group

(a) *Day*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
B	0.38	3	52	0.7660	*
E1	1.63	3	52	0.1946	*
E2	2.93	3	52	0.0421	4.3497
E3	6.55	3	52	0.0008	5.9189
E4	7.75	3	52	0.0002	6.3229
E5	11.22	3	52	0.0000	6.0335
E6	10.08	3	52	0.0000	7.2513
E7	16.71	3	52	0.0000	6.6998
R1	3.58	3	52	0.0198	5.6057
R2	2.42	3	52	0.0767	*
R3	3.09	3	52	0.0350	6.7452
(b) *TIB group*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
3 h	29.69	10	520	0.0000	3.9439
5 h	3.69	10	520	0.0051	3.9439
7 h	1.21	10	520	0.3082	*
9 h	0.05	10	520	0.9972	*

TIB, time in bed; HSD, honestly significant difference; PVT, psychomotor vigilance task.
*HSD values are not applicable to non-significant simple effects.

function of TIB group and day (collapsed across time of day), and Table 6 lists results of the ANOVA. The pattern for the fastest 10% of responses was similar to that found for mean speed: response speed decreased across experimental days in a TIB dose-dependent manner for the 3- and 5-h TIB groups but not for the 7- or 9-h groups. For the 3-h TIB group, speed on days E3–E7 was lower than on B; for the 5-h TIB group, speed on days E2–E7 was lower than on B. For both the 3- and 5-h TIB groups, speed increased non-significantly from E7 to R1; speed among days R1–R3 did not differ, and speed on all recovery days was lower than baseline (R1–R3 < B). During the experimental phase, speed for the 3-h TIB group was significantly lower than speed for the 7 and 9-h TIB groups on E3–E7; but mean speed did not differ among the 5-, 7-, and 9-h TIB groups during the experimental phase. During the recovery phase, although mean speed for the 3-h TIB group appeared to remain lower than that of the 9-h TIB group, these differences were not significant. Mean speed did not differ among the 5-, 7-, and 9-h TIB groups on any recovery day.

Objective and subjective sleepiness

Figure 6 shows latency to the first 30 s of stage 1 sleep from the SLT, and Fig. 7 shows sleepiness ratings from the SSS across days as a function of TIB group (means collapsed across time of day).

Daytime sleep latency

On the baseline day (B), average sleep latency was 6.3 min ± 0.64 SEM across all groups. Mean sleep latency did not differ between TIB groups on Baseline (TIB group simple effect, $P > 0.05$). Latency decreased across experimental days for the 3- and 5-h TIB groups, but not for the 7- and 9-h TIB groups (group $F_{3,50} = 7.61$, $P = 0.0003$; day $F_{(10,500)} = 4.29$, $P = 0.0002$; Group × Day: $F_{(300,500)} = 2.31$, $P = 0.0014$; day simple effects: 3-h, $F_{(10,\ 500)} = 4.29$, $P = 0.0002$; 5-h, $F_{(10,500)} = 4.29$, $P = 0.0002$; $P > 0.05$ for 7- and 9-h TIB groups). For the 3-h TIB group, latency on E5 was significantly shorter than B (Tukey HSD = 3.65, $P < 0.05$).

