Sleep and Biological Rhythms 2003; **1**: 5–13

REVIEW ARTICLE

Sleep debt: Theoretical and empirical issues*

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Abstract

The term 'sleep debt' is widely used to describe the effects of sleep loss. The construct of sleep debt, however, is poorly defined in the scientific literature. Cumulative build-up of sleep pressure appears to be a key feature of sleep debt. The concepts of 'core sleep' and 'basal sleep need' have been proposed to provide a theoretical framework, albeit without strong empirical basis. It has been hypothesized that adaptation to sleep debt may be possible over time, but experimental evidence for this hypothesis is ambiguous. Recent experiments using chronic sleep restriction have revealed significant effects of sleep debt on daytime sleep latency and behavioral alertness. In a series of strictly controlled laboratory studies, we found that sleep debt can lead to fundamentally different daytime responses, depending on whether homeostatic sleep pressure (as measured in the waking electroencephalogram (EEG)) or behavioral alertness (as measured with psychomotor vigilance lapses) is considered. This suggests the existence of an as yet unidentified regulatory mechanism of waking neurobehavioral function. To study the nature of this regulatory process under chronic sleep restriction, advantage can be taken of the natural variability in sleep need frequently cited in the literature. We also obtained evidence for interindividual differences in vulnerability to sleep loss regardless of sleep need. Statistical modeling of the effects of chronic sleep restriction on behavioral alertness, taking into account these interindividual differences, provided a reference for defining sleep debt. The results suggested that sleep debt may be defined as the cumulative hours of sleep loss with respect to a subject-specific daily need for sleep.

Key words: behavioral alertness, chronic sleep restriction, cumulative deficits, sleep debt, sleep need.

SLEEP DEBT

The term 'sleep debt' is used extensively when discussing the adverse effects of untreated sleep disorders, the consequences of night-shift work and jet lag, the effects of development-related sleep restriction, and experimentally induced periods of sleep loss. However, its theoretical and empirical basis in the consequences of chronic sleep restriction are not well defined in the scientific literature. Forty years ago, Kleitman used the phrase 'sleep debt' to describe the circumstances of delaying sleep onset time while holding sleep termination time constant.[1] He described the increased sleepiness and decreased alertness in individuals on such a sleep–wake pattern, and proposed that those subjects who were able to reverse these effects by extending their sleep on weekends were able to 'liquidate the debt' (p. 317). Since this work appeared, and especially in the past decade, 'sleep debt' has been widely used to describe effects associated with sleep loss, regardless of

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*We dedicate this paper to the memory of Dr Martin P Szuba.

Accepted for publication 26 October 2002.

the reason for the lost sleep. Dement and Vaughan discussed the construct extensively and reported that some researchers refer to it as 'sleep load' or 'sleep tendency' (p. 62).[2] Even though it is widely used, a definition of 'sleep debt' has remained absent in standard textbooks of sleep such as *Principles and Practice of Sleep Medicine*[3] and the *Encyclopedia of Sleep and Dreaming.*[4]

Based on the manner in which the concept of sleep debt is most often used by Dement and Vaughan[2] and others, it refers to the increased pressure for sleep that results from an inadequate amount of physiologically normal sleep. As such, sleep debt may be expressed via increases in the propensity for sleep (e.g. sleep latency tests), in physiological indices of sleep homeostasis (e.g. sleep or waking electroencephalogram (EEG)), in subjective sleepiness, and in neurobehavioral performance. Cumulative build-up of sleep pressure, especially from inadequate recovery sleep over multiple days, appears to be a key feature of sleep debt; see the discussion in Dement and Vaughan[2] of an earlier paper by Carskadon and Dement.[5] When inadequate sleep is obtained repeatedly on consecutive occasions, sleep debt may be referred to as 'chronic'. It is widely believed that chronic sleep debt in healthy individuals commonly occurs as a result of voluntarily reduced sleep durations in order to achieve extension of the daily wakefulness period. Chronic sleep debt is thought to result in cumulative increases in homeostatic sleep drive, in addition to cumulative increases in waking neurobehavioral deficits. Little is known about the relationship between the cumulative sleep homeostatic responses to chronic sleep restriction relative to the cumulative waking responses.

