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Time course of sleep inertia dissipation in human performance and alertness

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SUMMARY Alertness and performance on a wide variety of tasks are impaired immediately upon waking from sleep due to sleep inertia, which has been found to dissipate in an asymptotic manner following waketime. It has been suggested that behavioural or environmental factors, as well as sleep stage at awakening, may affect the severity of sleep inertia. In order to determine the time course of sleep inertia dissipation under normal entrained conditions, subjective alertness and cognitive throughput were measured during the first 4 h after habitual waketime from a full 8-h sleep episode on 3 consecutive days. We investigated whether this time course was affected by either sleep stage at awakening or behavioural/environmental factors. Sleep inertia dissipated in an asymptotic manner and took 2–4 h to near the asymptote. Saturating exponential functions fitted the sleep inertia data well, with time constants of 0.67 h for subjective alertness and 1.17 h for cognitive performance. Most awakenings occurred out of stage rapid eye movement (REM), 2 or 1 sleep, and no effect of sleep stage at awakening on either the severity of sleep inertia or the time course of its dissipation could be detected. Subjective alertness and cognitive throughput were significantly impaired upon awakening regardless of whether subjects got out of bed, ate breakfast, showered and were exposed to ordinary indoor room light ($\approx$150 lux) or whether subjects participated in a constant routine (CR) protocol in which they remained in bed, ate small hourly snacks and were exposed to very dim light (10–15 lux). These findings allow for the refinement of models of alertness and performance, and have important implications for the scheduling of work immediately upon awakening in many occupational settings.

KEYWORDS sleep inertia, circadian, alertness, performance, model, REM sleep, awakening

INTRODUCTION

It has been well documented that alertness and performance are impaired immediately upon waking from sleep. This impairment, referred to as 'sleep inertia', has been observed in a wide variety of performance tasks, including short-term memory, vigilance and other measures of cognitive functioning, as well as reaction time, ability to resist sleep and grip strength (for review see Dinges 1990). These adverse effects of sleep inertia are greatest immediately after awakening (Seminara and Shavelson 1969). However, even in studies involving emergency response simulations (Seminara and Shavelson 1969) and complex tasks such as flight simulations (Hartman and Langdon 1965), sleep inertia was evident for the entire 10–12-min period that the subjects were tested. Although sleep inertia has been reported to be worse near the trough of the core body temperature cycle (Wilkinson and Stretton 1971; Dinges *et al.* 1985; however, see Naitoh *et al.* 1993), sleep inertia has been observed upon awakening from sleep at all times of day and night (Wilkinson and Stretton 1971; Rosa *et al.* 1983; Dinges

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et al. 1985; Downey and Bonnet 1987; Lavie and Weler 1989; Tassi *et al*. 1992; Naitoh *et al*. 1993). Both partial and total sleep deprivation have been found to increase the intensity of sleep inertia, perhaps due to increasing intensity of sleep pressure or depth of sleep (Balkin and Badia 1988; Dinges 1990). In addition, sleep inertia is evident even if subjects were not previously sleep deprived (Achermann *et al*. 1995). Although the presence of intense continuous noise (Tassi *et al*. 1992) and continuous intensive cognitive performance for 10 min following awakening (Langdon and Hartman 1961) have been reported to help alleviate sleep inertia, neither source of stimulation is sufficient to completely or consistently eradicate the performance decrements associated with sleep inertia. It is thought that the sleep stage at awakening may affect the severity of sleep inertia, but it may be instead that 'sleep depth', defined as the auditory arousal threshold required to wake a subject, is a better predictor of the intensity of sleep inertia (Dinges 1990).

Estimates of how long sleep inertia lasts have varied widely, ranging from 1 min (Webb and Agnew 1964) to more than 3 h (Naitoh 1981) following awakening. To clarify the time course of the dissipation of sleep inertia, two studies have been conducted in which frequent tests have been given during the first 30–60 min of wakefulness after either a full night's sleep (Folkard and Åkerstedt 1992; Achermann *et al*. 1995) or after an afternoon nap (Achermann *et al*. 1995). Those studies found that upon awakening, subjective alertness (Folkard and Åkerstedt 1992; Achermann *et al*. 1995) and short-term memory scores (Achermann *et al*. 1995) improved in a saturating exponential manner. After fitting the data with saturating exponential curves, the time constant for the increase in subjective alertness was estimated to be 0.66 h by Folkard and Åkerstedt (1992) and to be 0.45 h by Achermann *et al*. (1995). The time constant for the improvement in short-term memory was estimated to be 0.30 h (Achermann *et al*. 1995). Unfortunately, Folkard and Åkerstedt (1992) gave no tests between $\approx$15 min and $\approx$55 min after awakening and Achermann *et al*. (1995) gave no tests between 1 h and 3 h after awakening, so that the entire time interval over which sleep inertia dissipation may occur was not sampled in either study. In addition, neither study reported any verification that the scheduled laboratory waketimes corresponded with the subjects' actual waketimes. Thus, it is possible that some subjects may have spontaneously awakened prior to their scheduled waketimes, thereby reducing the apparent sleep inertia measured in the performance and alertness tests.

