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Date of Publication from Ext Publisher: January 2018

Defence Research and Development Canada
External Literature (P)
DRDC-RDDC-2018-P006
January 2018
IMPORTANT INFORMATIVE STATEMENTS

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To cite this article: Henry T. Peng, Fethi Bouak, Wenbi Wang, Renee Chow & Oshin Vartanian (2018): An improved model to predict performance under mental fatigue, Ergonomics, DOI: 10.1080/00140139.2017.1417641

To link to this article: https://doi.org/10.1080/00140139.2017.1417641
An improved model to predict performance under mental fatigue

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ABSTRACT
Fatigue has become an increasing problem in our modern society. Using MATLAB as a generic modelling tool, a fatigue model was developed based on an existing one and compared with a commercial fatigue software for prediction of cognitive performance under total and partial sleep deprivation. The flexibility of our fatigue model allowed additions of new algorithms and mechanisms for non-sleep factors and countermeasures and thus improved model predictions and usability for both civilian and military applications. This was demonstrated by model simulations of various scenarios and comparison with experimental studies. Our future work will be focused on model validation and integration with other modelling tools.

Practitioner Summary: Mental fatigue affects health, safety and quality of life in our modern society. In this paper, we reported a cognitive fatigue model based on existing models with newly incorporated components taking both the operator’s state of alertness and task demand into account. The model provided the additional capability for prediction of cognitive performance in scenarios involving pharmaceutical countermeasures, different task demands and shift work.

1. Introduction
Mental fatigue has become an increasing problem in our modern society. It often refers to an inability or disinclination to continue an activity that people may experience after or during prolonged periods of wakefulness or cognitive activity (Boksem and Tops 2008). In our context, it is a related concept to sleepiness resulting from the neurobiological process regulating sleep and circadian rhythms (Dawson, Searle, and Paterson 2014). In this paper, fatigue is used interchangeably with sleepiness resulting from sleep deprivation and circadian desynchrony.

There are workers needed around the clock. Nurses, industrial processing plant workers, military trainees and pilots are some occupations that have fluctuating work schedules based on real-time demand. It is widely known that mental fatigue can lead to bad judgement and errors. Research showed that pilot performance can be reduced by more than 40% mostly due to crew scheduling and workload factors (Wilson, Caldwell, and Russell 2007). In occupations with irregular or fluctuating work schedules, incorrect decisions can be fatal as they involve human life and use dangerous, large-scale equipment. Employers must know their employees’ performance levels given a difficult work schedule in order to provide employees with appropriate opportunities to sleep. Still, it is in the interest of all organisations to understand human fatigue and establish reasonable work–rest schedules as fatigue decreases productivity and costs the world billions of dollars annually (Health and Safety Executive 2010). Not only does fatigue reduce effectiveness, it also creates more accidents in our daily lives. Take driving for example, in New York State, US, 40–60% of ‘run-off-road’ crashes are due to driver fatigue or drowsiness (Safeny 2010). In Australia, fatigue driving is responsible for 20% of fatal accidents (Transport Accident Commission 1998). More interestingly, in an experiment done by Dawson and Reid, it was found that performance of drivers after 22 h of wakefulness is equivalent to that of drivers with 0.08% blood alcohol concentration (Dawson and Reid 1997).

Some of common effects of fatigue include slowed reaction time, reduced vigilance, impaired decision-making skills, lack of attention, etc. Sleep loss leads to decreased performance and alertness. It has been demonstrated that short-term (<48 h) sleep deprivation impairs a wide host of cognitive outcome variables including simple attention, complex attention, processing speed, working memory and short-term memory (Lim and Dinges 2010). Moreover, two to three hours less sleep in a single night (acute sleep
loss) produces measurable impairment of tasks in the lab and in the real world (Dawson 2009). Sleep deprivation greater than 24 h have reported results that equal alcohol impairment. Extreme sleep deprivation greater than 40 h can cause severe short-term memory loss, and inability to perform cognitive tasks. Continued acute sleep loss for more than several days will gradually decrease performance capabilities.

The neural effects of sleep loss have been shown to be varied and context-dependent, leading to increases or decreases in brain activity as a function of cognitive and task demands (Chee and Chuah 2008; Dang-Vu et al. 2007; Drummond et al. 2000). However, there is some evidence to suggest that tasks loading on the prefrontal cortex are particularly vulnerable to the impact of sleep loss (Harrison and Horne 2000; Jones and Harrison 2001), including tasks that require revising plans and decisions, overcoming distractions, and effective communication. Cognitively demanding tasks produced greater mood and performance decrements as a function of sleep loss than less-demanding ones (Angus and Heslegrave 1985). In addition to cognitive demands of a task and contextual influences, the fatigue effects on performance depend on operator motivation and engagement (Matthews et al. 2010). Several studies have shown that motivation could be curvilinearly related to performance (Humphreys and Revelle 1984) and engagement could be associated with superior performance on a variety of attentionally demanding tasks (Matthews et al. 2002).

The effects of fatigue are especially severe in military operations since a fraction of second can often result in life or death difference. Sleep-induced mental fatigue has been well recognised as a major factor influencing military’s performance across all operational environments (Army, Navy and Air) (Alfred et al. 2010; Vila and Samuels 2011). Therefore, it is very important for the Canadian Armed Forces (CAF) to predict the effects of fatigue on human performance and find countermeasures to fatigue to ensure its soldiers are at optimal performance when needed.

Various strategies have been studied to mitigate the effect of sleep loss on health and performance. One way to recover from fatigue is to simply get sufficient sleep. However, there are many factors affecting the quality of sleep, and in modern society people tend to sleep less than in the past (Belenky et al. 2003). In military operations, soldiers are unlikely to get adequate sleep every day. Since the amount of available sleeping time is a major constraint, there is an urgent need to find an optimal sleep/awake schedule to achieve high performance. By sleeping at the right time, a soldier will be at peak performance when such a high level of effectiveness is required.