Despite the heuristic utility of sleep debt as a construct and its widespread use, a number of fundamental issues and critical questions either have yet to be addressed experimentally, or have yet to be definitively resolved scientifically. These include, but are not limited to the following:

- What is the full range of physiological, cognitive, and behavioral responses to various dosages of restricted sleep over chronic periods of time? What is the relationship between sleep responses and waking responses to sleep debt? Does chronic sleep debt result in simultaneous increases of sleep homeostasis, sleep propensity, daytime sleepiness and/or cognitive deficits?
- In what ways are the physiological and neurobehavioral effects of sleep debt from chronic partial sleep restriction similar or different to those from acute sleep loss?
- What role do interindividual differences in basal sleep need, waking capabilities or circadian physiology play in the responses to sleep debt?
- What aspects of recovery sleep determine resolution of the sleep debt and its expression in sleep and waking?
- What are the neurobiological bases underlying accumulation of sleep debt and recovery from sleep debt?

CHRONIC SLEEP RESTRICTION

Experimental protocols that restrict sleep chronically across consecutive days provide the most appropriate mechanism for the study of sleep debt.[2] Very few studies, however, have effectively used this paradigm to quantify the effects of sleep debt in a well-controlled, methodologically rigorous manner. Many of the early experimental reports (prior to 1965) bordered on the anecdotal, lacking adequate sample sizes and control groups. Subsequent reports in the 1970s and 1980s failed to ensure that subjects maintained the assigned sleep–wake schedules; used infrequent, confounded and/or insensitive measures of sleep and waking; lacked sophisticated time series analyses; and generally drew conclusions not substantiated by the quantitative results (for reviews see[6,7]).

An even smaller number of studies actually evaluated the cumulative effects of sleep restriction for a week or more. In fact, the majority of experiments on chronic sleep restriction that involved six or more consecutive days of limited time for sleep were conducted prior to 1980. Most of these early investigations purported to have sleep chronically restricted to between 4.3 and 6.0 h per night, although few actually kept subjects in the laboratory under controlled conditions to ensure they only obtained the sleep permitted and that they took no stimulants (e.g. caffeine). Nevertheless, a number of these reports concluded that there were few, if any, detrimental effects of chronic sleep restriction on adult daytime neurobehavioral functions,[8–11] or on sleepiness[10] or mood.[8] Despite the negative results from these widely cited studies, a meta-analysis of the effects of acute total sleep deprivation and chronic partial sleep deprivation concluded that there was evidence that the latter had a more profound effect on functioning than the former.[12]

More recent experiments on the effects of sleep chronically restricted to less than 8 h per night for six or more days in healthy adults, with additional experimental controls and sensitive measures, have yielded

statistically significant effects on daytime sleep latency,[5] on daytime behavioral alertness as measured by psychomotor vigilance performance lapses,[7] on morning metabolic responses,[13] on endocrine functions,[13] and on immune functions.[14] Moreover, it appears that the sleep latency and behavioral alertness effects are directly related to the accumulation of sleep debt across days of sleep restriction.[5,7]

CORE SLEEP AND OPTIONAL SLEEP

It has been proposed by Horne that a normal nocturnal sleep period is comprised of two types of sleep relative to functional adaptation.[15,16] The initial sleep period of the night is referred to as 'core' or 'obligatory' sleep, which he posits 'repairs the effects of waking wear and tear on the cerebrum' (p. 57).[16] All sleep obtained beyond this 'core sleep' duration is considered to be 'optional' or 'facultative' sleep, which 'fills the tedious hours of darkness until sunrise' (p. 57). According to this theory, only the 'core' portion of sleep, especially that dominated by EEG slow wave activity, is required for adequate daytime alertness and functioning to be maintained. Additional 'optional' sleep obtained does not contribute to this, although at times in these writings, it is allowed that the deprivation of 'optional' sleep can lead to some daytime sleepiness, which is theoretically distinct from core sleepiness. The proposed physiological basis for core sleep is not unique, as other theories also attribute a special function to EEG slow-wave activity.[17]

Relative to the construct of sleep debt, the critical issue in Horne's core versus optional sleep theory is the duration of core sleep needed to prevent sleep debt. To the extent that core sleep duration is less than total sleep duration, there should be no cumulative development of sleep debt as defined by cognitive deficits indicative of cerebral dysfunction. Initially, Horne placed this core duration at 4–5 h of sleep per night, depending on the chronicity of sleep restriction (pp. 62–63).[16] Faced with recent evidence of cumulative physiological sleepiness and neurobehavioral deficits at this level of nocturnal sleep, Horne has increased core sleep to be 6 h of good-quality, uninterrupted sleep for most adults (Horne JA, personal communication, 2002).