The current study was conducted to clarify the time course of sleep inertia dissipation in both subjective alertness and cognitive throughput. In this study, subjects were tested 5–6 times in the first hour after awakening, and 1–2 times/h in the second, third and fourth hours after awakening, so that the entire time interval over which sleep inertia is expected to occur was sampled. Unlike previous studies (Folkard and Åkerstedt 1992; Achermann *et al*. 1995), the actual waketimes of our subjects were determined from polysomnographic (PSG) data, rather than simply relying on scheduled waketimes. This has allowed us to evaluate whether the stage of sleep prior to awakening from a full night of sleep at habitual waketime influences the severity or time course of sleep inertia. In this study we have also investigated whether ambient lighting and/or postural and behavioural conditions affect sleep inertia, by comparing sleep inertia tests taken on normal scheduled days (ambulatory, regular meals, $\approx$150 lux ambient light intensity) to those taken under constant routine conditions (semi-recumbent posture, small snacks, $\approx$10–15 lux ambient light intensity).

METHODS

Subjects

Fifteen young men (mean age $\pm$ SD, 22.7 $\pm$ 3.4 years; range, 19–29 years) who were free from medical, psychiatric and self-reported sleep disorders were studied. Subjects were instructed to abstain from caffeine, nicotine, alcohol, medications and illicit drugs for 3 weeks before their study, and they were drug-free at the time of study, as verified by urinary toxicological analysis. All subjects denied a history of night work or shift work in the 3 years prior to study, and none reported crossing more than two time zones in the 3 months prior to study. Subjects were instructed to keep a regular sleep–wake schedule (bedtimes and waketimes within 1 h of self-selected target times) during the 3 weeks prior to their admission to the laboratory. Adherence to a regular schedule during the week prior to admission was verified with wrist actigraphy (Vitalog Monitoring, Inc., Redwood City, CA, USA; Ambulatory Monitoring, Inc., Ardsley, NY, USA). Thus, all waketimes reported here should have occurred at a stable circadian phase in all subjects.

Protocol

In this study, we have analysed the data collected after waketime during the first 3 days from subjects who were participating in an 11–16-day experiment in the laboratory in an environment free of time cues (for detailed description of protocol, see Jewett *et al*. 1997). During this time, subjects were scheduled to sleep for 8 h each night at their habitual times. During the waking portion of the first 2 days, subjects were ambulatory and exposed to ordinary indoor room light (referred to below as the *150-lux ambulatory* condition). On the third day, subjects were exposed to very dim light ($\approx$10–15 lux) during a constant routine (CR) protocol designed to measure endogenous circadian phase and amplitude (referred to below as the *dim light CR* condition). During this CR, subjects remained awake in a semi-recumbent posture for up to 30 h (see Czeisler and Jewett 1990 for details).

At the scheduled waketime each morning, a technician entered the room, awakened the subject verbally, adjusted the lights to the appropriate level (dim light or $\approx$150 lux), and raised the head of the subject's bed and the subject's knees to a comfortable position. It required $\approx$1 min to complete this awakening procedure, after which time subjects took the

neurobehavioural tests (described below) from this semi-recumbent position whenever the tests appeared on their bedside computer. In the *150-lux ambulatory* conditions, subjects got out of bed ≈35 min after waketime and took subsequent tests from a seated position at a desk. In the *dim light CR* condition, the subjects remained in bed throughout the entire waking period and took all their tests from a semi-recumbent position. In the *150-lux ambulatory* condition, subjects ate breakfast ≈1.25 h after scheduled waketime and took a shower ≈2.33 h after scheduled waketime. In the *dim light CR* condition, subjects were not allowed to shower, and were given hourly snacks starting at ≈1.25 h after waketime.

