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Fatigue in Sustained Attention:

Generalizing Mechanisms for Time Awake to Time on Task

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Time on task and time awake are two important influences on human cognitive performance. Both extended periods of wakefulness and extended periods of effort on a single task lead to performance declines (e.g., Angus & Heslegrave, 1985; Davies & Parasuraman, 1982; Dijk, Duffy, & Czeisler, 1992; Van Dongen & Dinges, 2005a; 2005b). Of particular interest in the present work is the *time on task effect* or *vigilance decrement*, which refers to progressively worse performance that is observed on vigilance tasks as the duration of those tasks increases (e.g., Davies & Parasuraman, 1982, Matthews & Davies, 2001; Methot & Huitema, 1998). In naturalistic settings, this issue poses a serious threat to productivity and safety, particularly when combined with the effect of extended wakefulness in today's 24x7 society (Angus & Heslegrave, 1985; Baker, Olson, & Morisseau, 1994; Caldwell, 2005; Dinges, 1995; Horne & Reyner, 1999; Pack et al., 1995).

Historically, fatigue effects resulting from time on task have been treated as distinct from those resulting from extended wakefulness. There is neurophysiological evidence to suggest that fatigue associated with sleep homeostatic and circadian processes is associated with subcortical processes (e.g., Forger, Gonze, Virshup, &Welsh, 2007; Saper, Chou, & Scammel, 2001; Saper, Scammel, & Lu, 2005). Sleep homeostatic effects may also be localized to specific cortical areas (Krueger & Obál, 1993; Krueger, Rector, Roy, Van Dongen, Belenky, & Panksepp, 2008).

In the research presented here, we ask whether performance changes associated with time on task can be simulated using the same computational mechanisms that replicate changes in cognitive performance associated with extended sleep deprivation. Such correspondence would raise the possibility that similar neural mechanisms underlie both effects – a possibility

addressed in the conclusion (see also Van Dongen et al., this volume). To situate this issue, we investigate human performance on a task measuring attentional vigilance, which refers to the ability to maintain focused attention on a task and respond rapidly to repetitive stimuli. There has been a considerable amount of research on sustained attention performance in humans from a variety of perspectives (e.g., Davies & Parasuraman, 1982; Matthews et al., 1992; Sakai, Baker, & Dawson, 1992; Thiffault & Bergeron, 2003; Van Dongen & Dinges, 2005a). The capacity to sustain attention is critical in monitoring tasks that are central in many transportation domains (e.g., Caldwell, 2005; Dean, Fletcher, Hursh, & Klerman, 2007), and in tasks that are central for national defense (e.g., Angus & Heslegrave, 1985; Basner et al., 2008; Caldwell, Caldwell, Brown, & Smith, 2004; Hancock & Hart, 2002; Hursh et al., 2004).

The specific task investigated here is the Psychomotor Vigilance Test, or PVT (Dinges & Powell, 1985). The PVT has been used extensively to investigate sustained attention performance changes stemming from time awake and circadian rhythms (e.g., Doran, Van Dongen, & Dinges, 2001; Dorrian et al., 2005; Van Dongen & Dinges, 2005b). In the task, participants wait for a stimulus to appear at a known location on a display. Each stimulus occurs randomly between 2 s and 10 s after the previous response, and when it does, participants respond by pressing a button as fast as they can. Though procedurally straightforward, the 10-minute duration of a typical session makes it a challenging task for individuals to perform, particularly when deprived of sleep (Van Dongen, Maislin, Mullington, & Dinges, 2003). The data we consider here come from a study described in Doran et al. (2001), where participants completed a 10-minute PVT session every 2 hours over the course of 88 hours of sustained wakefulness.

To evaluate performance, we classify reaction times in the PVT into one of 4 categories. *Alert responses* are when the reaction time to the appearance of the stimulus is between 150 ms and 500 ms. Faster responses (i.e., less than 150 ms) should not be physically possible, and together with responses made before the stimulus appears, are described as *false starts*. Slower responses beyond 500 ms reflect degraded performance and are termed *lapses*. Finally, there are occasions where participants completely fail to respond, even after 30 s. We refer to these trials as *non-responses*. In the experiment described in Doran et al. (2001), these trials were interrupted with a beep from the computer to alert the participant for the start of the next trial.

