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A unified mathematical model to quantify performance impairment for both chronic sleep restriction and total sleep deprivation ☆

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HIGHLIGHTS

- We developed a model of neurocognitive performance that incorporates sleep debt.
- Our model unifies total sleep deprivation and chronic sleep restriction scenarios.
- Our model captures the slower recovery process after chronic sleep restriction.
- Our model describes the beneficial effects of banking sleep on performance.

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ABSTRACT

Performance prediction models based on the classical two-process model of sleep regulation are reasonably effective at predicting alertness and neurocognitive performance during total sleep deprivation (TSD). However, during sleep restriction (partial sleep loss) performance predictions based on such models have been found to be less accurate. Because most modern operational environments are predominantly characterized by chronic sleep restriction (CSR) rather than by episodic TSD, the practical utility of this class of models has been limited.

To better quantify performance during both CSR and TSD, we developed a unified mathematical model that incorporates extant sleep debt as a function of a known sleep/wake history, with recent history exerting greater influence. This incorporation of sleep/wake history into the classical two-process model captures an individual's capacity to recover during sleep as a function of sleep debt and naturally bridges the continuum from CSR to TSD by reducing to the classical two-process model in the case of TSD. We validated the proposed unified model using psychomotor vigilance task data from three prior studies involving TSD, CSR, and sleep extension. We compared and contrasted the fits, within-study predictions, and across-study predictions from the unified model against predictions generated by two previously published models, and found that the unified model more accurately represented multiple experimental studies and consistently predicted sleep restriction scenarios better than the existing models. In addition, we found that the model parameters obtained by fitting TSD data could be used to predict performance in other sleep restriction scenarios for the same study populations, and vice versa. Furthermore, this model better accounted for the relatively slow recovery process that is known to characterize CSR, as well as the enhanced performance that has been shown to result from sleep banking.

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1. Introduction

Sleepiness increases the risk of human error and accidents. It also affects the health, safety, and quality of life of military and civilian personnel who are regularly exposed to work schedules that preclude adequate daily sleep duration and timing (Mallis et al., 2004). Critical to effective management of operational alertness and performance is the ability to accurately predict the impact of various work/rest schedules on individual operators. In this paper, we consider the problem of predicting the alertness

Abbreviations: CSR, chronic sleep restriction; EV, explained variance; PVT, psychomotor vigilance test; R^2, adjusted coefficient of determination; RMSE, root mean squared error; RRT, reciprocal response time; RT, response time; RTD, response time divergence; SWT, scheduled wake time; TIB, time in bed; TSD, total sleep deprivation

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and performance of a population for a set sleep/wake schedule. Biomathematical modeling provides the most promising strategy for addressing the problem of helping manage alertness and neurocognitive performance in operational environments (Friedl et al., 2004), thereby enhancing the safety and productivity of both military and civilian operators.

Borbély's seminal two-process model, originally developed to describe the mechanisms mediating sleep regulation (Borbély, 1982), has also served as the basis of many models used to predict human alertness and neurocognitive performance during sleep loss (Mallis et al., 2004). A basic postulate of this model is that alertness and performance are modulated by the additive interaction of two processes. The first, process *S*, is the sleep homeostat, responsible for increasing sleep propensity during waking, and reducing sleep propensity as recovery occurs during sleep. The fluctuations of *S* are described by exponential functions with fixed upper and lower asymptotes. The second process is the endogenous circadian rhythm, process *C*, which is driven by the internal clock residing in the suprachiasmatic nuclei of the anterior hypothalamus (Daan et al., 1984). This phenomenological model, based on findings from acute total sleep deprivation (TSD) studies, has been extended beyond its original goal of predicting slow-wave activity as a function of sleep/wake history, and now also provides a theoretical framework for quantifying the effects of sleep deprivation on objective and subjective alertness and neurocognitive performance.

More recently, several groups have investigated performance degradation resulting from different levels of sleep restriction (Belenky et al., 2003; Van Dongen et al., 2003; Rupp et al., 2009, 2012; Carskadon and Dement, 1981; Dinges et al., 1997). In contrast to TSD studies, results from these well-controlled chronic sleep restriction (CSR) studies have shown that models of neurocognitive performance based solely on Borbély's two-process model fail to accurately predict the observed degradation (Van Dongen et al., 2003; Carskadon and Dement, 1981; Dinges et al., 1997; Mollicone et al., 2010). It has also been observed that the rate of neurocognitive performance recovery after CSR is considerably slower than the rate of recovery after acute TSD (Belenky et al., 2003; Johnson et al., 2004) and that this class of models does not accurately capture this difference (Johnson et al., 2004).