Mathematical models to predict fatigue are valuable tools (Dawson et al. 2011; Gundel, Marsalek, and Ten Thoren 2007; Raslear, Hursh, and Van Dongen 2011; Van Dongen and Belenky 2012). The need for mathematical models to estimate human cognitive performance based on sleep/work schedules has been recognised by unions, employers and government in the recent years (Shahrok-Shahraki and Bakar 2011). Most of these models are based on sleep/wake schedules and validated in aviation and transportation industries (Dawson et al. 2011). Research started in early 1980s to predict people’s performance when prior sleep/awake history is known. The work on predicting human performance is now known as fatigue modelling. Research initiatives have made significant progress in the fatigue modelling. Bio-mathematical modelling of fatigue attempts to present viable estimates of performance for different schedules. They are estimates because it is nearly impossible to model the complex neuro-physiological mechanisms of the brain. Also, due to the individual differences when conducting sleep experiments, the data cannot be considered completely accurate for all. The model is designed using data from large groups of volunteers and the average performance is often used to fit as many participants as possible. Recently, individual differences have been included in fatigue modelling based on current and previous measures of performance of a specific individual to predict that individual’s future performance (Rajaraman et al. 2009; Van Dongen, Bender, and Dinges 2012). The effect of caffeine has been modelled by multiplying the performance in the absence of caffeine with a caffeine-effect factor which was calculated from pharmacokinetic–pharmacodynamic effects (Ramakrishnan et al. 2013). Seasonal or artificial light which can alter human circadian pacemaker has also been considered in some fatigue models (Jewett and Kronauer 1999; Kronauer, Forger, and Jewett 1999). Task characteristics (Neville et al. 2000) including: task duration, feedback, task difficulty, working memory requirements, task pacing and level of proficiency, and level of external stimulation in the task environment such as social interaction (Zhang et al. 2009) and attention stimuli (e.g. noises) (Szalma and Hancock 2011), have not been taken into consideration in fatigue modelling for their effects on human performance.

Today there are a number of bio-mathematical fatigue models available (Dawson et al. 2011; Gundel, Marsalek, and Ten Thoren 2007). Among those, one of the widely used models is Sleep, Activity, Fatigue, and Task Effectiveness (SAFTE) model developed by Hursh et al. (2004, Van Dongen 2004) and commercially available as Fatigue Avoidance Scheduling Tool (FAST Model). Currently, Defence Research and Development Canada (DRDC), Toronto Research Centre is using FAST model as a base to develop a new fatigue model to help the CAF better
predict the performance of its soldiers. Improvements are made to our SAFTE-based model to include task demand, pharmaceutical countermeasures, jet lag, night shift and light countermeasures that are considered as important factors in the development of bio-mathematical models of fatigue and performance (Dinges 2004; Friedl et al. 2004).

Given the importance of countermeasures and workload in sleep and performance models (Balkin et al. 2004; Van Dongen and Balkin 2004), this study is focused on incorporation of pharmaceutical and task effects into our fatigue model. Specifically, a component was added to the model to account for the consumption of stimulating drugs, such as caffeine, which is common in many work situations for many individuals. The model was also expanded to examine the combined effect of fatigue and human mental capacity on performance at different task demands, based on existing mental resource theories, particularly the single resource model proposed by Kahneman (1973).

In addition, in order to make the model more capable to evaluate the effects of work schedules (e.g. shift work and irregular working hours), a sleep prediction function was added based on the structure of sleep, which would allow us to integrate our fatigue model with a Navy crewing analysis tool which generates work schedules based on different crew sizes and missions. Figure 1 illustrates various factors included in our model to predict performance as a result of the combined effects of work/sleep schedules, specific types of task characteristics and pharmaceutical countermeasures of fatigue. Our model-predicted performance refers to an ability to perform simple tasks such as psychomotor vigilance and reaction time, and complex tasks such as memory and logical reasoning.

This report summarises our in-house development of a bio-mathematical model to predict cognitive performance under mental fatigue from an initial base model comparable with the FAST model to additions to our model for pharmaceutical and task demand factors as well as sleep predictions for shift work. Further development and application of our fatigue model are discussed.

2. Methods

2.1. Cognitive fatigue model

We focused on phenomenological models which are based on physiological phenomena at a systematic level, but not all model parameters have particular physiological meanings and the molecular mechanisms behind have only recently been subjected to detailed investigation (Saper et al. 2010). Our base model was developed based on Sleep, Activity, Fatigue, and Task Effectiveness (SAFTE) model which is a typical three-process model with additive combination of homeostatic process, circadian rhythm and sleep inertia (Hursk et al. 2004). However, rather than using a linear recovery during sleep, our model uses an exponential recovery during sleep. The exponential recovery is in accordance with an exponential characteristic pattern of elevated slow-wave sleep from EEG studies (Achermann 2004). The equations and parameters of the model are as follows:

\[
R_t = R_{t-1} - Kt \quad \text{during wake}
\]

\[
R_t = 2400(a_s + 1)(1 - \exp(-\Delta t/\tau_d)) + R_{t-1} \exp(-\Delta t/\tau_d) \quad \text{during sleep}
\]

where \( R_t \) is the homeostatic sleep reservoir, \( K \) is the depletion rate of 0.5 units/min, \( t \) is the time since awakening or sleep, \( a_s \) is a conversion factor with a value of 0.235, \( \tau_d \) is the recovery time constant of 4.2 h.

The circadian process tracks circadian rhythm generated by the biological clock inside the brain, which can be influenced by light exposure and other time cues and is close to 24-h rhythms in core body temperature and various hormones, such as melatonin (Dijk and Lockley 2002).

![Conceptual framework for modelling the relationship between various factors and performance.](image-url)
The process is modelled by a function composed of the sum of two cosine waves as below. This is in consistence with experimental studies showing the feeling of sleepiness experienced in the early morning at around 0500 h, and the feeling of alertness experienced in the evening at around 1900 h (Schmidt et al. 2007).

\[ C_t = \cos(2\pi(t - p)/24) + \beta \cos(4\pi(t - p - p')/24) \] (3)

where \( C_t \) is the circadian rhythm, \( p \) is the peak of the 24-h rhythm and has a value of 18 h, \( p' \) is the peak of the 12-h rhythm relative to the 24-h rhythm and has a value of 3 h, and \( \beta \) is a weighting factor with a value of 0.5.

The final process is sleep inertia which is a temporary disturbance in performance that occurs immediately after awakening and lasts for a few hours (Tassi and Muzet 2000). Upon awakening, there is a rapid activation of brain stem and thalamic arousal regions that is not matched with the slow activation of the frontal cortical areas used in cognitive thinking (Balkin et al. 2002). As a result, there is a performance decrease immediately after awakening. The sleep inertia is modelled to have a constant initial effect of a 5% performance decrease followed by an exponential decay over the next two hours:

\[ I_t = -I_{\text{max}} e^{-t/(I_{\text{max}} - I_0)} \] (4)

where \( I_t \) is the sleep inertia at time \( t \) within 2 h after awakening; \( I_{\text{max}} \) is the maximal inertia effect on awakening, set to 5% and \( I_0 \) is the inertia time constant, set to 0.04.

