

ON THE RELATIONSHIP BETWEEN INTER-INDIVIDUAL DIFFERENCES IN PERFORMANCE IMPAIRMENT FROM SLEEP LOSS AND INTER-INDIVIDUAL DIFFERENCES IN SLEEP ARCHITECTURE

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Introduction

Sleep deprivation studies have shown that sleep loss adversely affects waking performance (e.g., Dinges & Kribbs, 1991), and recovery sleep reverses this effect in a dose-response manner (Jewett et al., 1999). Recent experiments have demonstrated that there are considerable trait-like inter-individual differences in neurobehavioral performance impairment from sleep deprivation (Leproult et al, 2003; Van Dongen et al., 2003) as well as in sleep architecture (Blaauw et al., 2002). Thus far, no published studies have investigated whether inter-individual differences in the duration and/or structure of sleep are systematically related to inter-individual differences in waking performance during sleep deprivation, and vice versa. In this study, the relationship between inter-individual differences in psychomotor vigilance performance during total sleep deprivation and inter-individual differences in the architecture of the preceding (baseline) and subsequent (recovery) sleep periods was investigated.

Methods

A total of n=21 subjects (9 females, 12 males) aged 21–38 participated in the study. They were physically and psychologically healthy, were neither extreme morning-types nor extreme evening-types, and reported habitually sleeping between 6.5 and 8.5 hours per day. As part of a larger study, each subject underwent two identical laboratory sleep deprivation sessions separated by a minimum interval of two weeks. Each laboratory session began with a 12-hour baseline sleep opportunity (22:00–10:00). Then there was a 36-hour sleep deprivation period during which wakefulness was continuously monitored. This was followed by a 12-hour recovery sleep period (22:00–10:00). These laboratory sessions took place at the General Clinical Research Center (GCRC) in the Hospital of the University of Pennsylvania. Light exposure (<50 lux during scheduled wakefulness; <1 lux during scheduled sleep) and ambient temperature (~21°C) were kept constant. Metabolically controlled, BMI-adjusted meals were served every 4 hours. Caffeine, alcohol and nicotine were not allowed; water was available ad libitum. In the week preceding each of the laboratory sessions, subjects were asked to stay in bed for 12 hours (22:00–10:00) each day in order to satiate their need for sleep.

During the sleep deprivation periods, 1-hour neurobehavioral performance tests were scheduled every 2 hours. The neurobehavioral test battery included a 20-minute psychomotor vigilance task (PVT; Dinges & Powell, 1985). From this task, the standard deviation of reaction times (“SD-RT”), the mean of the 10% slowest inverted

reaction times (“10% slowest”), the total time of lapses (“lapse time”), and the number of false starts (“false starts”) were extracted, among other variables. To assess performance impairment, the average over the last 24 hours of sleep deprivation was computed for these PVT outcome variables. Between test bouts, subjects were free to engage in non-vigorous activities like reading or watching television, but they abstained from exercise.

The baseline sleep period preceding the 36 hours of sleep deprivation, and the recovery sleep period that followed it, were recorded polysomnographically (Vitaport III recorder, Temec Instruments, The Netherlands). Electrodes were positioned at F_z, C₄, C₃ and O_z, referenced to A₁–A₂ and grounded at C_z. The recordings were scored manually using conventional criteria (Rechtschaffen and Kales, 1968), and commonly used sleep variables (TST, S1, S2, S3, S4, REM, WASO, SE, SL) were computed. Complete data were available for n=18 subjects.

We used the intraclass correlation coefficient (ICC), which expresses between-subjects variance as a fraction of total variance, to quantifying trait-like inter-individual differences (on a scale from 0 to 1). Using mixed-model analysis of variance (ANOVA) and controlling for order effects, the ICC was computed for each PVT outcome variable and each sleep architecture variable (baseline as well as recovery). For all PVT variables and all sleep variables with statistically significant ICC values, the partial correlation between the subject-specific PVT outcome variables and the subject-specific sleep variables was then computed, controlling for sleep deprivation session and for order effects. This served to assess whether inter-individual differences in baseline and recovery sleep variables were systematically related to inter-individual differences in PVT performance during sleep deprivation.

Results

All PVT outcome variables presently investigated had ICC values greater than 0.7, representing substantial trait-like inter-individual variability (Landis & Koch, 1977) in vulnerability to performance impairment from sleep loss. Although statistically significant ICC values were also observed for various baseline and recovery sleep variables, these were consistently lower than those for neurobehavioral performance. Here we consider only sleep variables with ICC values greater than 0.5, representing at least a moderate degree of trait-like inter-individual variability (Landis & Koch, 1977). For all of these, the correlation between baseline and recovery sleep was significantly positive ($p < 0.05$). As shown in table 1, however, we observed only moderate correlations between psychomotor vigilance performance during sleep deprivation and the architecture of baseline and recovery sleep. In these assessments, no attempt was made to account for the intrinsic correlation among different PVT variables and among different sleep variables.

Discussion

The relationship between inter-individual differences in sleep architecture and inter-individual differences in psychomotor vigilance performance during sleep deprivation was modest. Subjects with the greatest performance deficits during sleep deprivation showed the greatest amounts of stage 1 and 2 sleep and the smallest amounts of stage 4 sleep and WASO during recovery sleep. In other words, those subjects who were more impaired during sleep deprivation also showed more consolidated recovery sleep, although that sleep appeared to be less intense. Subjects who were more impaired during sleep deprivation also showed greater sleep efficiency and smaller amounts of WASO during baseline sleep. Thus, inter-individual differences in sleep

architecture did not show an obvious causal or reciprocal relationship with inter-individual differences in performance deficits during sleep deprivation. Rather, it may be hypothesized that some individuals had a higher demand for sleep and at the same time a greater vulnerability to performance impairment from sleep loss. This may reflect inter-individual variability in a common homeostatic drive underlying both sleep regulation and waking neurobehavioral performance capability. Whether this would be the same phenomenon as the inter-individual differences in tolerance for homeostatic pressure documented for short and long sleepers (Aeschbach et al., 1996), remains to be investigated.

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Table 1: Partial correlations between sleep variables (all with ICC>0.5), in baseline (B) and recovery (R) sleep, and psychomotor vigilance performance variables (all with ICC>0.7), controlling for sleep deprivation session and for order effects. In all cases, positive correlations indicate that greater values for the sleep variables corresponded with greater performance impairment during sleep deprivation. The table shows only statistically significant partial correlations (p<0.05).

		SD-RT	10% slowest	lapse time	false starts
S1	B				
	R	0.356	0.403		
S2	B				
	R	0.446		0.387	0.387
S3	B				
	R				
S4	B				
	R	-0.365			-0.500
WASO	B	-0.404	-0.487	-0.439	
	R	-0.377	-0.452	-0.360	
SE	B	0.371	0.408	0.386	
	R				

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