Polysomnograph recordings and scoring

In order to evaluate the relationship between sleep parameters and the degree of subsequent sleep inertia, as well as to determine the subjects' actual waketimes, the sleep stage data from the last hour prior to scheduled waketime were analysed. Sleep data were acquired to a Nicolet Ultrasom (Nicolet Biomedical, Madison, WI, USA) digital sleep–wake analyser system. Scalp EEG sites C3, C4, O1 and O2 were paired with contralateral mastoid reference electrodes. Bipolar submental EMG, two channels of referential EOG and 2-lead EKG were also recorded. Sleep records were scored in 30-s epochs by standard criteria (Rechtschaffen and Kales 1968). Scored waketime was defined to be the time of the first epoch that was scored as wakefulness in the series of uninterrupted wake epochs leading to the scheduled waketime. In the event that a subject was asleep until the scheduled waketime, scored waketime was equal to scheduled waketime. Final sleep stage was defined as the last stage of sleep prior to scored waketime in which the subject remained asleep for at least 1 min (2 contiguous 30-s epochs). PSG data from waketime 3 were unavailable for one subject (no. 1659), and thus the sleep inertia battery from that waketime has been omitted from all further analysis.

Subjective alertness and cognitive throughput measurements

The sleep inertia computerized test battery consisted of measurements of both subjective alertness and cognitive throughput. Subjective alertness was measured using a 100 mm visual analogue scale (VAS), with one end labelled 'alert' and the other end labelled 'sleepy'. Using a mouse or trackball, subjects placed a mark at the point at which they felt best described their level of alertness and the VAS was then scored as mm from the 'sleepy' end of the scale. Cognitive throughput was measured using a 2-min addition task (ADD) in which subjects were asked to complete sums of two 2-digit numbers as quickly and as accurately as possible. When the answer to each set of two numbers was entered, a new set appeared on the screen. The ADD was scored as the number of sums attempted in 2 min, irrespective of errors.

As is shown in Fig. 1, the sleep inertia test battery consisted of VAS and ADD tests given at approximately: 1 min, 11 min, 21 min, 31 min, 51 min, 60 min (VAS only); 71 min, 90 min, 120 min (VAS only), 150 min, 180 min (VAS only), 210 min, and 240 min (VAS only) after scheduled waketime. Following the sleep inertia test battery, the VAS was given every 30 min and the ADD was given every 1 h for the remainder of wakefulness each day.

Data analysis

For each subject, the mean VAS and ADD scores were calculated for tests given between hours 2 and 26 (a full circadian cycle) from the CR that occurred on their third day of study. All scores from the sleep inertia test batteries were then converted into deviation from this mean, in order to account for interindividual differences in interpretation of the VAS and in mathematical ability (ADD test). Each VAS and ADD test was assigned a time since scored waketime, as determined by the PSG criteria described above. Within each subject, the scores from the VAS and ADD collected during the sleep inertia test battery each day were averaged in four 15-min bins during the first hour after scored waketime, two 30-min bins during the second hour after scored waketime and one 2-h bin during the third and fourth hour after scored waketime.

Two-way ANOVAs with factors 'final sleep stage' and 'hours since scored waketime' were conducted using these binned VAS and ADD data from all three waketimes. For the binned VAS data, saturating exponential functions were fitted to the data within each final sleep stage group and Student's *t*-tests were conducted between the first test bin in the sleep inertia battery (centred at 7.5 min after scored waketime) and the last test bin in the sleep inertia battery (centred at 3 h after scored waketime). Two-way ANOVAs with factors 'waking condition' [*150-lux ambulatory (waketimes 1 and 2)* vs. *dim light CR(waketime 3)*] and 'hours since scored waketime' were conducted on the binned subject data. In order to investigate the possibility of an order effect (since the waking conditions always occurred in the same order), two-way ANOVAs with factors 'day of study' and 'hours since scored waketime' were also conducted. Saturating exponential functions were fitted to the binned data from each waketime ($n=44$) for the VAS and ADD data and Student's *t*-tests were conducted between the first and last sleep inertia test battery bins. The data from all three waketimes were then pooled together for each neurobehavioural measure, and a Student's *t*-test was conducted between the first and last sleep inertia test battery bins. Finally, saturating exponential curves were fitted to the pooled bins for the VAS and ADD tests. The SAS program (SAS Institute, Inc., Cary, NC, USA) was used for all parameter estimation and statistical analysis.