The cognitive performance is then:

\[ E_t = 100 - (100 - E_t(\text{base})) \left( \frac{100 - PB}{100} \right) \] (9)

where PB is the performance boost factor, \( C \) is the plasma concentration of drug, \( k_{50} \) is the concentration that results in half of the maximum performance boost, \( E_t \) is performance, and \( E_t(\text{base}) \) is performance excluding the effect of any drugs. \( k_{50} \) has values of 2.0, 1.1 and 0.06 for caffeine, modafinil and dextroamphetamine, respectively. The maximum performance boost is 100 as this results in an increase in performance from its current level to 100%. These numbers were optimised for a best fit to number of data-sets (Bonnet et al. 1994; Bonnet et al. 1995; Kamimori et al. 2005; Killgore et al. 2008; Wesensten, Killgore, and Balkin 2005). This saturation effect of concentration on performance results in a very small effect of caffeine on performance when performance is already high, such as on a normal sleep schedule. However, when performance is low, such as during sleep deprivation, the effect is much larger and is capable of returning performance (e.g. psychomotor vigilance speed) to baseline levels, and has drug-specific duration of action, as seen in two studies (Killgore et al. 2008; Wesensten, Killgore, and Balkin 2005).

2.1.1. Pharmaceutical countermeasures

The drug plasma concentration was predicted by a two-compartment pharmacokinetic model as follows (Csajka et al. 2005):

\[ \frac{dA_1}{dt} = -k_0 A_1 \] (6)

\[ \frac{dC}{dt} = k_0 \frac{A_1}{V_c} - \frac{CL}{V_c} C \] (7)

where \( A_1 \) is the amount of drug in mg in the absorption from an initial dose, and \( C \) is the concentration of drug in plasma in mg/L. \( CL \) is the plasma clearance in L/min, \( V_c \) is the volume of distribution in L (Csajka et al. 2005). The parameters for these equations were estimated based on the half-lives and time to peak concentration for each of the drugs. The half-lives were 5.3 h (Csajka et al. 2005), 11.0 h, and 12.0 h (Killgore et al. 2008; Wesensten, Killgore, and Balkin 2005) and the time to peak concentrations were 1, 4 and 4 h (Killgore et al. 2008; Wesensten, Killgore, and Balkin 2005) for caffeine, modafinil and dextroamphetamine, respectively. This resulted in \( k_t \) values of 0.064, 0.013 and 0.013 and \( CL \) values of 0.083, 0.067 and 0.058, and \( V_c \) values of 38.6, 66.8, 460, respectively.

2.1.2. Task demand

Task demand in our model denotes how difficult a cognitive task is and how much attention is required for the task. It is modelled as an external fatigue factor similarly to the Fatigue/Risk Index (Folkard, Robertson, and Spencer 2007). Basically, on top of the baseline performance level associated to sleep–wake patterns, we included a task-related component associated with the difficulty of the task and a recovery to the baseline level from rest breaks during work. Mathematically, we introduced a new function to represent a person’s ability to maintain sustained attention which can be depleted at a rate during work periods (Equation (10)) and recovered at a different rate during rest breaks and non-work periods (Equation (11)). The depletion rate is amplified by two factors: task demand and

\[ F_t = \frac{F_t(\text{base})}{B} \] (10)

\[ R_t = \frac{R_t(\text{base})}{B} \] (11)

where \( F_t \) is the fatigue level at time \( t \) and \( F_t(\text{base}) \) is the baseline fatigue level; \( R_t \) is the recovery level at time \( t \) and \( R_t(\text{base}) \) is the baseline recovery level; and \( B \) is a boosting factor that amplifies the depletion and recovery rates.
sleep pressure levels, since studies have shown that performance decreases quicker for higher demanding tasks (Baulk et al. 2007; Gould et al. 2009; Morris and Leung 2006; Richter et al. 2005; Wilson, Caldwell, and Russell 2007), and in more sleep-deprived individuals (Belenky et al. 2003; Jung et al. 2011). We then added a function to the basal cognitive performance in equation (5) to describe a further degradation in performance on tasks requiring different amount of attention.

\[ W_t = W_{t-1} - \left( W_d \Delta t L_c \right) / (100 + R_t) \text{ (decrease),} \]  

\[ W_t = W_{t-1} + W_c \Delta t \text{ (increase),} \]  

\[ E_t = E_{t\text{ (base)}} - (W_c - W_t) / W_c \]

where \( 0 \leq W_t \leq W_c \), \( W_c \) represents the current attention capacity, \( W_c = 75 \) units is the maximum attention capacity, \( W_d = 1.14 \) units/h is the depletion rate, \( W_r = 11 \) units/h is the recovery rate, \( L \) is the rating from zero to three depending on demanding levels ranging from low to very high, \( E_{t\text{ (base)}} \) and \( E_t \) are corresponding cognitive performance in Equations (5) and (12).

It is noteworthy that the attention capacity was introduced in the model to indicate a performance moderator influenced by operators’ mental source. As a first step, a single parameter was used in the current study which corresponds to Kahneman’s single resource model (Kahneman 1973). It is our plan to implement the multiple resource model (Wickens 2008) in the future work.

The depletion and recovery rates were originally based on the Fatigue/Risk Index which states that a break of thirty minutes length is sufficient for complete recovery after six hours of work (Folkard, Robertson, and Spencer 2007). The parameters were then optimised to fit a number of published data-sets for performance on cognitive tasks with various levels of difficulties under shift schedules (Baulk et al. 2009; Gillberg et al. 2003), rest/work schedule (Hayashi, Chikazawa, and Hori 2004) and normal sleep (Lim et al. 2010). The model was then used to predict performance with low and high task demands under total sleep loss (Gould et al. 2009; Wilson, Caldwell, and Russell 2007).

2.2. Sleep prediction model

It is considered that the human arousal system consists of two primary components: a circadian system and a sleep homeostasis system. The circadian system \( C \) oscillates in an approximately 24-h cycle, resulting in variations in sleepiness within a single day. The sleep homeostasis system \( S \), in contrast, produces continuous declines in sleepiness as time awake increases, which then recovers with sleep.

Two sleep models have been investigated: one developed by Achermann and Borbély in Switzerland (Achermann 2004; Borbély and Achermann 1999; Daan, Beersma, and Borbély 1984), the other by Akerstedt’s group in Sweden (Åkerstedt, Axelsson, and Kecklund 2007; Akerstedt and Folkard 1996; Åkerstedt, Folkard, and Portin 2004). We incorporated the first one into our fatigue model in the current study. The equations and parameters are given below as reported (Borbély and Achermann 1999):

\[ S_t = 1 - e^\left(-\frac{t}{\tau}\right) (1 - S_{t-\Delta t}) \text{ during wake} \]

\[ S_t = e^\left(-\frac{t}{\tau}\right) S_{t-\Delta t} \text{ during sleep} \]

where \( S \) is the homeostatic sleep pressure as a function of time \( t \); \( \Delta t \) is the time step; and \( t_d (18.2 \text{ h}) \) and \( t_a (4.2 \text{ h}) \) are time constants for the rise and decay of the homeostatic process during wakefulness and sleep, respectively.

\[ C = A \sum_{k=1}^{S} a_k \sin \frac{2\pi k}{\tau} (t - t_0) \]

\( C \): circadian process independent of sleep and waking; \( A \): amplitude of skewed sine wave (0.12); \( t \): time; \( \tau \): period of \( C \) (i.e. 24 h); \( t_0 \): defines the circadian phase at the beginning of the simulation (8.6 h); \( a_1 = 0.97, a_2 = 0.22, a_3 = 0.07, a_4 = 0.03, a_5 = 0.001 \).