ADAPTATION TO SLEEP RESTRICTION

If it is true that not all of the sleep obtained is required for waking neurocognitive functions to remain unaffected, then it may be possible for subjects to adapt to sleep restricted to durations above or at the core threshold, such that decrements in neurobehavioral functioning do not manifest or are very slow to develop. A few early experiments attempted to test this hypothesis, but methodological limitations were too severe to rely upon the findings.

A recent carefully controlled experiment on the rate by which subjects accumulated loss of sleep was carried out by Drake *et al*.[18] As expected, one night of total sleep deprivation was found to produce a significant decline of neurobehavioral performance capability. Subjects whose time in bed was restricted to 4 h per night or 6 h per night for several nights displayed neurobehavioral performance declines as well. These decrements were less substantial than after total sleep deprivation, but were greater after 2 days with 4 h in bed per night than after 4 nights with 6 h in bed per night. It was argued that each of these three conditions constituted a total of 8 h of sleep loss. Because the 6 h time-in-bed condition showed less neurobehavioral impairment, it was concluded that a compensatory adaptive mechanism operated during slow accumulation of sleep debt.

Aside from the fact that sleep duration was equated to time in bed rather than polysomnographically measured total sleep time, this interpretation is dependent upon two critical assumptions: (i) that sleep loss accumulates in a linear, additive manner; and (ii) that basal sleep need is satisfied with 8 h time in bed for sleep.

BASAL SLEEP NEED

Sleep debt implies some fundamental duration of sleep below which waking deficits begin to accumulate. While Horne's arguments for core sleep place this value below that of typical nocturnal sleep durations,[15,16] the concept of basal sleep need suggests this threshold is much closer to 8 h on average. Although not clearly defined in the literature, the construct of basal sleep need has been operationalized as habitual sleep duration in the absence of pre-existing sleep debt (compare[2], pp. 68–70). The basal need for sleep has been argued to be, on average, approximately 8 h per day in healthy adult humans, based on at least one study in which prior sleep debt was completely eliminated through repeated nights of long duration sleep.[19]

From the perspective of basal sleep need, sleep debt is easily accumulated as a result of even modest reductions of sleep duration. In fact, the only critical difference between conceptualizations of sleep debt predicated on core sleep versus basal sleep need concerns the threshold of sleep duration at which

cumulative deficits develop. Both theoretical perspectives acknowledge considerable interindividual differences in sleep duration (core and/or basal need).

Among the most extensive and provocative data on sleep need are epidemiological studies showing high proportions of adults with self-reported sleep durations of less than 8 h per night.[20–22] For example, Kripke *et al.* recently reported that approximately 20% of more than 1.1 million Americans indicated they slept 6.5 h or less each night.[22] In a recent poll of 1000 American adults by the National Sleep Foundation, 15% of subjects reported sleeping less than 6 h on weekdays and 10% reported sleeping less than 6 h on weekends over the past year.[23]

A most interesting result in the large epidemiological study by Kripke *et al.* was the provocative finding that an elevated risk of mortality was found both for those who reported sleeping less than 6.5 h per night and those who reported more than 7.4 h per night.[22] This means that the modal group reporting sleep durations of 7.5–8.4 h per night had a higher mortality risk than those reporting 6.5–7.4 h per night. The reasons for the elevated risks below 6.5 h and above 7.4 h remain unclear, as does the reliability of self-reported data to reflect either basal or core sleep need.

SLEEP DEBT AND THE LIMITATIONS OF WAKEFULNESS

One theme that characterizes all of the constructs of core sleep, optional sleep, basal sleep need, and adaptation to sleep restriction is the emphasis on duration of quality sleep. In contrast to sleep duration, little attention has been paid to wakefulness as a final determinant of sleep debt. For example, Horne dismisses the relevance of wake duration when he writes, 'There is no reason to assume that there are only enough spare circuits to keep us going for the 16 h of one waking day, and there is probably provision for a reserve capacity' (p. 54).[16] However, the two-process model of sleep regulation[17,24,25] explicitly posits that the sleep homeostatic drive waxes and wanes as a function of prior wakefulness.