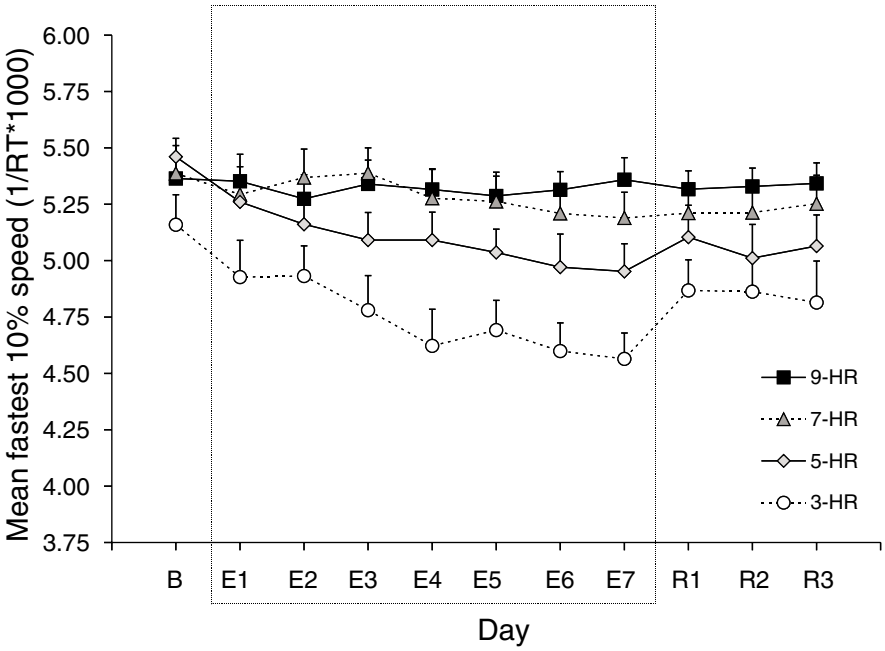

Figure 5. Mean psychomotor vigilance task speed for the fastest 10% of responses (and standard error) across days as a function of TIB group.

Table 6 Analyses of variance results for PVT mean fastest 10% of responses. (a) Main effects and interactions, (b) Simple effects of TIB group at each day, (c) Simple effects of day for each TIB group

(a) *Effect*	*F*	DF_{num}	DF_{denom}	*P*	
TIB Group (G)	4.57	3	52	0.0065	
Day (D)	11.81	10	520	0.0000	
Time of day (T)	9.48	3	156	0.0001	
G × D	2.75	30	520	0.0004	
G × T	0.55	9	156	0.7799	
D × T	0.98	30	1560	0.4767	
G × D × T	0.87	90	1560	0.7077	
(b) *Day*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
B	1.20	3	52	0.3185	*
E1	2.12	3	52	0.1089	*
E2	2.32	3	52	0.0857	*
E3	5.03	3	52	0.0039	0.4828
E4	6.25	3	52	0.0011	0.4942
E5	5.86	3	52	0.0016	0.4439
E6	6.98	3	52	0.0005	0.4702
E7	9.47	3	52	0.0000	0.4399
R1	2.61	3	52	0.0609	0.4668
R2	2.91	3	52	0.0431	0.4818
R3	3.01	3	52	0.0383	0.5291
(c) *TIB group*	*F*	DF_{num}	DF_{denom}	*P*	*HSD value*
3 h	9.93	10	520	0.0000	0.2546
5 h	6.72	10	520	0.0000	0.2546
7 h	1.88	10	520	0.0924	*
9 h	0.33	10	520	0.9070	*

TIB, time in bed; HSD, honestly significant difference, PVT, psychomotor vigilance task.
*HSD values are not applicable to non-significant simple effects.

Although latency appeared to increase from the experimental to the recovery phase in the 3-h group, differences between experimental and recovery days were not significant (Tukey HSD, $P > 0.05$); also, although it appeared that sleep latencies failed to recover to baseline levels, R1–R3 were not significantly different from B (Tukey HSD, $P > 0.05$). Significant time ($F_{1,50} = 23.03$, $P = 0.0000$) and Group × Time ($F_{3,50} = 9.3$, $P = 0.0000$) effects indicated dose-ordered differences in sleep latency (9- > 7- > 5- > 3-h) in the morning but no sleep latency differences in the afternoon.

Stanford sleepiness scale

Sleepiness ratings increased significantly during the experimental phase in the 3-h group but did not change in the 5-, 7-, and 9-h groups (group $F_{3,56} = 3.05$, $P = 0.0359$; day $F_{(10,560)} = 6.58$, $P = 0.0000$; Group × Day: $F_{(30,\ 560)} = 4.42$, $P = 0.0000$; day simple effect for 3-h, $F_{(10,560)} = 18.28$, $P = 0.0000$; day simple effects for 5-, 7-, and 9-h groups, $P > 0.05$). For the 3-h group, sleepiness ratings on E2–E7 were significantly higher than B, and E5–E6 were higher than E1 (Tukey HSD = 0.44, $P < 0.05$). Sleepiness ratings decreased significantly from experimental to recovery days (R1–R3 < E2–E7; Tukey HSD $P < 0.05$) and returned to baseline during the latter (R1–R3 not different from B; Tukey HSD $P > 0.05$). Within days, sleepiness ratings for the 3-h group were significantly higher than other TIB groups as follows: E2 ($F_{3,56} = 4.54$, $P = 0.0064$), 3 > 9; E3 ($F_{3,56} = 4.28$, $P = 0.0086$), 3 > 7, 9; E4 ($F_{3,56} = 2.79$, $P = 0.0488$), Tukey HSD, NS; E5 ($F_{3,56} = 6.01$, $P = 0.0013$) and E6 ($F_{3,56} = 5.46$, $P = 0.0023$), 3 > 7, 9; and E7 ($F_{3,56} = 3.97$, $P = 0.0123$), 3 > 7. No differences among TIB groups were found on B, E1, and R1–R3 (group simple effects, $P > 0.05$).