RESULTS

Final sleep stages

Scheduled waketimes occurred 2.01 ± 1.06 h (mean ± SD) after the fitted minimum of the core body temperature (CBT_{min})

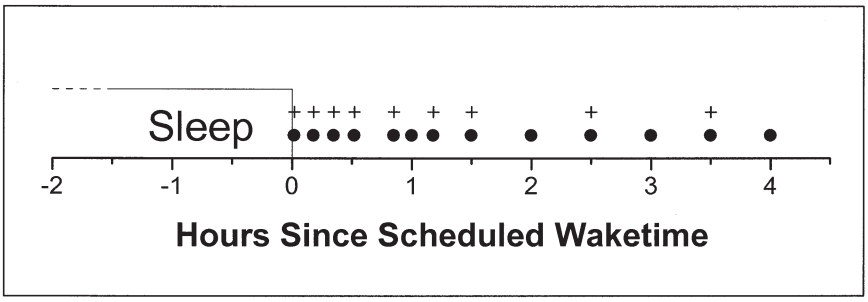

Figure 1. Schematic diagram of the sleep inertia test battery. Filled circles indicate approximate times when the visual analogue scale (VAS) was taken and plus signs indicate approximate times when the addition test (ADD) was taken.

measured during the CR (Brown and Czeisler 1992), and scored waketimes occurred 1.90 ± 1.04 h (mean ± SD) after CBT_{min}. In 16 (≈36%) of the waketimes the final sleep stage was rapid eye movement (REM) sleep, in 8 (≈18%) of the waketimes the final sleep stage was Stage 1 sleep, in 19 (≈43%) of the waketimes the final sleep stage was Stage 2 sleep, and in one (≈2%) of the waketimes the final sleep stage was Stage 3 sleep. Data from this single Stage 3 sleep waketime was omitted from further sleep stage analyses due to low sample size for this level of the factor 'final sleep stage'.

There was a significant effect of the factor 'hours since scored waketime' on the VAS [$P<0.0001$,variance accounted for (VA) = 17.9%] and ADD ($P<0.0001$, VA = 13.2%) scores. There was neither a significant interaction with nor a significant effect of the factor 'final sleep stage' on the ADD data (Fig. 2b). However, there was a small but significant effect of 'final sleep stage' on the VAS data ($P<0.02$, accounting for less than 2.5% of the variance) with no interaction (Fig. 2a). This is most probably due to the overall lower scores of the Stage 1 data (Fig. 2a, filled circles), since 'final sleep stage' is no longer significant when Stage 1 waketimes are excluded from the ANOVA. As can be seen in Table 1, the time course of sleep inertia dissipation is similar for all final waking stages in the VAS data. In addition, all final sleep stages showed significant differences between the first and last sleep inertia VAS test battery bins (Stage 1: $P<0.0007$; Stage 2: $P<0.0001$; REM: $P<0.0002$).