Performance on the PVT is sensitive to both time awake and circadian rhythms, providing an assay of overall cognitive functioning, or behavioral alertness (Dinges & Powell, 1985; Dorrian et al., 2005; Van Dongen & Dinges, 2005b). As alertness declines, false starts, lapses, and non-responses all increase in probability, and the median reaction time of the remaining alert responses increases (i.e., the whole RT distribution shifts, see Lim & Dinges, 2008). Our research efforts involving the PVT have been focused on developing computational accounts of these performance changes (e.g., Gunzelmann, Gluck, Price, Van Dongen, & Dinges, 2007; Gunzelmann, Gross, Gluck, & Dinges, 2009; Gunzelmann, Moore, Gluck, Van Dongen, & Dinges, in press). We developed a computational model of the PVT and integrated mechanisms to account for changes in cognitive processing resulting from time awake and circadian rhythms. The model produces behavior that is in line with human performance, and tracks changes that occur over the course of 88 hours of continuous wakefulness (Gunzelmann, Gross, et al., 2009). The model accounts for systematic fluctuations in median alert reaction time, as well as changes in the likelihood of false starts, lapses, and non-responses, for both aggregate human data (Gunzelmann, Gross, et al., 2009) and for individual participants as well (Gunzelmann, Moore,

Gluck, et al., in press). However, the vigilance decrement had not yet been addressed in this work.

The focus of the current effort is to evaluate the generalizability of the explanatory mechanisms we previously proposed in the context of the effects of sleep deprivation, now in the context of the effects of time on task. Changes in alertness associated with sleep and circadian rhythms are orchestrated by subcortical mechanisms (e.g., Saper et al., 2001; Saper et al., 2005). However, in the case of time on task, it is likely that other neural mechanisms are responsible for producing the effect. It has been hypothesized that local, use-dependent sleep regulatory mechanisms in cortical columns and other neuronal assemblies drive the homeostatic (i.e., time awake-related) pressure for sleep as well as the time on task effect (e.g., Krueger et al., 2008; Van Dongen, Belenky, & Krueger, this volume). Indeed, effects of time on task in PVT performance have been found to go hand in hand with changes stemming from sleep loss and circadian rhythms (Doran et al., 2001; Wesensten, Belenky, Thorne, Kautz, & Balkin, 2004; Van Dongen & Belenky, 2008).

Nevertheless, questions remain regarding whether the impacts of these distinct processes on the general construct of *fatigue* are similar or different, and whether the same computational mechanisms can serve to explain them jointly. In the next sections, we describe our methodological approach, computational model, and mechanisms for fatigue in more detail. Then, we provide an evaluation of whether the mechanisms we have proposed to account for the effects of sleepiness on cognitive processing generalize to time-on-task phenomena.

Modeling Fatigue in a Cognitive Architecture

Gigerenzer and Brighton (2009) argue for a transition from verbal theoretical arguments about cognition to the development of mathematical and computational models that instantiate theoretical claims and produce testable quantitative predictions about performance. Our use of a cognitive architecture reflects this research orientation. More specifically, we use the Adaptive Control of Thought – Rational, or ACT-R, cognitive architecture (Anderson, 2007; Anderson et al., 2004) for modeling the effects of fatigue. ACT-R is a general theory of human cognition, which has been implemented in computer software. It has been used to provide accounts of a variety of psychological phenomena (e.g., Anderson, 2007; Anderson et al., 2004; Anderson & Lebiere, 1998). A complete overview of the ACT-R architecture and its theoretical components and computational mechanisms is beyond the scope of this chapter. Here we describe components that are critical to the account of fatigue we have implemented in the context of the PVT. Further details about the mechanisms of ACT-R can be found elsewhere (e.g., Anderson, 2007; Anderson et al, 2004).

In the context of our research, there are two critical characteristics of ACT-R. The first is the division of cognitive processing into a number of integrated modules, reflecting claims about modularity and localization of function in the human cortex. Each module contains specialized information processing mechanisms that process requests from central cognition and return the results to buffers, which serve as the interface with central cognition. In ACT-R's vision module, for example, a request for a shift in visual attention leads to an update of two buffers representing the location and identity of the item that is attended. For the PVT, ACT-R's perceptual and motor modules are also critical as they give ACT-R the capacity to interact directly with a software implementation of the performance task, leading to observable model behavior that we compare to human performance data.