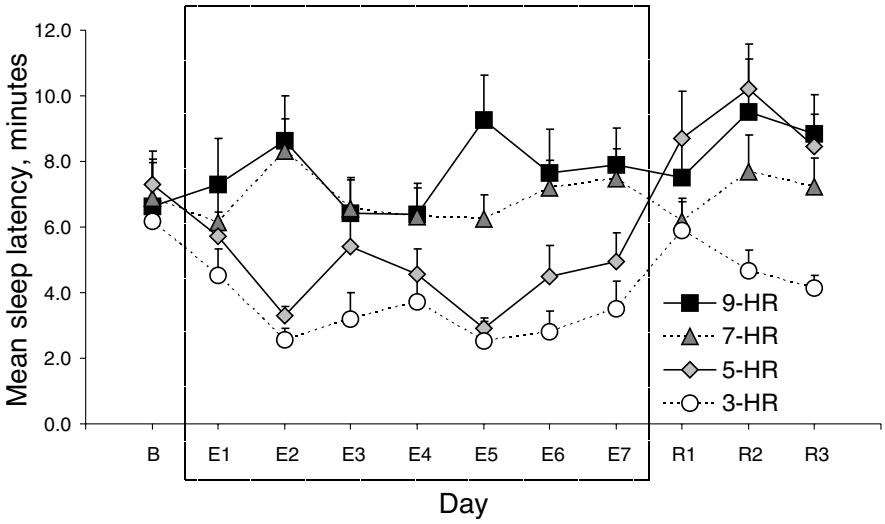

Figure 6. Mean latency to the first 30 s of stage 1 sleep (and standard error) across days as a function of time in bed group.

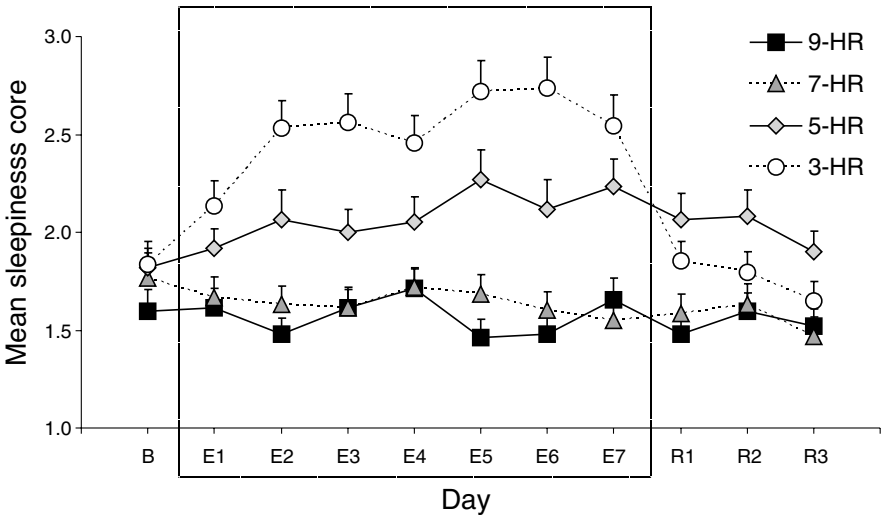

Figure 7. Mean Stanford Sleepiness Scale scores (and standard error) across days as a function of time in bed group.

Significant effects for time of day ($F_{3,168} = 4.27$, $P = 0.0121$), and Group × Time of Day ($F_{9,168} = 2.39$, $P = 0.0255$) indicated that dose-dependent differences in sleepiness scores were maximal in the morning.