Over the past 5 years, we have been systematically addressing some of the empirical and theoretical questions about sleep debt raised above. In a series of tightly controlled and highly ambitious experiments, we documented the dose-response profiles of cumulative neurobehavioral consequences from sleep debt, their precise time-course, their relationship to sleep homeostasis, and the role of individual differences in their expression. In the sections that follow, we provide some of the results from these studies, and in doing so, illustrate the need for more thorough and quantitatively sophisticated approaches to the study of sleep debt.

EXPERIMENT 1

In a recent experiment we conducted, $n = 36$ healthy subjects spent 20 days inside a laboratory undergoing a strict schedule of performance testing and restricted sleep. After three baseline days with 8 h time in bed (23:30–07:30 hours) subjects underwent sleep restriction for 14 days. Time in bed (TIB) was either 4 h (03:30–07:30 hours; $n = 13$), 6 h (01:30–07:30 hours; $n = 13$) or 8 h (23:30–07:30 hours; $n = 9$). Neurobehavioral performance was tested every 2 h during wakefulness, and included a 10-min psychomotor vigilance test (PVT).[26] Daily averages (09:30–23:30 hours) were computed for PVT lapses (reaction times >500 msec).

Digital polysomnographic records (sampled at 128 Hz) were made on two baseline, and 10 of the 14 restricted nights (complete data were available for $n = 9$ subjects in the 4-h TIB condition, $n = 7$ subjects in the 6-h TIB condition, and $n = 7$ subjects in the 8-h TIB condition). The polysomnographic records were visually scored in 30-s epochs using conventional criteria.[27] Average sleep efficiencies were found to be 97% in the 4-h TIB condition, 91% in the 6-h TIB condition, and 84% in the 8-h TIB condition.

Waking EEG (C3–A1/A2 derivation) was digitally recorded every 2 h on two baseline and 10 of the 14 restriction days, during a Karolinska Drowsiness Test (KDT) involving 5 min of subjects staring at a dot (data were available for $n = 6$ subjects in the 4-h TIB condition, $n = 6$ subjects in the 6-h TIB condition, and $n = 6$ subjects in the 8-h TIB condition). Data from the KDT bout at 11:45 hours were subjected to power spectral analysis in 2-s bins following visual artifact rejection. The 5-min average powers in the theta (4–8 Hz) and alpha (8–12 Hz) frequency bands were then computed as objective markers of sleepiness.[28]

To analyze the daily averages of PVT performance lapses (expressed relative to baseline), we performed repeated-measure analysis of variance (ANOVA) over the 14 days of sleep restriction with condition as between-subjects factor. There were significant (Huynh–Feldt corrected) effects of day ($F_{13,416} = 22.5$; $P < 0.001$) and day by condition interaction ($F_{26,416} = 2.40$, $P = 0.035$; Fig. 1a). These results reflected a sleep-dose-dependent progressive increase of neurobehavioral performance deficits over the 14 days of sleep restriction. Cumulative

changes in psychomotor performance capability were greatest in the 4-h TIB condition and smallest in the 8-h TIB condition, with the 6-h TIB condition in between. Cumulative impairment in the 8-h TIB condition was marginal even after 14 days, but significantly different from zero ($F_{13,104}=5.73$, $P=0.013$).

For waking EEG theta power, repeated-measure ANOVA yielded a significant effect of day ($F_{8,120}=3.19$; $P=0.003$). For alpha power, the effect of day was not significant ($F_{8,120}=2.75$; $P=0.10$). Neither theta nor alpha power showed any effect of condition or day by condition interaction. Thus, the waking EEG did not show sleep-dose-dependent progressive increases over the 14 days of sleep restriction (Fig. 1b).

Assuming that alpha and theta activity in the waking EEG continued to be objective indicators of sleepiness during our study of chronic sleep restriction, this finding suggests that some form of adaptation to chronic sleep loss occurred, so that the response to the experiment was comparable for each of the three conditions. This contradicts the finding for neurobehavioral performance, however, which indicated that chronic sleep loss leads to cumulative, dose-related increases in neurobehavioral deficits without evidence of adaptation. It would seem, therefore, that sleep debt can lead to fundamentally different responses depending on the marker of waking function.