The sleep and wake-up time was determined as follows: when \( S > 0.67 + C \) during rest period, time to sleep; when \( S < 0.17 + C \), time to wake up.

The sleep model was added to the base cognitive fatigue model which can create sleep schedules based on shift work schedules and then predict cognitive performance.

2.3. Model implementation and simulations

All equations in our model were programmed using MATLAB (Version: 7.14, The MathWorks, Inc., MA, USA). Through a Graphical User Interface (GUI) in MATLAB as illustrated in Figure 2, users are able to input sleep date, sleep time, wakeup date and wakeup time in Microsoft Excel (expdata.xsl). Inclusion of a work schedule, administration of a drug dose and selection of task demand are optional inputs. The simulation is run by selecting the required Excel workbook from the directory box and clicking model button on the GUI. The program is available upon request.

Experimental data based on various cognitive tasks were obtained from the literature. Specifically, data point values were digitised from corresponding graphs in the
publication using a Microsoft Excel-based application called Grab It! (https://grabitxp.en.softonic.com/). Since the model outputs performance on a scale from 0 to 100% while most of the experimental data are in a real measurement such as reaction time, to compare with model predictions for mental fatigue or cognitive performance the data were transformed by a linear regression method as reported by van Dongen (2004) as detailed below.

In step one experimental data were plotted against the predicted performance. The model prediction at each time point was matched up with the experimental data at the same time point to give a set of experimental against model data. The line of best fit was taken and the slope (a value) and y intercept (b value) were obtained from the linear line fit. In step two scaled predictions were generated by multiplying slope with predicted performance then plus y intercept values. Mathematically, this can be represented as follows:

Step one:

\[ y_{\text{exp},i} = a \times \text{model predictions} + b \]

Step two:

\[ y_{\text{pred},i} = a \times \text{model predictions} + b \]

The mean square error (MSE) between the prediction \( y_{\text{pred},i} \) and experimental data \( y_{\text{exp},i} \) was calculated as:

\[ \text{MSE} = \frac{1}{n} \sum_{i=1}^{n} (y_{\text{pred},i} - y_{\text{exp},i})^2 \]  

where \( n \) is the number of data points.

In addition, log likelihood \( \text{LL} \) was calculated as follows (Bonate 2006):

\[ \text{LL} = -\frac{n}{2} \ln(2\pi) - \frac{n}{2} \ln(\text{MSE}) - \frac{n}{2} \]

We compared the predictions between FAST and our model by Akaike’s Information Criterion (AIC) which is accepted as one of the most reliable methods for comparing different models (Bonate 2006).

\[ \text{AIC} = -2\text{LL} + 2p \]  

where \( p \) is the number of estimated parameters in each model. The model with the lower AIC is considered better.

The MSE and AIC methods have been well used for the comparison of different bio-mathematical models of fatigue and performance (St. Hilaire et al. 2017; Van Dongen 2004).

The confidence intervals (CI) of the model prediction were determined using the following method as described in Bates et al. (Bates and Watts 2007):

\[ \nu = \left[ \begin{array}{c} \frac{\partial f(x, \hat{\theta})}{\partial \hat{\theta}^T} \end{array} \right] \hat{\theta} \]  

\[ J = \left[ \begin{array}{c} \frac{\partial f}{\partial \hat{\theta}^T} \bigg| x_1 \\ \vdots \\ \frac{\partial f}{\partial \hat{\theta}^T} \bigg| x_n \end{array} \right] \]  

\[ f(x, \hat{\theta}) \pm s \sqrt{\nu^T R_1^{-1} \bigg| t(N - P, \alpha/2) \bigg|} \]  

where \( \nu \) was determined using a first-order Taylor series approximation (also known as derivative vector); \( f \) is the model prediction with a vector of model parameters \( \theta \) at time; \( s \) is the standard deviation of a sample on N-P degrees of freedom; \( \hat{\theta} \) is the vector with the best estimate of the model parameters; \( R_1 \) is a matrix following QR decomposition of the derivative matrix (\( J \)); \( t \) represents t test, \( N \) is
the number of observed responses; $P$ is the number of the parameters; $\alpha$ is a significance level of 0.05.

2.4. One-night sleep deprivation study

We also compared model predictions with our experimental data from a study for the effects of a single night of sleep deprivation on cognitive performance (Vartanian et al. 2014). In the study, 12 healthy volunteers (3 females, 9 males, average age was 31.50 years (SD = 8.38)) were recruited. The participants were instructed to have normal sleep and wear a wrist activity monitor (www.ambulatory-monitoring.com/motionlogger.html) continuously 1 week prior to sleep depression session. They arrived at our laboratory prior to 20:00 on the evening and were instructed not to consume any caffeine, nicotine or alcohol for 24 h prior to and during the experiment. They were not allowed to leave the laboratory during the sleep deprivation session, and were monitored by staff at all times to ensure that they did not fall asleep. They completed the Stanford Sleepiness Scale (SSS) and the Psychomotor Vigilance Task (PVT) hourly between 20:00 and 6:00 (Dinges and Powell 1985).

2.5. Model sensitivity analysis

The sensitivity analysis was performed for several model parameters and sleep input. Among the parameters investigated were $K$, $\alpha$, $\tau_d$, $p$, $\beta$, $p'$, $i$ and $f$. The parameter and sleep input values were varied by $\pm$ 5% of their original values one at a time while the other parameters were kept at the initial values and the MSE with the changed parameter was then calculated (Achermann et al. 1993).

To assess the sensitivity of each model parameter, we computed the relative MSE, defined as the difference in the MSEs with the changed and unchanged parameters or sleep input (i.e. wakeup time prior to sleep deprivation) divided by the MSE with unchanged parameters or sleep input for an 88-h total sleep deprivation scenario (Van Dongen 2004).

3. Results and discussion

Our model was built based on a number of models in the literature and created using MATLAB. Our model has also been expanded in order to incorporate several factors influencing performance. Most of the other models either take a sleep schedule or a work schedule as input in order to predict performance or fatigue (Gundel, Marsalek, and Ten Thoren 2007). The only required input for our model is a sleep schedule. Additionally, the user can choose to input a work schedule or a travel schedule with different task demand levels as well as doses of pharmacological agents at any time. This allows for a robust model capable of predicting performance for a wide variety of real-life and military situations.

