Systematic individual differences in sleep homeostatic and circadian rhythm contributions to neurobehavioral impairment during sleep deprivation

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A B S T R A C T

Individual differences in vulnerability to neurobehavioral performance impairment during sleep deprivation are considerable and represent a neurobiological trait. Genetic polymorphisms reported to be predictors have suggested the involvement of the homeostatic and circadian processes of sleep regulation in determining this trait. We applied mathematical and statistical modeling of these two processes to psychomotor vigilance performance and sleep physiological data from a laboratory study of repeated exposure to 36 h of total sleep deprivation in 9 healthy young adults. This served to quantify the respective contributions of individual differences in the two processes to the magnitudes of participants' individual vulnerabilities to sleep deprivation. For the homeostatic process, the standard deviation for individual differences was found to be about 60% as expressed relative to its group-average contribution to neurobehavioral performance impairment. The same was found for the circadian process. Across the span of the total sleep deprivation period, the group-average effect of the homeostatic process was twice as big as that of the circadian process. In absolute terms, therefore, the impact of the individual differences in the homeostatic process was twice as large as the impact of the individual differences in the circadian process in this study. These modeling results indicated that individualized applications of mathematical models predicting performance on the basis of a homeostatic and a circadian process should account for individual differences in both processes.

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1. Introduction

In recent years it has become widely recognized that there are trait individual differences in vulnerability to performance impairment due to sleep deprivation (Van Dongen et al., 2004). It has been suggested that such trait vulnerability may at least partially be explained by habitual sleep restriction (Rupp et al., 2009), but no correlation has been found between individual differences in vulnerability to sleep deprivation and self-reported habitual sleep duration (Van Dongen et al., 2004). Furthermore, experimental manipulation of prior sleep ration only marginally affects the expression of the trait (Van Dongen et al., 2004). Recent reports of genetic predictors of vulnerability to sleep deprivation cast further doubt on a mere behavioral explanation of the trait (King et al., 2009), suggesting instead that it may be fundamentally neurobiological in nature. Genetic polymorphisms identified as candidate predictors of vulnerability to sleep deprivation (Goel et al., 2010; Rétey et al., 2006; Viola et al., 2007) are believed to be associated with the sleep homeostatic and circadian regulation of sleep. This suggests involvement of the homeostatic and circadian processes in determining trait vulnerability to performance impairment during sleep deprivation, and raises interest in quantifying the respective contributions of individual differences in these two underlying processes (Van Dongen, 2006).

The laboratory study that first established the trait-like nature of vulnerability to sleep loss (Van Dongen et al., 2004) involved repeated exposure to 36 h of total sleep deprivation. A neurobehavioral test battery was administered every 2 h, and impairment was assessed by averaging performance measurements across the test bouts in the final 24 h (i.e., one circadian cycle) of each sleep deprivation period. This yielded multiple assessments of vulnerability to performance impairment during sleep deprivation per subject, which is essential for the disentanglement of systematic between-subjects variance from within-subjects variance and measurement noise. By averaging the data within each sleep deprivation period, however, the sleep homeostatic and circadian rhythm processes driving performance deficits (Van Dongen and Dinges, 2005) remained intertwined. Therefore, questions about the relative contributions of the two processes to trait vulnerability

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