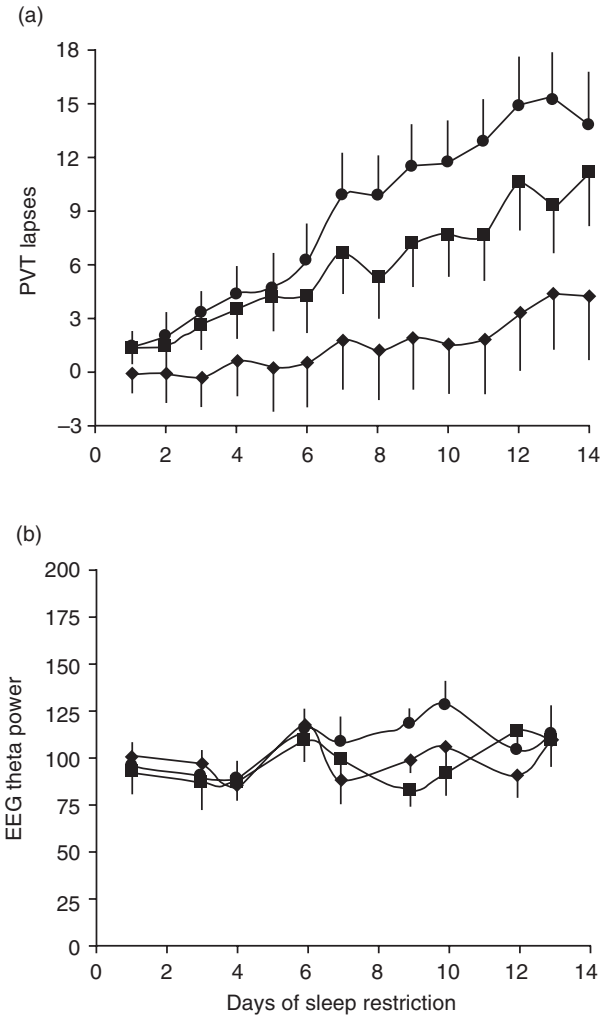

Figure 1 Changes in neurobehavioral function (average psychomotor vigilance test (PVT) lapses per test bout, relative to baseline) and waking electroencephalogram (EEG) theta power (percentage of baseline) over 14 days of sleep restriction. Group means and standard errors are shown for the 4-h time in bed condition (●), the 6-h time in bed condition (■), and the 8-h time in bed condition (◆), in experiment 1. Chronic partial sleep deprivation led to cumulative, sleep-dose-dependent increases in neurobehavioral impairment, but not in waking EEG theta power.

MODELING THE TEMPORAL DYNAMICS OF NEUROBEHAVIORAL FUNCTIONING

To understand the temporal profile of neurobehavioral functioning from a regulation point of view, variations of the two-process model of sleep regulation have been used.[17,29] This model was designed to predict the timing and duration of sleep, and consists of a homeostatic process (process S) and a circadian process (process C). These two processes combined are used to estimate the timing of the onset and offset of sleep. The homeostatic process represents the drive for sleep that increases progressively during wakefulness, and decreases during sleep (symbolizing the physiological recovery obtained from sleep). Sleep is triggered when the homeostatic drive increases above a certain threshold (unless wakefulness is deliberately maintained). Wakefulness is spontaneously invoked when the homeostatic drive has decreased sufficiently during sleep to fall below another threshold. The circadian process represents the daily oscillatory component in the drive for sleep and wakefulness, which is modeled as circadian variation in the threshold values.

The homeostatic and circadian components of the two-process model can be used to predict waking neurobehavioral alertness.[24,30,31] When focusing on daily averages, as in experiment 1, the circadian process cancels out of the equations (in first-order approximation), and day-to-day changes in homeostatic pressure are

exposed. The two-process model predicts that chronic partial sleep deprivation to 4, 6 or 8 h in bed per day results in acute sleep-dose-related increases in waking homeostatic pressure. Within a few days, however, the average predicted waking homeostatic pressure stabilizes (compare[32]) at approximately 133% of baseline in the 4-h TIB condition and 112% of baseline in the 6-h TIB condition, while staying at 100% in the 8-h TIB condition. This suggests homeostatic adaptation to chronic sleep restriction.

The homeostatic process can be tracked during wakefulness by theta power in the EEG.[33] We analyzed the difference between predicted theta power as a marker of homeostatic pressure and observed theta power in experiment 1, both expressed relative to baseline for each individual, using repeated-measure ANOVA over days of sleep restriction with the sleep restriction condition as between-subjects factor. No statistically significant effects were found, indicating that observed theta power did not contradict the predictions in any of the three conditions.