The model is also capable of producing error estimates based on user-provided experimental data. This error can also be compared to predictions from another model in order to assess if the prediction is more accurate.

Many additions have been made to the model and many more can be made. However, these changes must be validated by comparison to experimental data to show that they actually improve the model. In some cases, additions were made to the model but a lack of experimental evidence to support these changes prompted their removal. For the original model as well as each addition, a number of experimental data-sets were gathered for comparison.

The effects of sleep deprivation are dependent on task demands (Drummond et al. 2012; Van Dongen, Belenky, and Krueger 2011). Unless specified, we compared our model predictions with the experimental data based on PVT which is reliable and sensitive to sleep loss (Basner and Dinges 2011). The PVT test has become a standard for assessment of the effects of sleep deprivation on cognition (Killgore 2010) and been extensively used to evaluate goodness of fit of various fatigue models (Van Dongen 2004). Although specific tasks vary in cognitive demands, and deficits in cognitive capacity under sleep deprivation would produce different decrements in performance of the tasks it is reasonable to assume that the changes in task performance would be correlated with changes in the underlying cognitive capacity (Hursh and Van Dongen 2011). For example, there is a systematic relationship between model predictions optimised to predict PVT performance, and driving simulator accident data from subjects given varying amounts of daily sleep for a week (Balkin et al. 2000). On the other hand, a study that investigated homeostatic, circadian and sleep inertia influences on higher order cognitive functions of inhibitory control and selective visual attention showed differential modulation of cognitive performance by the sleep–wake regulatory processes (Burke et al. 2015).

3.1. Model simulations: sleep loss

Figure 3 presents the cognitive performance predicted by FAST and our model in comparison with experimental data under total sleep deprivation. There is an agreement between predicted and observed data as well as between FAST and our model. Approximately 66% experimental data were within 95% CI of our model predictions. In addition to graphical comparisons, the mean square error (MSE) was calculated for each model in various experiments. The smaller the mean square error, the better model predictions fit experimental data. As summarised
effects of caffeine on fatigue performance of sleep-deprived individuals (Benitez et al. 2009; Puckeridge et al. 2011; Ramakrishnan et al. 2013). Seng et al. reported a pharmacokinetic–pharmacodynamic model to capture the effects of caffeine on psychomotor functions under only alertness conditions (Seng et al. 2010). Similarly, the ‘Performance Boost’ factor in our model is based on pharmacokinetic–pharmacodynamic Hill relationship for the effects of various drugs on performance restoration. The one reported by (Ramakrishnan et al. 2013) may be the latest model for fatigue performance with caffeine effects. There is no fatigue performance model with other pharmaceutical countermeasures. However, bio-mathematical models have been reported for sleep–wake switch effects (Johnson et al. 2012), circadian phase-shifting effects (Breslow et al. 2013), and pharmacokinetics of exogenous melatonin (Peng et al. 2013).

In our work, we incorporated a two-compartment pharmacokinetic model into our fatigue model to predict plasma drug concentration at different doses of three pharmaceuticals: caffeine, dextroamphetamine and modafinil. Then the drug concentration was used to calculate a performance boost factor and final performance using such a function that gave an increase in performance under sleep deprivation with a saturation effect. Caffeine, dextroamphetamine and modafinil are some of the most commonly used and extensively studied pharmacological countermeasures for sleep loss.

Figure 3 shows a graphical comparison of cognitive performance between the model predictions and experimental data when either placebo or caffeine was used under sleep deprivation. The effects of caffeine on the cognitive performance were well captured by our model in consistence with experimental data which is not the case for FAST. This is explicitly indicated by a much smaller MSE of our model than that of FAST (142.27 vs. 707.06) as calculated from the experiment data with the caffeine effects under sleep loss (Bonnet et al. 1995).

3.2. Model simulations: pharmaceutical countermeasures

Caffeine has been well documented for its effects on fatigue and cognition (Lorist and Tops 2003). It is the pharmacological countermeasure of fatigue widely used in a variety of occupational and non-occupational settings (Ker et al. 2010). Only few models incorporate the

differences.

Table 1. Summary of model-predicted performance with experimental data in different sleep conditions.

<table>
<thead>
<tr>
<th>References of experiment data</th>
<th>FAST</th>
<th>Our model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MSE</td>
<td>LL</td>
</tr>
<tr>
<td>Total sleep deprivation (40 h) (Jung et al. 2011)</td>
<td>6.4</td>
<td>−44.6</td>
</tr>
<tr>
<td>Total sleep deprivation (88 h) (Van Dongen 2004)</td>
<td>12.0</td>
<td>−114.5</td>
</tr>
<tr>
<td>14 days with 4-h daily sleep (Van Dongen 2004)</td>
<td>7.4</td>
<td>−121.0</td>
</tr>
<tr>
<td>14 days with 6-h daily sleep (Van Dongen 2004)</td>
<td>2.3</td>
<td>−80.8</td>
</tr>
<tr>
<td>7 days with 3-h daily sleep (Belenky et al. 2003)</td>
<td>15.0</td>
<td>−183.0</td>
</tr>
<tr>
<td>7 days with 5-h daily sleep (Belenky et al. 2003)</td>
<td>4.7</td>
<td>−129.6</td>
</tr>
<tr>
<td>7 days with 7-h daily sleep (Belenky et al. 2003)</td>
<td>0.8</td>
<td>−68.3</td>
</tr>
<tr>
<td>One-night sleep deprivation (own study)</td>
<td>22.8</td>
<td>−32.8</td>
</tr>
</tbody>
</table>

Figure 4 shows a graphical comparison of cognitive performance between the model predictions and experimental data when either placebo or caffeine was used under sleep deprivation. The effects of caffeine on the cognitive performance were well captured by our model in consistence with experimental data which is not the case for FAST. This is explicitly indicated by a much smaller MSE of our model than that of FAST (142.27 vs. 707.06) as calculated from the experiment data with the caffeine effects under sleep loss (Bonnet et al. 1995).

Figure 5 shows a graphical comparison of the cognitive performance between the model predictions and experimental studies where different pharmaceutical countermeasures were used under sleep loss. Our
model could simulate both improved performance for all drugs and different durations of performance enhancement according to the pharmacokinetics of the drugs: the longer duration of action by modafinil than dextro-amphetamine and caffeine is due to the longer half-life of modafinil. This is in the same order of duration of action by each drug as observed in the human study (Wesensten, Killgore, and Balkin 2005). In addition, Table 2 shows that our model had both smaller MSE and AIC than FAST indicating better predictions for performance in sleep deprivation with pharmaceutical countermeasures, while the two models were comparable based on their similar MSE and AIC values when no stimulant drugs were used.