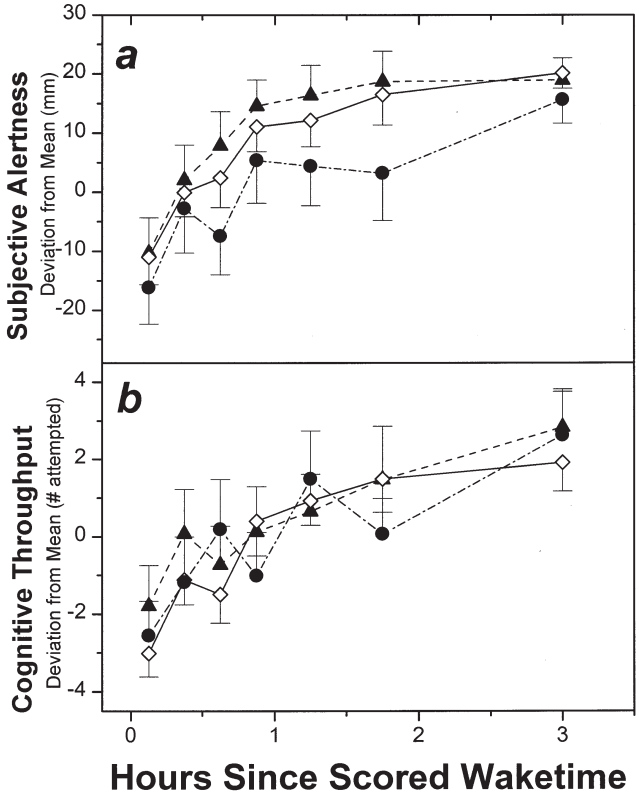

Figure 2. (a) Subjective alertness (measured using the VAS) for waketimes in which the final sleep stages were Stage 1 sleep (filled circles), Stage 2 sleep (open diamonds) and REM sleep (filled triangles). Bin means ± SEM of VAS scores (measured on a 0–100-mm scale) are presented as deviation from the mean alertness score measured for each subject between hours 2 and 26 of the subject's constant routine (CR). (b) Cognitive throughput (measured using the ADD) for final sleep Stages 1, 2 and REM (symbols as in panel a). Bin means ± SEM of ADD scores (measured as number of problems attempted in 2 min) are presented as deviation from the mean throughput score measured for each subject between hours 2 and 26 of the subject's constant routine (CR).

Waking conditions: 150-lux ambulatory vs. dim light CR

Both VAS and ADD scores varied significantly with hours since scored waketime (VAS: $P<0.0001$, VA = 18.01%; ADD: $P<0.0001$, VA = 10.7%) and with waking condition [VAS: $P<0.0001$, VA = 5.85%; ADD: $P<0.052$ (trend), VA = 1.1%]. There was no interaction between 'hours since scored waketime' and 'waking condition' for either the VAS or the ADD scores.

In order to investigate the possibility that the effect of waking condition on the VAS and ADD scores might actually be due to an order effect (since the *dim light CR* condition always occurred on the third waketime), the binned sleep inertia battery data from each of the three waketimes were plotted (Fig. 3) and a two-way ANOVA with factors 'day of study' and 'hours since scored waketime' was conducted. When the data from all three waketimes were included in the two-way ANOVAS, both VAS and ADD scores varied significantly with day of study (VAS: $P<0.0001$,VA = 8.0%; ADD: $P<0.002$, VA = 3.6%)

Table 1 Parameters values of best fit to VAS and ADD data using the model: Score = A + C*e*((-1/T)*Hours Since Scored Waketime)

	Asymptote (A)	*Coefficient (C)*	*Time constant (T)*
VAS dataset			
Waketimes from Stage 1 sleep	15.79 (11.77)	−32.47 (10.60)	1.17 h (1.10)
Waketimes from Stage 2 sleep	20.42 (4.70)	−36.26 (6.03)	0.76 h (0.33)
Waketimes from REM sleep	19.33 (3.96)	−38.65 (8.55)	0.47 (0.22)
Waketime 1 (150 lux ambulatory)	25.25 (4.82)	−32.97 (6.99)	0.67 (0.35)
Waketime 2 (150 lux ambulatory)	20.22 (4.15)	−42.17 (6.42)	0.64 (0.23)
Waketime 3 (dim light CR)	10.96 (5.36)	−33.14 (7.56)	0.70 (0.40)
Pooled waketimes	18.88 (2.86)	−35.84 (4.22)	0.67 (0.19)
ADD dataset			
Waketime 1 (150 lux ambulatory)	3.33 (1.97)	−7.53 (1.73)	1.27 (0.82)
Waketime 2 (150 lux ambulatory)	4.08 (1.66)	−6.22 (1.48)	1.22 (0.83)
Waketime 3 (150 lux ambulatory)	−0.14 (0.39)	−6.82 (10.02)	0.12 (0.17)
Pooled waketimes (dim light CR)	2.69 (0.94)	−5.58 (0.86)	1.17 (0.52)

Asymptotic standard errors are shown in parentheses.