We also examined whether the homeostatic process of the two-process model of sleep regulation would predict neurobehavioral function. For this, we analyzed the difference between predicted homeostatic pressure and observed PVT performance lapses in experiment 1, both expressed relative to baseline for each individual. Repeated-measure ANOVA (without intercept) revealed a significant effect of condition ($F_{3,31} = 6.11$, $P = 0.002$) and a significant interaction of days by condition ($F_{39,403} = 3.61$, $P = 0.024$). These results reflected substantial sleep-dose-dependent differences, increasing across days, between the observations for neurobehavioral function and the two-process model predictions for process S. This confirmed our earlier observation that sleep debt can lead to different responses depending on the marker of waking function. In addition, it showed that neurobehavioral performance capability is not predicted by the two-process model of sleep regulation under conditions of chronic sleep restriction.

The concept of sleep homeostasis was experimentally substantiated by experiments involving acute total sleep deprivation followed by recovery sleep.[17] The difference between total sleep deprivation and chronic sleep restriction is that the latter involves repeated perturbation of the sleep–wake regulatory systems, while acute total sleep deprivation is only a one-time disturbance of sleep and wakefulness. We hypothesize that the repeated perturbations associated with chronic partial sleep deprivation expose further regulatory mechanisms of waking function in addition to sleep homeostasis. In terms of the two-process model of sleep regulation, there appears to be an unidentified third process affecting waking behavioral alertness across days of chronic partial sleep deprivation.

INTERINDIVIDUAL DIFFERENCES IN SLEEP NEED AND VULNERABILITY TO SLEEP LOSS

If the two-process model of sleep regulation cannot accurately predict neurobehavioral performance across multiple days of sleep restriction, it should be possible to formulate an alternative model that predicts the observations more closely. Clues for what such a model might look like may be found in empirical evidence about parameters that would be expected to affect the model's predictions.

First, the predicted magnitude of performance decline across days of sleep restriction should depend on the amount of sleep needed per day to maintain optimal waking function ('sleep need'). There is natural variability in sleep need, as has been widely reported in the literature.[22] Interindividual differences in sleep need have also been studied experimentally. By using the waking EEG as a physiological marker of sleep homeostasis, it was found that naturally short sleepers tolerate a higher homeostatic pressure for sleep than naturally long sleepers.[34] There may be a genetic basis for this variability in natural sleep need.[35,36] Not everybody who reports to be a naturally short sleeper actually is one though.[37] Many can sustain living on a short sleep schedule for a while but eventually accumulate a sleep debt. Typically, this results in the need to extend sleep on the weekend.

Another source of natural variability is 'vulnerability to sleep loss'; that is, the magnitude of performance impairment given a fixed amount of sleep loss. Differential vulnerability to sleep loss is a concept developed by our laboratory based on experimental evidence. It is illustrated below in experiment 2.

EXPERIMENT 2

In a recent study in our laboratory, $n = 15$ healthy subjects each completed two laboratory-based exposures to sleep deprivation, at intervals of 2–4 weeks. The study protocols for the two sessions were identical, and involved 36 h (from 10:00 until 22:00 hours the next day) of behaviorally monitored total sleep deprivation in a controlled, isolated laboratory environment. Ample time was reserved for sleep in the 7 days prior to each

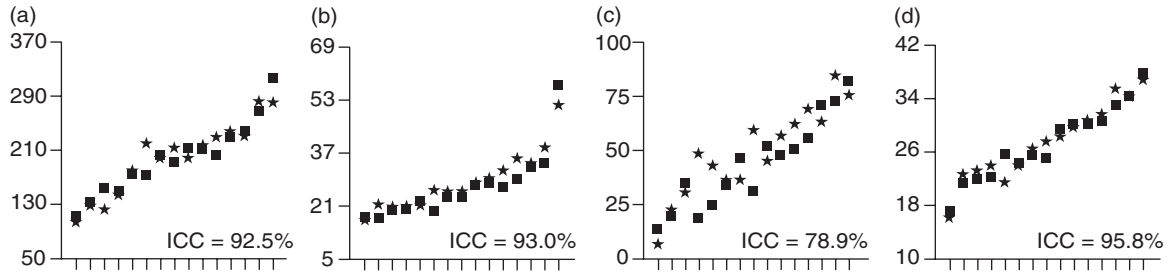

Figure 2 Response to total sleep deprivation during the first exposure (■) versus the second exposure (★) in experiment 2, for each subject (tic marks on the abscissa), on different neurobehavioral performance measures. Upwards means more impairment for the critical tracking task (CTT) and psychomotor vigilance test (PVT), and less impairment for the digit–symbol substitution task (DSST) and word detection task (WDT). In each graph, subjects are rank-ordered on the basis of average performance across the two experimental sessions. Trait-like interindividual differences in this experiment were substantial, as quantified with the intra-class correlation (ICC), which ranged from 78.9 to 95.8%. (a) DSST # correct; (b) CTT control failures; (c) PVT lapses; (d) WDT # correct.

of the sleep deprivation sessions (12 h time in bed per day), and the last night before sleep deprivation was spent inside the laboratory.