Compared to the latest model (Ramakrishnan et al. 2013), the performance-restoring effects of caffeine during sleep deprivation in our model were mathematically described similarly using the Hill equation, however, the pharmacokinetics of caffeine was modelled differently (two compartments vs. one compartment). In addition, our model showed a better prediction as indicated by a smaller mean squared error (79.84 vs. 92.35) which was calculated using the same PVT lapses data. In addition, our model can simulate the performance-enhancing effects of other drugs using their corresponding pharmacokinetic parameters. For example, we have developed a physiologically based pharmacokinetic model for melatonin, a well-known countermeasure for fatigue due to jet lag and shift work (Peng et al. 2013). Together with the model for melatonin effects on phase shifts (Breslow et al. 2013), it is feasible to develop a model to predict the melatonin effects on performance under sleep loss (Peng and Bouak 2015).

Moreover, our model would not only account for the performance-enhancing effects of any pharmaceuticals, but also allow specification of the pharmaceutical doses and administration timing that would produce optimal performance for any particular operational scenarios.

Figure 4. Comparison between experimental cognitive performance as measured by visual vigilance tests (Bonnet et al. 1995) and predicted performance by FAST and our model when placebo and caffeine were used. Caffeine 400 mg was given at 01:30 each night during sleep deprivation. The performance was measured as proportion of visual vigilance relative to baseline as reported in the literature (Scerbo, Warm, and Fisk 1986).

Figure 5. Comparison between experimental performance as measured by psychomotor vigilance tasks (Wesensten, Killgore, and Balkin 2005) and predicted performance by our model under pharmaceutical countermeasures. An oral dose of caffeine 600 mg, dextroamphetamine (dextro) 20 mg or modafinil 400 mg was administered after 64-h awake.

<table>
<thead>
<tr>
<th>References of experiment data</th>
<th>FAST MSE</th>
<th>FAST LL</th>
<th>FAST AIC</th>
<th>Our model MSE</th>
<th>Our model LL</th>
<th>Our model AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep deprivation (52 h) (Bonnet et al. 1995)</td>
<td>160.6</td>
<td>−31.7</td>
<td>77.3</td>
<td>165.7</td>
<td>−31.6</td>
<td>77.6</td>
</tr>
<tr>
<td>Total sleep deprivation (52 h) with caffeine (Bonnet et al. 1995)</td>
<td>707.1</td>
<td>−37.6</td>
<td>91.2</td>
<td>142.4</td>
<td>−31.2</td>
<td>84.4</td>
</tr>
<tr>
<td>Total sleep deprivation (85 h) (Wesensten, Killgore, and Balkin 2005)</td>
<td>171.7</td>
<td>−87.8</td>
<td>189.6</td>
<td>157.3</td>
<td>−86.9</td>
<td>187.7</td>
</tr>
<tr>
<td>Total sleep deprivation (85 h) with caffeine (Wesensten, Killgore, and Balkin 2005)</td>
<td>770.6</td>
<td>−99.6</td>
<td>213.2</td>
<td>221.9</td>
<td>−90.6</td>
<td>203.3</td>
</tr>
<tr>
<td>Total sleep deprivation (85 h) with dextroamphetamine (Wesensten, Killgore, and Balkin 2005)</td>
<td>1554.9</td>
<td>−101.9</td>
<td>217.7</td>
<td>219.7</td>
<td>−90.5</td>
<td>203.1</td>
</tr>
<tr>
<td>Total sleep deprivation (85 h) with modafinil (Wesensten, Killgore, and Balkin 2005)</td>
<td>1044.7</td>
<td>−93.0</td>
<td>200.0</td>
<td>116.6</td>
<td>−83.6</td>
<td>189.1</td>
</tr>
<tr>
<td>Total sleep deprivation (61 h) with 12-h recovery sleep (Killgore et al. 2008)</td>
<td>36.4</td>
<td>−109.4</td>
<td>232.7</td>
<td>31.9</td>
<td>−107.1</td>
<td>228.2</td>
</tr>
<tr>
<td>Total sleep deprivation (61 h) with caffeine and 12-h recovery sleep (Killgore et al. 2008)</td>
<td>64.9</td>
<td>−119.2</td>
<td>252.4</td>
<td>37.2</td>
<td>−109.7</td>
<td>233.4</td>
</tr>
<tr>
<td>Total sleep deprivation (61 h) with dextroamphetamine and 12-h recovery sleep (Killgore et al. 2008)</td>
<td>69.4</td>
<td>−120.3</td>
<td>254.6</td>
<td>19.6</td>
<td>−98.8</td>
<td>211.6</td>
</tr>
<tr>
<td>Total sleep deprivation (61 h) with modafinil and 12-h recovery sleep (Killgore et al. 2008)</td>
<td>43.9</td>
<td>−112.5</td>
<td>239.0</td>
<td>19.6</td>
<td>−98.8</td>
<td>211.6</td>
</tr>
</tbody>
</table>
3.3. Model simulations: task demand

It is well known that performance is influenced by task characteristics (e.g., task difficulty, task type and time on task) under sleep deprivation (Van Dongen, Belenky, and Krueger 2011; Wilson, Caldwell, and Russell 2007) and shift schedules (Baulk et al. 2007; Pilcher et al. 2016). It has been recommended that bio-mathematical models should include work-related factors (such as cognitive demands or task difficulty) (Dinges 2004).

Our base model is based on the three processes as some of existing fatigue models for performance predictions under acute and chronic sleep loss (Raslear, Hursh, and Van Dongen 2011). These three components are task invariant and thus unable to distinguish performance on different types of tasks. To improve the model, we included a fourth component to take account of task difficulties characterised by varying levels of attentional demands. Such an expansion of the model allows us to examine the impact on operators’ performance by their tasks’ attentional demands (L) and the influence of sleep loss (R) on their mental capacity (W).

It should be noted that our current model does not account for multiple tasks which require multiple resources (Wickens 2008) and changes in task difficulty over time (time-on-task effect) (See et al. 1995), and is distinct from human performance models considering workload as a performance shaping factor (Gore 2011) and dynamic relationships between workload and performance without sleep loss (Mracek et al. 2014).

When compared with field data, Figure 6 shows that our model could differentiate the performance decrease according to task demand levels, but overestimated the performance for low task demand and underestimated impairment of sleep loss on performance in high task demand. Figure 7 also shows that our model predictions were more consistent with experimental data than FAST predictions for the performance on an operator vehicle interface task of low and high difficulty (Wilson, Caldwell, and Russell 2007). The model initially predicted identical performance levels for high and low demanding tasks for approximately 10 h into performance, and then showed more performance impairment in the high demanding task. This implies that the sleep loss manifested the effects of task demand during long wakefulness. Together with Table 3, it is demonstrated that our model could predict performance for different task demands.