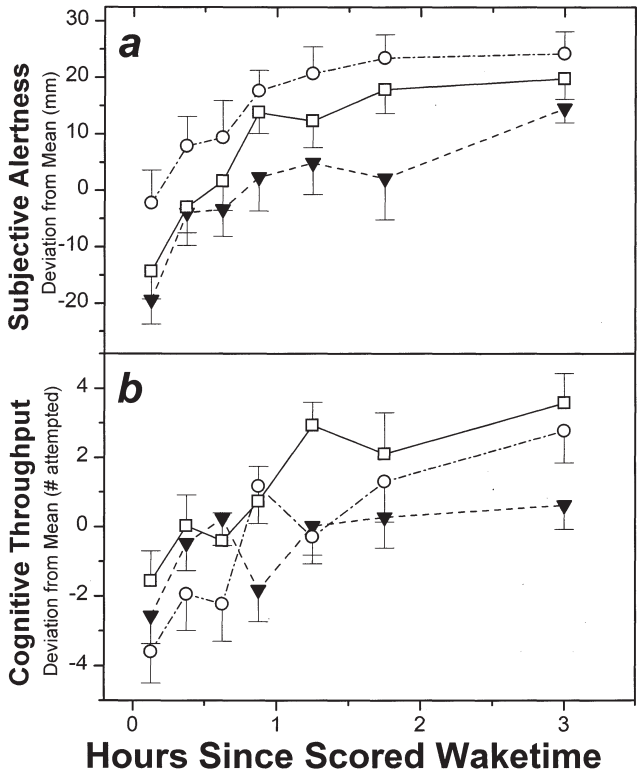

Figure 3. (a) Subjective alertness for waketime 1 (*150-lux ambulatory* condition, open circles), waketime 2 (*150-lux ambulatory* condition, open squares) and waketime 3 (*dim light CR* condition, filled triangles). Data plotted as in Figure 2(a). (b) Cognitive throughput for waketimes 1, 2 and 3 (symbols as in panel a). Data plotted as in Figure 2b.

and with hours since scored waketime (VAS: $P<0.0001$, VA = 21.4%; ADD: $P<0.0001$, VA = 15.5%), with no significant interaction. Once again, the effects of day of study were small, accounting for only a small percentage of the total variance.

As can be seen in Fig. 3(a), the VAS scores from waketime 1 (open circles) appear to be higher than those from waketime 2 (open squares), which in turn are higher than those from waketime 3 (filled triangles). However, as can be seen in Table 1, the time course of sleep inertia dissipation is similar for all three waketimes. In addition, the scores in the first sleep inertia VAS test battery bin are significantly lower than the scores in the last bin for all three waketimes (waketime 1: $P<0.0008$; waketimes 2 and 3: $P<0.0001$). In Fig. 3(b), the ADD scores from waketime 2 (open squares) appear to be higher than those from waketimes 1 (open circles) and 3 (filled triangles). When only the first and third waketimes were included in a two-way ANOVA with factors 'time since scored waketime' and 'day of study', ADD scores no longer varied significantly with day of study. Hours since waketime was still significant ($P<0.0001$, VA 14.2%) and showed a significant interaction with day of study ($P<0.04$, accounting for less than 6.2% of the variance). Thus, the significant effect of waking condition on the ADD scores can be primarily accounted for by the higher scores appearing on the second day of the *150-lux ambulatory* condition, rather than a difference due to the *dim light CR* condition. The scores in the first sleep inertia ADD test battery bin are significantly lower than the scores in the last bin for all three waketimes (waketime 1: $P<0.0001$; waketime 2: $P<0.0002$; waketime 3: $P<0.006$). In addition, when saturating exponential functions were fitted to these data, the time course for sleep inertia dissipation was somewhat faster, but not significantly different in the *dim light CR* condition than in the two *150-lux ambulatory* waketimes (see Table 1).

Time course of sleep inertia dissipation

In order to obtain an overall estimation of the time course of sleep inertia dissipation for subjective alertness and cognitive throughput, the data from all three waketimes were pooled together for the VAS (Fig. 4a) and ADD (Fig. 4b) tests. Both the VAS and ADD scores increased asymptotically, nearing the asymptote 2–4 h after scored waketime. The first bin (centred 7.5 min after scored waketime) was significantly lower than the last bin (centred 3 h after scored waketime) for both the