Every 2 h, subjects were tested on a neurobehavioral performance battery, which included a digit–symbol substitution task (DSST), a critical tracking task (CTT), a word detection task (WDT) and a psychomotor vigilance test (PVT). The neurobehavioral outcome measures (number correct for the DSST and the WDT, number of control failures for the CTT, and number of lapses for the PVT) were averaged over the last 24 h of each 36-h total sleep deprivation period. The intraclass correlation (ICC) was computed over these averages in order to express between-subjects variance as a fraction of total variance in performance deficits from sleep loss. The ICC thus quantified trait variance for vulnerability to sleep loss. For each of the four neurobehavioral performance measures, the ICC was found to be significantly greater than zero ($F_{14,14} > 9.4$, $P < 0.001$). This means that there were substantial trait-like interindividual differences in vulnerability to sleep loss (Fig. 2).

A MIXED-EFFECTS MODEL OF SLEEP DEBT

Based on the idea that at least 'sleep need' and 'vulnerability to sleep loss' should affect the build up of performance impairment during chronic sleep restriction, we set out to formulate a model to predict the neurobehavioral deficits observed in experiment 1. In particular, we evaluated an additive model of sleep debt; that is, we essentially made the same assumption of linearity as Drake *et al.*[18] did. Thus, we postulated that each hour of sleep loss is equally important in determining the performance deficits resulting from chronic sleep restriction for a given individual. Interindividual variability in 'sleep need' and 'vulnerability to sleep loss' was explicitly included in the model. Furthermore, we made no assumption about the 'sleep need' population average.

The linear model of sleep debt, applied to PVT performance deficits, was formulated as:

$$\mathrm{IMP}_D \sim \alpha\,(\lambda\, D - \mathrm{CTST}_D),$$

where D is the day of sleep restriction (1–14); CTST_D is cumulative, polysomnographically determined total sleep time (in hours) on day D; IMP_D is neurobehavioral impairment (in PVT lapses) on day D; α is vulnerability to sleep loss (in PVT lapses per hour of sleep loss); and λ is sleep need (in hours). Between-subjects variability for α and λ was incorporated in the model as random effects for these parameters, assuming a bivariate normal distribution. The model was fit to the data using mixed-effects regression, for which we applied the SAS procedure, NLMIXED.[38]

It was found that the model had good predictive potential – the residual error variance was only 17.4% of the overall variance in the data. Thus, 82.6% of the variance was explainable by the model when interindividual differences in 'sleep need' and 'vulnerability to sleep loss' were taken into account. Without these random effects, the explained variance dropped dramatically to 21.9%. Considerable variability in both 'sleep need' as well as 'vulnerability to sleep loss' contributed to the additional variance explained when interindividual differences were incorporated in the model. These results highlight the importance of interindividual dif-

ferences in modeling the effects of cumulative sleep debt.[39]

CONCLUSION

Parameter estimation for the mixed-effects model of sleep debt revealed that the estimated sleep need in experiment 1 was 8.2 h per day. The estimated standard deviation for interindividual differences in daily sleep need was 2.6 h. These values may provide reference for defining sleep debt. Our postulation that each hour of sleep loss is equally important in determining the performance deficits resulting from chronic partial sleep deprivation was substantiated by the excellent goodness-of-fit of the linear model describing the empirical data. Thus, under conditions of chronic sleep restriction, sleep debt may be defined as the cumulative hours of sleep loss with respect to the subject-specific daily need for sleep.

ACKNOWLEDGMENTS

The authors wish to acknowledge the contributions of James Crabbe, Greg Maislin, Anaïs de Brunier and Maurice Baynard. The research was supported by NIH grants NR04281, HL70154 and RR00040, AFOSR grants F49620-95-1-0388 and F49620-00-1-0266, NASA grant NAG9-1161, and NASA cooperative agreement NCC 9–58 with the National Space Biomedical Research Institute.

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