The second critical component of the architecture for this research is the representation of central cognition, which takes the form of a production system. This system is represented as a distinct module in ACT-R. It operates through a sequence of recognize-decide-act cycles, where the current pattern of information across the entire system is compared against potential actions, and one action is selected and then executed (fired). The pattern of information consists of the contents of the set of buffers in ACT-R, as well as information representedfrom in the environment (e.g., a featural representation of the visual environment in ACT-R's visual icon). When a production fires, it leads to a modification to the pattern of information by sending requests to modules for various actions. These may be perceptual (e.g., shifts of visual attention), cognitive (e.g., a declarative retrieval request), or motor (e.g., an action in the environment).

The integration of perceptual and motor mechanisms with mechanisms for cognitive processing is essential for examining the impact of fatigue. Perceptual and motor capabilities enable interaction with software-based performance tasks, meaning that ACT-R is able to participate in experiments just as humans do, where the models produce behavioral traces that are equivalent to those produced by human participants doing the same tasks. That is, ACT-R generates virtual behavior like button presses, which can be recorded and which affect the task software in the same way as human-generated actions. Thus, performance predictions of the ACT-R computational model can be compared directly to the performance of human participants at whatever levels of granularity are most appropriate for the research. This is critical to the research described here, both in terms of validating the computational models and mechanisms, and in demonstrating the potential benefits in applied settings.

In the context of time awake and circadian rhythms, we have developed mechanisms that influence the behavior of our computational models, which provide accounts for both how and

why cognitive performance changes as a consequence of fluctuations in alertness (e.g., Gunzelmann, Byrne, Gluck, & Moore, 2009; Gunzelmann, Gross, et al., 2009; Gunzelmann, Gluck, Kershner, Van Dongen, & Dinges, 2007). To track the dynamics of alertness, we rely on the predictions from biomathematical models, which represent the effects of time awake and circadian rhythms on alertness (e.g., Daan, Beersma, & Borbély, 1984; Achermann, 2004; Dinges, 2004; Mallis et al., 2004). Overviews of such models are available (e.g., Mallis et al., 2004; Van Dongen, 2004), as are more detailed descriptions of specific instances of such models (e.g., Hursh et al., 2004; Jewett & Kronauer, 1999; McCauley, Kalachev, Smith, Belenky, Dinges, & Van Dongen, 2009).

Our approach has been to use the output of biomathematical models as predictions of overall cognitive functioning, which can be tied to specific parameters and mechanisms in ACT-R. Thus, we used predictions of alertness as input into mechanisms associated with different components of human information processing. An advantage of this approach is that it should apply across tasks, allowing us to assess the robustness and validity of our theoretical account by making a priori quantitative performance predictions in novel task contexts based upon model fits derived from different task (e.g., Gunzelmann, Byrne, et al., 2009; Gunzelmann & Gluck, in press; Gunzelmann, Moore, Salvucci, & Gluck, in press).

Next, we describe the particular theoretical account we have developed to understand changes in human performance on the PVT that arise during extended periods of wakefulness. After this description, we present results exploring the capacity of those same mechanisms to capture changes in performance associated with time on task.

Model and Mechanisms

Our computational model of the PVT places particular emphasis on ACT-R's central cognitive processes, which must execute a rapid series of coordinated actions to make a fast response. The foundation of the model consists of processes that (1) wait for the stimulus to appear during the delay period, (2) shift visual attention to the stimulus when it appears, and (3) generate a response in the form of a virtual button press. These processes are represented as productions in ACT-R. The first two processes are sensitive to the presence or absence of the stimulus. When the stimulus appears, the first process no longer applies whereas the second does, and it generates a request for ACT-R's visual system to shift attention to the item. The third process generates a response through a request to ACT-R's motor system. Importantly, responses may occur in the absence of the stimulus, creating the possibility of false starts (see Gunzelmann, Gross, et al., 2009). The selection of a production to execute on each cycle is driven by an estimation of the expected utility of the available options:

$$U_i = P_i G - C_i + \varepsilon \qquad \text{Eq. 1.}$$

In Eq. 1, P_i represents the probability that the goal will be achieved with production *i*, and C_i represents the anticipated cost of achieving the goal should that production be fired. We use the value of 33 ms for C_i for all productions, which varies from the default value of 50 ms in ACT-R (e.g., Anderson et al., 2004), but is influenced by significant individual differences, which can be accurately captured with this parameter (Gunzelmann, Moore, Gluck, et al., in press). Noise (ε) is added to the calculation of utility to produce stochasticity in the selection process. It is selected from an approximately normal distribution with a mean of 0 and a standard deviation of approximately 0.453. *G* is a global parameter, which has been referred to as "the value of the goal" in seconds (Anderson & Lebiere, 1998, p. 60), but has been associated with motivation (e.g., Belavkin, 2001) and arousal (e.g., Jongman, 1998) as well.

We have manipulated *G* to represent the impact of changes in alertness caused by time awake and circadian rhythms based on the predictions of alertness from biomathematical models. Reductions in the value of *G* increase the likelihood that no productions exceed the utility threshold, T_u, during a cognitive cycle. This leads to the key consequence of fatigue in our model, namely small delays in cognitive processing which we call *microlapses*. When no productions exceed T_u, a microlapse occurs that lasts for the duration of the cognitive cycle (i.e., approximately 33 ms). Our results have shown that such brief breakdowns in cognitive performance can explain results attributed to cognitive slowing and cognitive lapsing in the research literature (Gunzelmann, Gross, et al., 2009).

In addition to dynamic changes in *G*, we have manipulated the threshold for action in the procedural system (T_u). In this case, the threshold is lowered to instantiate the hypothesized impact of compensatory effort on cognitive performance. This attribution is based upon theoretical commitments in ACT-R and neurophysiological findings from the sleep research community (e.g., Portas et al., 1998; Thomas et al., 2000; Doran et al., 2001). Within a single trial of the PVT, utility values decline during microlapses, which progressively decreases the likelihood of a response as microlapses accumulate.

Changes in reaction times are thus produced by the interaction of the dynamic changes in the two parameters *G* and T_u. Cognitive slowing is manifested by the increasing probability that small numbers of microlapses will introduce delays in making the response. Lapses, and eventually non-responses, increase as a consequence of probabilistically longer sequences of these brief interruptions. False starts are primarily a result of manipulations of T_u, representing compensatory effort.

Lastly, we have done some initial explorations for capturing individual differences in performance on this task (Gunzelmann, Moore, Gluck, et al., in press). We have found that altering the duration of cognitive cycles in ACT-R (i.e., a manipulation of the processing speed) provides an effective way of capturing these differences. Interestingly, this evaluation did not provide evidence that it would be useful to alter the processing speed as a function of time awake or circadian rhythm, which may have implications regarding whether *cognitive slowing* is an appropriate theoretical construct for accounting for changes in performance associated with fatigue. As noted above, we use a value of 33 ms here, which remains constant across both time awake and time on task in the model fits presented below.

Next we explore whether these mechanisms can be extended to account for fatigue effects beyond those resulting from time awake and circadian rhythms. Specifically, we evaluate whether changes in performances resulting from prolonged time on task can also be explained by the mechanisms we have described.

Model Evaluation

We conducted our evaluation of time on task effects using the same empirical data that we used in developing the mechanisms described above, which came from a study of sleep deprivation described in Doran et al. (2001). Briefly, after three nights in the laboratory with 8 hours time in bed (23:30–07:30), participants were kept awake continuously for 88 hours. They completed a 10-minute PVT session every 2 hours during scheduled wakefulness throughout the study. To evaluate the effects of time on task, we aggregated the data from sessions taking place during each day of the sleep deprivation period. This includes 8 sessions during the day when the 88 hours of extended wakefulness began, which we refer to as the baseline day, and 12 sessions

on each of the remaining days, referred to as days 1, 2, and 3 of total sleep deprivation (TSD). Thus, our analysis includes changes resulting from sleep deprivation, but averages over variations associated with circadian rhythms. We then divided the data within sessions into each minute of performance, based upon when the response was made. Although this is a relatively coarse level of evaluation, it helps to ensure that there are adequate data to identify systematic changes in performance. The comparison between the human data and the model was conducted using the proportion of responses within each minute classified as false starts, lapses, and non-responses, and the median reaction time for the alert responses.