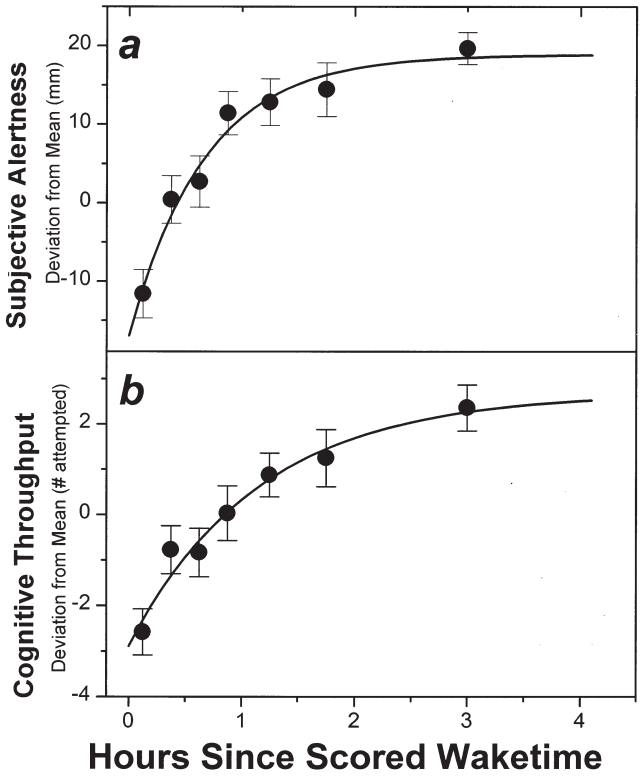

Figure 4. (a) Bin means ± SEM of subjective alertness for pooled waketimes 1, 2 and 3 (filled circles). Data plotted as in 2a. A saturating exponential function (solid line) was fitted to the data. (b) Bin means ± SEM of cognitive throughput for pooled waketimes 1, 2 and 3 (filled circles). Data plotted as in Figure 2b. A saturating exponential function (solid line) was fitted to the data.

VAS ($P<0.0001$) and the ADD ($P<0.0001$) data. A saturating exponential function provided a good fit to the data for each neurobehavioural measure (Fig. 4, solid lines; see Table 1 for parameter estimates).

DISCUSSION

Sleep inertia is often thought to be a fleeting phenomenon (Dinges 1990), but we found that subjective alertness and cognitive performance could be impaired for more than 2 h after awakening, even in subjects who were not sleep deprived and who were sleeping at their habitual times. Both subjective alertness and cognitive throughput scores were significantly lower when subjects first awoke than 2–4 h later in the day (Fig. 4). The subjective alertness and cognitive throughput scores rose rapidly in the first hour after awakening and then began to level off about 2 h after waketime, suggesting asymptotic dissipation of sleep inertia. Thus, our results support the hypothesis that sleep inertia dissipates in an asymptotic manner, which has been suggested previously by Folkard and Åkerstedt (1992) and Achermann *et al.* (1995). We also found that the time constant for the dissipation of sleep inertia in subjective alertness (0.67 h) was quite similar to that reported by Folkard and Åkerstedt (1992; $T=0.66$ h), although it was slightly larger than that reported by and Achermann *et al.* (1995; $T=0.45$ h). The time constant for the dissipation of sleep inertia in cognitive throughput ($T=1.17$ h) was much larger than that of subjective alertness or that reported for short-term memory by Achermann *et al.* (1995; $T=0.30$ h). This suggests that some neurobehavioural functions may be more sensitive to sleep inertia than others. In particular, performance on the cognitive throughput task may take much longer to recover from sleep inertia than is reflected in subjective self-assessments of alertness. However, these differences in time constant may also be due to methodological differences between these studies (see Introduction for details).

It has been suggested that sleep inertia may be due to a decline in cerebral metabolism resulting from thermal down-regulation during sleep (Dinges 1990). If this were true, then we would expect activities that increased cerebral metabolism and core body temperature to reduce the effects of sleep inertia. Surprisingly, for subjective alertness and cognitive throughput neither ambient lighting nor behavioural conditions (in bed/small snacks/10 lux vs. upright/showered/breakfast/150 lux) had a significant effect on either the severity of sleep inertia or the rate of its dissipation. However, there is a possibility that the effects of behaviour and lighting on sleep inertia are obscured by an order effect, since the *dim light CR* condition occurred after the *150-lux ambulatory* condition in all subjects. It may be that to detect the effects of behaviour on sleep inertia, subjects need to have more intense stimulation, leading to a sharper increase in cerebral metabolism. This stimulation may also need to occur sooner after waketime, since in our study subjects did not get out of bed until $\approx$35 min after waketime in the *150-lux ambulatory* condition.

As can be seen in Fig. 3, we found that overall ratings of subjective alertness declined across the first 3 days that subjects were in the laboratory. It is not clear whether the subjects were more alert near the beginning of their studies due to their unfamiliar surroundings or whether they required a few days to become accustomed to using the visual analogue scale. However, even if the subjects *were* more alert during their first few mornings in the laboratory, sleep inertia was clearly evident at all waketimes. In addition, the time courses of the dissipation of sleep inertia were very similar for all three waketimes (see Table 1), suggesting that unfamiliar surroundings are not sufficient to reduce the length of time that sleep inertia persists in subjective alertness.