It should be noted that the task demand in our model was defined based on how much attention is required by the task itself. It is an external fatigue factor inherent in a given task and is different from the workload defined as the balance between task demands and resources (Young et al. 2015). For example, a visual tracking task that requires sustained undivided attention was commonly considered a high demanding task with a rating of 3, while seldom attention in case of automatic processing was given a rating of 0. The experimental data were selected for modelling based on their performance measurements at multiple time points on tasks of different difficulties. Task characteristics were also manipulated by the time on cognitive tests and found to have no effects on performance under normal and restricted sleep conditions (Goel et al. 2014). This is consistent with the report that mental workload was more sensitive to the change in task difficulty than on task (Haga, Shinoda, and Kokubun 2002).

3.4. Sensitivity analysis of model input parameters

Table 4 summarises the results of the sensitivity analysis of model parameters and sleep input performed for an 88-h total sleep loss condition. Among the model parameters, the model showed the highest sensitivity to changes of the parameter $K$ (depletion rate constant of sleep reservoir), $p$ (the peak of the 24-h rhythm) and $p'$ (the peak of the 12-h rhythm), and least sensitivity to changes of the empirical parameters $a$, $i$ and $f$. Compared to the model parameters, the sleep input had greater influence on model prediction. This indicates that our model is robust to minor deviations from the estimated parameter values and can be used to predict performance with sleep input.

3.5. Model simulations: shift work schedules

Figure 8 depicts the simulations of two shift work schedules in comparison with the experimental measures of
3.6. Model simulations: sleep schedules

There are a number of models to predict sleepiness in the literature (Achermann and Borbély 2003; Åkerstedt 2014). Some models can also predict sleep latency (Åkerstedt and Folkard 1996). In our current model, we incorporated the two-process model of sleep regulation reported by Achermann and Borbély et al. (Achermann 2004; Achermann and Borbély 1994; Borbély and Achermann 1999; Daan, Beersma, and Borbély 1984). We are comparing different sleep prediction models to determine which one is the best to use in the future.

Analysis of the sleep model predictions incorporated into our fatigue model was conducted to compare with experimental sleep data for shift work schedules. As indicated in Tables 5 and 6, the sleep model predicted all sleep episodes in agreement with the experimental findings and the predictions for sleep, wake-up time and sleep duration were within a 2-h range compared to the experimental data. The discrepancy depends on the type of shifts. The model was considered acceptable for sleep predictions between night and afternoon shift when within one standard deviation of the experimental mean was used as a criterion.

It should be noted that although the guiding principle for the sleep model predictions were based on the homeostatic and circadian processes as for the performance model predictions the underlying mathematical representations are different.

3.7. Model limitations

Even with additional factors incorporated our current model is designed to predict cognitive performance under mental fatigue from sleep loss and circadian displacement, and does not account a number of importance factors such as individual differences in responses to sleep loss cognitive performance (Axelsson et al. 2004; Baulk et al. 2009). Specifically, two steps were involved. First, both FAST and our model were used to predict sleep from the shift work schedules using their sleep prediction algorithm (FAST AutoSleep module and our sleep prediction model). The estimated sleep–wake schedule is then used as the input in each model to predict performance, respectively. The accuracy of the model is reflected by the consistency in the values of predicted performance with the experimental data for both shift schedules. Clearly, both model simulations and experimental studies consistently illustrate the reduced performance during the night shifts. In addition, our model provided a slightly more accurate prediction for cognitive performance than FAST in both sleep scenarios as indicated by their MSEs (4.33 and 31.85 vs. 8.44 and 43.66).

### Table 3. The goodness-of-fit of each model to experimental performance with low and high task demand under sleep loss.

<table>
<thead>
<tr>
<th>References of experiment data</th>
<th>FAST MSE</th>
<th>LL</th>
<th>AIC</th>
<th>Our model MSE</th>
<th>LL</th>
<th>AIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep deprivation (60 h) Low (Gould et al. 2009)</td>
<td>116.0</td>
<td>−30.4</td>
<td>74.7</td>
<td>96.7</td>
<td>−29.6</td>
<td>77.3</td>
</tr>
<tr>
<td>Total sleep deprivation (60 h) High task demand (Gould et al. 2009)</td>
<td>2216.2</td>
<td>−42.2</td>
<td>98.3</td>
<td>234.9</td>
<td>−33.2</td>
<td>84.4</td>
</tr>
<tr>
<td>One-night sleep deprivation Low (Wilson, Caldwell, and Russell 2007)</td>
<td>56.0</td>
<td>−17.2</td>
<td>48.3</td>
<td>72.0</td>
<td>−17.8</td>
<td>53.6</td>
</tr>
<tr>
<td>One-night sleep deprivation High (Wilson, Caldwell, and Russell 2007)</td>
<td>930.6</td>
<td>−24.2</td>
<td>62.4</td>
<td>357.7</td>
<td>−21.8</td>
<td>61.3</td>
</tr>
</tbody>
</table>

### Table 4. Sensitivity analysis of model parameters and sleep input for performance prediction under an 88-h total sleep deprivation condition. Each parameter was varied to ± 5% of its initial value and the new MSE was calculated. The sensitivity was determined by the relative deviations to the initial MSE calculated with the unvaried model parameters and sleep input.

<table>
<thead>
<tr>
<th>Model input parameters</th>
<th>Sensitivity</th>
<th>Sleep input</th>
</tr>
</thead>
<tbody>
<tr>
<td>$K$</td>
<td>−0.073, 0.153</td>
<td>0.255, 0.541</td>
</tr>
<tr>
<td>$a_t$</td>
<td>0.000, 0.000</td>
<td>0.000, 0.000</td>
</tr>
<tr>
<td>$\tau_d$</td>
<td>0.001, −0.000</td>
<td>0.000, 0.000</td>
</tr>
<tr>
<td>$p$</td>
<td>0.077, −0.017</td>
<td>0.000, 0.000</td>
</tr>
<tr>
<td>$\beta$</td>
<td>−0.002, 0.004</td>
<td>0.000, 0.000</td>
</tr>
<tr>
<td>$p'$</td>
<td>0.002, −0.004</td>
<td>0.000, 0.000</td>
</tr>
<tr>
<td>$i$</td>
<td>0.000, 0.000</td>
<td>0.255, 0.541</td>
</tr>
<tr>
<td>$f$</td>
<td>0.000, 0.000</td>
<td>0.541, 0.255</td>
</tr>
</tbody>
</table>
requires more research to determine the validity of the component. In addition, current sleep and performance predictions were based on the assumption of standard 8-h sleep with a 23:00 bedtime and 07:00 wake time prior to sleep loss and excellent sleep quality. We are further developing our model to address these limitations such as replacement of the current circadian component with a light-based circadian module (Peng and Bouak 2015). Our model predictions are valid for simple measures of cognitive performance, attention and vigilance, but like other bio-mathematical models of neurobehavioural performance, research must be carried out to show their validity relative to the effects of sleep deprivation on many higher level cognitive capacities, including perception, memory and executive functions. A recent review suggests that cognitive performance on complex tasks (e.g. decision-making tasks, multiple tasks, working memory tasks and tasks requiring team communication) may not be impaired by disrupted sleep as severely as cognitive performance on simple tasks (e.g. psychomotor vigilance task and basic reasoning tasks) (Wickens et al. 2015).