DISCUSSION

Seven days of sleep restriction degraded psychomotor vigilance performance in a sleep-dose-dependent manner. With mild to moderate sleep restriction (7- and 5-h TIB), performance initially declined and, after a few days, appeared to stabilize at a lower-than-baseline level for the remainder of the sleep restriction period. In contrast, with severe sleep restriction (3-h TIB) performance declined continuously across the sleep restriction period, with no apparent stabilization of performance. Sleep augmentation (9-h TIB) had no effect on performance over the 7-day experimental period. Based on these findings, it appears that the inflection point (i.e. the minimum amount of nightly sleep required to achieve a state of equilibrium in which daytime alertness and performance can be maintained at a stable, albeit reduced, level) is approximately 4 h per night. If less than 4 h of sleep per night is obtained, daily decrements in performance capacity may be unavoidable – at least across a 7-day period of sleep restriction.

Core vs. optional sleep

These findings are reminiscent of Horne's hypothesis that a minimum amount of nightly sleep (approximately 4.5 h per night) is required to satisfy the brain's physiological need for recuperation (core sleep), and that any sleep obtained in excess of this minimum is essentially appetitive (Horne 1988). According to this view, utilization of spare cerebral capacity enables the brain to perform well with less than 8 h of sleep each night. The possibility of such spare cerebral capacity is

consistent with the suggestion of Drummond *et al.* (2000) that cognitive performance during sleep deprivation involves recruitment of resources from additional, non-task specific – and therefore relatively non-fatigued – brain regions.

Although the present findings tend to support Horne's hypothesis that there is a physiological limit to the ability to tolerate sleep restriction and that this limit is approximately 4 h of sleep per night, these findings also suggest a more continuous gradient of recuperative value than that suggested by the core sleep and optional sleep designations. For example, our finding that PVT performance was decremented during recovery following 7 days of a mild, 1-h reduction of nightly TIB – from 8 to 7 h – suggests that meaningful recuperation continues to accrue as sleep durations are extended beyond the hypothesized core sleep requirement. The present findings suggest that core sleep might best be considered as the minimum amount of sleep needed by the brain to achieve a state of equilibrium in which alertness and performance are maintained at a stable but lower-than-normal level. In this view, sleep durations that do not satisfy the core sleep requirement would, across days, result in continued degradation of alertness and performance; whereas sleep durations that satisfy the core requirement would produce deficits in alertness and performance relative to baseline, but degradation would not continue across days indefinitely – an asymptotic, stable level of reduced alertness and performance would eventually be achieved; and additional sleep (i.e. incremental increases in the duration of sleep beyond the core requirement) would produce correspondingly higher, and stable, levels of alertness and performance.

Performance stabilization during, and failure to recover following, sleep restriction – evidence for neuromodulatory changes

Following chronic, mild to moderate sleep restriction (5 or 7 h TIB), 3 days of recovery sleep (8-h TIB) did not restore performance to baseline levels. These findings suggest that in response to chronic sleep restriction the brain undergoes adaptive changes that serve to sustain a stable (albeit reduced) level of performance; the findings further suggest that these changes persist into the recovery period and prevent rapid return to baseline performance. Such adaptive changes may act as a rate limiter or governor that reduces (caps) the operational capacity of the brain, allowing the brain to operate in the face of a restricted sleep budget. They may serve to prevent injury from occurring in the brain if it continued to perform at full capacity in the face of restricted sleep. Plausible hypothetical mechanisms underlying such an adaptive response to sleep restriction include alterations in gene expression. Adaptive alterations in gene expression could, in turn, cause relatively persistent changes in synaptic transmission (neuromodulation) (Siegelbaum *et al.* 2000). The possibility that acute TSD and chronic sleep restriction could each produce distinct and different patterns of up and down regulation in gene expression is being investigated in our laboratory.

The nature of sleep dose-dependent performance effects

It has been hypothesized (e.g. Lubin 1967; Williams *et al.* 1959) that all sleep-loss-induced performance deficits are the result of 'lapses' in performance – perhaps due to brief episodes of EEG-defined sleep, and that performance between lapses (i.e. during EEG-defined wakefulness) may be unaffected by sleep loss. Although this hypothesis has generally and repeatedly been shown to be incorrect (for review, see Dinges and Kribbs 1991), we tested this hypothesis by analyzing the fastest 10% of responses (as this subset of the data would not be impacted by occasional lapses to the same extent as measures of central tendency). However, as expected, this analysis revealed a pattern of results that was comparable with that produced by analysis of mean speed, suggesting that performance degradation during sleep restriction in the present study cannot be explained solely by an increased incidence of lapses. Had speed decrements been restricted only to a few outliers (lapses), it could be argued that the effects were not neuromodulatory but rather were due to an increased frequency of transitory attention lapses against a background of otherwise normal performance. The finding that the fastest 10% of responses (i.e. responses other than lapses) as well as mean speed was affected suggests a persistent, pervasive change in brain function.