Finally, we did not observe an effect, after a full night of sleep, of prior sleep stages on the intensity or duration of sleep inertia for either subjective alertness or cognitive throughput. Since subjects were scheduled to wake at their habitual times, it is not surprising that subjects woke out of Stage 1 ($n=16$), Stage 2 ($n=8$) or REM ($n=19$) sleep rather than out of slow wave sleep (SWS) (Stages 3 + 4 sleep, $n=1$). It may be that subjects waking out of SWS would show even more severe sleep inertia than that observed here (Dinges 1990). This is of particular relevance to emergency personnel, medical house staff and other extended-duty workers who are likely to need to perform following waketimes that occur at unusual circadian

phases, after shorter sleep episodes, and after prior sleep debt, which may increase the likelihood of waking out of SWS. To establish whether SWS may play a role in the severity of sleep inertia, further studies are needed in which the full time course of sleep inertia dissipation is investigated in subjects waking from SWS.

Sleep inertia has been included as an important component in a number of models of alertness and performance (Folkard and Åkerstedt 1992; Achermann and Borbély 1994; Jewett 1997). Two other major components of these models are a $\approx$24-h circadian component (*C*) and a homeostatic component (*H*) that declines during wakefulness and recovers during sleep. Recent data from a number of neurobehavioural measures indicate that the amplitude of *C* is dependent upon the level of *H* (Czeisler *et al*. 1994; Dijk *et al*. 1992; Jewett 1997; Wyatt *et al*. 1997), such that when subjects first awaken (and *H* is high) the amplitude of *C* is quite low, but as subjects are awake for longer periods of time (and *H* is lower) the amplitude of *C* increases. These findings suggest that *H* and *C* interact in a non-linear manner. Although in all current alertness and performance models, sleep inertia (*W*) acts independently of *H* and *C*, it is quite possible that *H* and *W* may also interact in a non-linear manner. For example, the magnitude of *W* at waketime and/or the time constant of the dissipation of *W* may increase when subjects have been previously sleep deprived (so that *H* is low). Similarly, *C* and *W* may interact such that sleep inertia is worse at some circadian phases than at others. Since we have only tested sleep inertia at one circadian phase in subjects who were sleep satiated, we are not able to determine from our data whether such interactions between *W* and *C* or *H* exist. Furthermore, we do not know the extent to which the improvement following waketime in our subjects may have been due to their waking during a rising portion of *C*. However, we think that this is unlikely to have played a major role, given the low amplitude of *C* just after waketime (see above). Clearly, further studies are needed in which the time course of sleep inertia dissipation is measured at different circadian phases and when subjects are carrying various sleep debts.

In summary, we found that both subjective alertness and cognitive performance were significantly impaired upon awakening, as reported in many previous studies (Dinges 1990), even in subjects who were not sleep deprived and who were awakened at their habitual time. It took longer for the impairment in cognitive throughput to dissipate than has been reported for other neurobehavioural metrics. Subjects did not approach their asymptotic levels of alertness and performance until 2–4 h after waketime. Sleep inertia seems to be a fairly robust phenomenon that was not highly sensitive to ambient lighting, behavioural conditions or PSG stage (Stage 1, 2 or REM) upon awakening from a full night of sleep. However, this hypothesis needs to be tested further in subjects who engage in intense arousing activities soon after waking in protocols where sleep inertia is sampled at regular intervals throughout the 2–4-h dissipation period.

The results presented here are useful in the refinement of sleep inertia parameters in mathematical models of alertness and performance. They are also important in the design of work schedules, especially when employees are allowed to sleep on-site and are expected to perform soon after awakening. For example, if prophylactic naps are used as a means of improving performance in long-duration flights (Rosekind *et al*. 1994), pilots must be given sufficient time after waking from the naps for sleep inertia to dissipate before they are required to perform at a high level. In fact, Ribak *et al*. (1983) found that even following a normal night's sleep, the nearer a pilot is to the hour of his waking, the higher is the chance of an accident occurring. Although our results suggest that the duration of impaired performance may extend for more than 2 h following a full night's sleep, further studies measuring the time course of sleep inertia dissipation in the field, particularly after short naps, are needed to determine the generalizability of our findings.

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