New performance prediction models have been proposed to explain these observations. Van Dongen et al. (2003) described the effects of sleep restriction in terms of "excess wakefulness" or cumulative wake-time extensions rather than as a homeostatic process, whereas Johnson et al. (2004) and Hursh et al. (2004) introduced a "slow" process modulating the homeostat based on sleep/wake history. The latter model provides accurate predictions for aggregate, daily mean performance during seven days of CSR. However, it has not been used to describe performance variations within each day. Avinash et al. (2005) used this slow process to manipulate the upper and lower asymptotes of the sleep homeostat process in the two-process model so that they simultaneously rise during wakefulness and decay during sleep while maintaining a constant, fixed difference between them. A limitation of this approach is the requirement for an *a priori* estimate of the exact value of the fixed difference between the asymptotes, which is likely to vary across different data sets. In addition, they found that although these models accurately fit data collected under CSR, they substantially underestimate performance impairment under TSD Avinash et al. (2005).

McCauley et al. (2009) showed that these approaches belong to a broader class of homeostatic models and incorporated the two-process model and Avinash et al.'s model into a generalized state-space model. This state-space model similarly maintains a constant, fixed difference between the homeostat asymptotes as they rise and fall. In addition, it predicts a bifurcation of the performance trajectory; that is, when daily wakefulness is maintained below a critical threshold, performance tends to stabilize at a deteriorated level, whereas when daily wakefulness is increased beyond this threshold, the model predicts a continuous degradation in performance over time. This predicted bifurcation of the performance trajectory follows a timescale much longer than the duration of their 14-day CSR study on which the model was based and thus has not been experimentally validated. Moreover, the inclusion of seven additional parameters beyond the seven parameters of the classical two-process model (Avinash et al., 2005; Achermann and Borbély, 1994) makes it difficult to estimate the model parameters from limited CSR data. Furthermore, the inherently nonlinear interaction between the homeostatic and circadian processes in this model can place the lower asymptote above actual performance data.

To address these limitations, we developed a model we call the "unified model." Results of recent studies suggest that CSR induces relatively long-term, slow-recovering changes in brain physiology that affect alertness and performance (Belenky et al., 2003; Johnson et al., 2004; Alhola and Polo-Kantola, 2007). We hypothesized that these long-term changes alter the homeostatic process during sleep such that the capacity of an individual to recover during sleep changes as a function of prior sleep/wake history, i.e., as a function of sleep debt. Mathematically, we modeled this hypothesis by allowing the lower asymptote of the classical two-process model to increase or decrease based on the accumulation or restoration of sleep debt, respectively, while keeping the upper asymptote constant. Because the lower asymptote bounds performance impairment from below, constraining the minimum amount of impairment, by modulating the lower asymptote as a function of sleep debt we effectively constrain the rate of performance recovery during sleep. Sleep debt, in turn, is modeled based on a "fading memory" filter, representing the notion that sleep losses or sleep extensions that occurred in the remote past have less effect on the present sleep debt and performance than comparable events in the recent past. Belenky et al. (2003) proposed a similar notion to explain the slow rate of performance recovery after CSR; our work builds on their observations by constructing a mathematical model to describe the phenomenon. A similar notion of fading memory is used in the Fatigue Audit InterDyne (FAID) model developed by Dawson and Fletcher (2001); the fading-memory filter we propose in this paper goes beyond the FAID model in that it also incorporates the possible beneficial effects of sleep banking (Rupp et al., 2009). The idea of fading memory has not been incorporated into Borbély's two-process model (and other closely related neurocognitive performance models), which assume a constant capacity to recover from sleep loss regardless of prior sleep/wake history.

The unified model is so named because it bridges the continuum between CSR and TSD and reduces to Borbély's classical two-process model in the case of total sleep loss. We validated the proposed model using data from three prior studies (Belenky et al., 2003; Van Dongen et al., 2003; Rupp et al., 2012) in which subjects were exposed to TSD as well as different CSR schedules.

2. Methods

2.1. Borbély's two-process model of sleep regulation

Borbély's two-process model (Borbély and Achermann, 1999; Achermann and Borbély, 1992) is based on the interaction of two processes: (1) the homeostatic process *S*, which rises monotonically during wakefulness and declines monotonically during sleep (Daan et al., 1984) and (2) a circadian process *C*, which is a 24-h