Total sleep deprivation vs. chronic sleep restriction

The pattern of results from the present study do not support the general assumption that acute TSD and chronic sleep restriction are physiologically equivalent phenomena, differing only in magnitude and time course. The present findings indicate that acute TSD and chronic sleep restriction cause differential performance effects that are most salient during the recovery period. That is, chronic sleep restriction leads to long-time-constant changes that may have adaptive value, serving to stabilize performance. However, these adaptive changes appear to come at a cost – brain operational capacity is capped in a manner that apparently precludes rapid recovery to baseline levels of alertness and performance when sleep durations are extended to baseline levels. In contrast, rapid recovery to baseline is typical of performance following acute TSD.

Sleep latency as a gauge of performance capacity

In studies of sleep deprivation and sleep restriction, sleepiness is generally (albeit often implicitly) assumed to be the intervening variable that mediates sleep loss-induced performance deficits. Measures such as the MSLT and maintenance of wakefulness test (MWT) – both of which measure the tendency to initiate sleep – are considered relatively direct ways to gauge the extent of the brain's physiological need for sleep (Carskadon and Dement 1981). In contrast, psychomotor performance (while obviously impacted by sleep loss) is thought to reflect sleepiness less directly because of the relatively wide

array of additional, non-sleep-related factors known to impact performance. Therefore, it is interesting to note that in the present study TIB groups were more reliably distinguished by PVT performance than by sleep latency scores. This may have been due in part to sleep latency floor effects and/or relatively reduced statistical power (because SLTs were administered half as often as PVTs). However, it may also have been due to the fact that sleep latency scores were somewhat restricted, with a mean sleep onset latency of 6.3 min (i.e. near the pathological range) on the baseline day (despite spending 8 h TIB on the previous three nights). This finding is consistent with those of Harrison and Horne (1996) who reported that sleep latencies for a large portion of their normal subjects were within the pathological range – prompting them to hypothesize that individuals vary considerably with respect to 'sleepability' – the capacity to initiate sleep regardless of sleep need. A crucial implication of these findings is that human performance capacity cannot be reliably inferred from sleep latency data and as it is ultimately human performance capacity (not tendency to initiate sleep) that determines safety and productivity in the operational environment as well as quality of life for sleep disordered patients, greater efforts to define fitness for duty and severity of sleep-related disorders on the basis of measured performance capacity are warranted.

Implications for modeling and performance prediction

Extant mathematical models predicting alertness and performance from preceding sleep–wake history typically involve three factors – sleep homeostasis, circadian rhythm, and sleep inertia (Akerstedt and Folkard 1997). These three-factor models are relatively successful in predicting the effects of acute TSD on alertness and performance and the time course of subsequent recovery. They are less successful in predicting the effects of chronic sleep restriction on alertness and performance and the time course of subsequent recovery (Van Dongen and Dinges 2002; Van Dongen *et al*. 2002). It appears that this failure cannot be remedied by better model parameterization. Rather, this failure likely represents a basic structural inadequacy of three-factor models in predicting the effects of chronic sleep restriction. The present findings suggest that incorporation of a fourth factor would substantially improve prediction – a factor (presumably reflecting neuromodulatory changes) representing slow (days, weeks) adaptation to chronic sleep restriction and slow de-adaptation following restoration of normal sleep amounts.

Is there an 'optimum' sleep amount?

With respect to the question of what constitutes optimum sleep, our findings that performance tracked sleep in a dose-dependent manner across the range of sleep restriction/augmentation suggests that humans benefit from progressively greater nightly amounts of sleep (although the amount of benefit accrued with progressively greater sleep amounts may be asymptotic; Harrison and Horne 1995). Future work will replicate and extend these and other findings in chronic sleep restriction, describe the time course of performance degradation and recovery in both acute TSD and chronic sleep restriction, and characterize individual differences in tolerance to sleep deprivation and sleep restriction. The present results highlight the need for careful attention to the sleep–wake history of subjects for the weeks (or even months) preceding the actual experimental observations. Further studies of sleep restriction will facilitate the development of mathematical models with greater power and generality for the prediction of alertness and performance based on sleep–wake history.

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