4. Conclusions

A DRDC fatigue model including a base model comparable with the commercial FAST, additional factors affecting fatigue and performance, and sleep predictions have been developed. The improved model was able to take into account of pharmaceutical countermeasures, task demand and shift work schedules with sleep predictions. The flexibility of our MATLAB® model would also allow further usability improvements, customisation and direct integration into other tools. Future work may be focused on validation of the DRDC model with our own laboratory and field study data. Ultimately, we aim to develop a multi-faceted and user-friendly cognitive fatigue model that can be integrated into current military applications to

![Figure 8](image_url)

**Figure 8.** Comparison between experimental performance as measured by psychomotor vigilance tasks and predicted performance by our model and FAST based on two shift work schedules: (A) experimental study comprised night shift (N-shift: 21:00–06:00), afternoon shift (A-shift: 14:00–21:00) and morning shift (M-shift: 06:00–14:00) (Axelsson et al. 2004) and (B) experimental study comprised two day shifts (07:00–19:00) and two night shifts (19:00–07:00) (Baulk et al. 2009).

<p>| Table 5. Comparison between model-predicted sleep schedules with experimental data. |
|-------------------------------------------------|---------------------|---------------------|---------------------|</p>
<table>
<thead>
<tr>
<th>Sleep episodes</th>
<th>Experimental data (Axelsson et al. 2004)</th>
<th>Model Prediction</th>
<th>Experimental – model (h)</th>
<th>Within 1 standard deviation of the experimental mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep before N-shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed time</td>
<td>23:20 ± 1:13</td>
<td>00:30</td>
<td>−1.67</td>
<td>No</td>
</tr>
<tr>
<td>Rise time</td>
<td>8:04 ± 1:31</td>
<td>7:15</td>
<td>0.75</td>
<td>Yes</td>
</tr>
<tr>
<td>Duration (h)</td>
<td>8.73 ± 1.86</td>
<td>6.75</td>
<td>1.98</td>
<td>No</td>
</tr>
<tr>
<td>Sleep between N-A-shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed time</td>
<td>6:22 ± 0:22</td>
<td>6:00</td>
<td>0.37</td>
<td>Yes</td>
</tr>
<tr>
<td>Rise time</td>
<td>11:15 ± 0:45</td>
<td>10:30</td>
<td>0.75</td>
<td>Yes</td>
</tr>
<tr>
<td>Duration (h)</td>
<td>4.89 ± 0.75</td>
<td>4.5</td>
<td>0.39</td>
<td>Yes</td>
</tr>
<tr>
<td>Sleep between A-M-shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed time</td>
<td>22:35 ± 0:44</td>
<td>23:00</td>
<td>−0.42</td>
<td>Yes</td>
</tr>
<tr>
<td>Rise time</td>
<td>4:29 ± 0:22</td>
<td>6:00</td>
<td>−1.52</td>
<td>No</td>
</tr>
<tr>
<td>Duration (h)</td>
<td>5.91 ± 0.67</td>
<td>7.00</td>
<td>−1.09</td>
<td>No</td>
</tr>
</tbody>
</table>

Notes: Experimental data were represented as mean ± standard deviation. N = night shift (21:00–06:00), A = afternoon shift (14:00–21:00), M = morning shift (06:00–14:00).
improve the design of future work systems or work schedules, or to improve the real-time management of sleep and countermeasures for sleep deficit in the face of challenging or fluctuating work schedules.

Acknowledgements
The authors wish to thank the following co-op students for their technical support: Hao Zhou, Saad Ali, John Catton and Yusuf Ali.

Disclosure statement
No potential conflict of interest was reported by the authors.

Funding
The work was supported by Defence Research and Development Canada under [project number 01ab] and [project number 14dm].

References

<table>
<thead>
<tr>
<th>Sleep episodes</th>
<th>Experimental data (Baulk et al. 2009)</th>
<th>Model Prediction</th>
<th>Experimental – Model (h)</th>
<th>Within 1 standard deviation of the experimental mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sleep before the first day shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed time</td>
<td>N/A</td>
<td>00:30</td>
<td>7:00</td>
<td>1.22</td>
</tr>
<tr>
<td>Rise time</td>
<td>7:00</td>
<td>6.5</td>
<td>1.22</td>
<td>Yes</td>
</tr>
<tr>
<td>Duration (h)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep between the two day shifts</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed time</td>
<td>N/A</td>
<td>22:45</td>
<td>7:00</td>
<td></td>
</tr>
<tr>
<td>Rise time</td>
<td>7:00</td>
<td>8.25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration (h)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep before the night shift</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed time</td>
<td>N/A</td>
<td>23:00</td>
<td>7:00</td>
<td></td>
</tr>
<tr>
<td>Rise time</td>
<td>7:00</td>
<td>8.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration (h)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep between the two night shifts</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bed time</td>
<td>N/A</td>
<td>7:00</td>
<td>11:15</td>
<td>negatively correlated</td>
</tr>
<tr>
<td>Rise time</td>
<td>7:00</td>
<td>4.25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration (h)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Experimental data were represented as mean ± standard deviation. Day shift (07:00–19:00) and night shift (19:00–07:00); N/A = not available.


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<tr>
<td>Peng, H.; Bouak, F.; Wang, W.; Chow R.; Vartanian, O.</td>
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Fatigue has become an increasing problem in our modern society. Using MATLAB as a generic modelling tool, a fatigue model was developed based on an existing one and compared with a commercial fatigue software for prediction of cognitive performance under total and partial sleep deprivation. The flexibility of our fatigue model allowed additions of new algorithms and mechanisms for non-sleep factors and countermeasures and thus improved model predictions and usability for both civilian and military applications. This was demonstrated by model simulations of various scenarios and comparison with experimental studies. Our future work will be focused on model validation and integration with other modelling tools.

Practitioner Summary: Mental fatigue affects health, safety and quality of life in our modern society. In this paper, we reported a cognitive fatigue model based on existing models with newly incorporated components taking both the operator’s state of alertness and task demand into account. The model provided the additional capability for prediction of cognitive performance in scenarios involving pharmaceutical countermeasures, different task demands and shift work.

Bio-mathematical model, mental fatigue, cognitive performance, sleep, shift work