

# Tenth International Conference on Managing Fatigue: Abstract for Review

## Accounting for Sleep Inertia in the Differential Equation Framework of a Biomathematical Model of Fatigue

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**Problem:** The biomathematical model of fatigue published by McCauley et al. (2013) predicts performance impairment on the basis of a sleep homeostatic process interacting with a circadian process and an allostatic process that adjusts the homeostatic setpoint as a function of sleep/wake history. Implemented as a set of ordinary differential equations, the model captures the immediate and cumulative effects on performance of total sleep deprivation, sustained sleep restriction, and banking sleep, as well as the non-linear interaction between the homeostatic and circadian processes. We sought to expand the model equations to also account for sleep inertia.

**Method:** The dynamics of waking performance in the model of McCauley et al. (2013) are given by a system of first-order ordinary differential equations, as follows:

$$\frac{dp(t)}{dt} = \alpha_w [p(t) + \beta_w u(t)] + \kappa(t) [c(t) + \mu_w], \quad (1)$$

$$\frac{d\theta(t)}{dt} = \eta \theta(t), \quad (2)$$

$$\frac{d\theta(t)}{dt} = \omega \left( 1 - \frac{\theta(t)}{\xi} \right), \quad (3)$$

where  $p(t)$  is the prediction of performance impairment in terms of lapses on a psychomotor vigilance test (PVT),  $u(t)$  is the allostatic process,  $\kappa(t)$  represents dynamic changes in circadian amplitude,  $c(t)$  is a 24-hour sinusoidal oscillator driving the circadian rhythm, and the remaining mathematical symbols are fixed model parameters. We set out to add sleep inertia to this model as an additional differential equation acting on performance after awakening, without otherwise changing the model dynamics.

**Results:** In the literature, sleep inertia has been described as a transient augmentation of performance impairment, relative to background performance levels, that is associated with awakening and declines exponentially over time awake. To include sleep inertia in the differential equation framework of the model, we formulated an additional differential equation, as follows:

$$\frac{dx(t)}{dt} = \rho x(t), \quad (4)$$

where  $x(t)$  represents the magnitude of the sleep inertia effect, which is assigned a nominal initial value of 1 upon awakening and declines exponentially over time awake with rate constant  $\rho$ .

Simply adding  $x(t)$  as an additional term in Eq. (1) would not preserve the original model dynamics for background performance. Therefore, we designated a new prediction outcome variable for the model,  $f(t)$ , which captures the combined performance effects of the original model per Eq. (1) and sleep inertia per Eq. (5):

$$\frac{df(t)}{dt} = [1 + \gamma x(t)] \frac{dp(t)}{dt} + \gamma p(t) \frac{dx(t)}{dt}, \quad (5)$$

where the magnitude of sleep inertia is posited to be proportional to the background level of performance impairment,  $p(t)$ , and scaled by fixed parameter  $\gamma$ .

**Discussion:** We expanded the fatigue model of McCauley et al. (2013) to include prediction of the performance effect of sleep inertia, which had previously not been accounted for. In the process, we also enhanced the differential equation framework to enable future additions to the model in two distinct categories. Firstly, by adding new terms to Eq. (1), mediators that impact the dynamics of sleep/wake regulation can be incorporated. This was already possible in the original model formulation and has been used by Honn et al. (2016) to account for differential fatigue from low versus high task load. Secondly, by adding new terms to Eq. (5), moderators that influence performance only transiently can be incorporated. That was done here for sleep inertia. Another possible example of a moderator of performance is physical activity.

The enhanced model framework also provides a straightforward means of converting predictions to a different outcome metric. For example, there is an approximately quadratic relationship between lapses on the PVT as used by McCauley et al. (2013) and effectiveness scores as used in the SAFTE model of Hursh et al. (2004). Transforming the outcome metric of the former to that of the latter could be accomplished by extending Eq. (5), as follows:

$$\frac{df(t)}{dt} = [v_1 + 2v_2 + \gamma x(t)] \frac{dp(t)}{dt} + \gamma p(t) \frac{dx(t)}{dt}, \quad (6)$$

where  $v_1$  and  $v_2$  are the coefficients of the linear and quadratic terms of the relationship between the two metrics. Finally, use of the differential equation framework emphasizes the *dynamics* of changes in performance, which is useful for investigating the nature and complexity of the underlying neurobiology.

**Summary:** Predictions of performance impairment by the fatigue model of McCauley et al. (2013) are based on intrinsic interactions between sleep homeostatic, circadian and allostatic processes. Here we expanded the model with an additional process to account for sleep inertia. We enhanced the differential equation framework of the model to differentiate between mediators acting on the fundamental model dynamics and moderators causing transient changes, and added an exponentially declining differential equation for sleep inertia as a moderator taking effect upon awakening. The magnitude of the sleep inertia effect on performance was assumed to

be proportional to the background level of performance impairment, such that sleep inertia is worse after awakening under conditions of sleep insufficiency and circadian misalignment. In further research, measurements of sleep inertia obtained under a variety of homeostatic, circadian and allostatic process states will be needed to estimate and validate the sleep inertia model parameters.