As a first step in assessing the ability to capture the observed trends over time on task with the existing computational model and set of fatigue mechanisms, a space of possible parameter values was explored. Allowing each of the parameters previously considered (i.e., G and T_u) to vary would result in extremely good fits to the data, but also lacks theoretical constraint comparable to what our previous research had achieved through the incorporation of biomathematical model predictions. Therefore, we set out to investigate the nature of time on task effects in the aggregate data of each day of sleep deprivation.

An ANOVA revealed significant differences for time within session in relation to lapses and non-responses. Median reaction time was also marginally significant. These results are presented in Table 1. When considering the mean daily biomathematical prediction of alertness using the Jewett & Kronauer (1999) model, and minute within session as independent predictors for each of our outcome measures (false starts, median reaction time, lapses, and non-responses) on the aggregate human data, all showed significant linear relations with minute ($p < .001$, $p < .01$, $p < .001$, and $p < .001$, respectively) and with the biomathematical prediction of alertness ($p < .001$ for all). Support for an additional interaction term between the two predictors was mixed,

with the least support for median reaction time ($p = .57$), and the strongest support from non-responses ($p < .001$).

Based upon these results, we followed a similar approach to our previous research by using a linear function to constrain the changes in parameter values across the 10 minutes of the sessions. Therefore, the performance of the computational model is based upon estimating separate linear functions (intercept and slope over time on task) to constrain the values of *G* and T_u. Figure 1 presents the best-fitting values for the ACT-R parameters obtained in our unconstrained fit of the model, as well as the values generated using a linear function to constrain the changes across minutes of performance within each PVT session. There was evidence for differences in the estimated intercepts across levels of sleep deprivation for both *G* and T_u ($p < .001$), whereas change in the slope across levels of sleep deprivation (i.e. an interaction) was not supported ($p \approx .4$ for both *G* and T_u).

As to whether the slopes were non-zero, results were mixed (for *G*: $p < .001$; for T_u: $p=.19$), however, previous work has consistently shown that both parameters have a strong relationship with alertness. So, the same manipulations were applied to both *G* and T_u. Specifically, the slope for changes in these parameters within a session was held constant for both across days of sleep deprivation, while the intercept varied. The changes in the intercept, however, were constrained by the dynamics of alertness predicted by the sleep homeostatic process in a biomathematical model. The trend with freely varying slopes was toward more steeply declining values as level of sleep deprivation increased. The lack of a significant effect may reflect limited statistical power in the comparison, which may be related to averaging data over sessions, the duration of each session (10 minutes), and/or the sample size (N=13). Whatever the explanation, this is a result that requires further investigation.

The performance of the model was measured using G and T_u values constrained by linear change over minutes with only intercept values varying over days. Changes in intercept were constrained by the mean daily biomathematical model predictions as described above. The results are shown in Figure 2 along with data from the human participants (averaged over N=13). The computational model captured the qualitative trends in the human data for each of the dependent measures of interest., and also produced data that are inline with the quantitative levels of performance of the participants in the study. Table 2 presents the correlations and root-mean-squared deviations (RMSD) between the model and the human data across these four measures.

Conclusions

The results of the current evaluation support the idea that the declines observed in individuals deprived of sleep are qualitatively similar to the declines observed as a consequence of prolonged time on task. The mechanisms that we have proposed to account for performance changes resulting from time awake and circadian rhythms are also able to capture the declines observed over the course of the 10-minute duration of the sustained attention task, suggesting that the two phenomena may share the same underlying neurobiology. The key adaptation in this model was to allow for time-based declines in ACT-R parameters reflecting alertness (G) and effort (T_u), leading to progressive declines in the model's behavior as time on task increased, mirroring behavioral changes seen in human performance on the PVT.

Extensive research has explored the neurophysiological basis for sleep and circadian rhythms (e.g., Saper et al., 2005). Many of the underlying processes seem to be rooted in the subcortical arousal systems (e.g., basal forebrain, suprachiasmatic nucleus, ventrolateral preoptic

area, thalamic regions, locus coeruleus, etc.). Dinges and colleagues (Doran et al., 2001; Lim and Dinges, 2008) posited that under conditions of sleep deprivation, these mechanisms cause sleep to intrude into wakefulness even in the face of compensatory effort. This *state instability* hypothesis explains the occurrence of lapses, non-responses and false starts, but it does not specifically address the time on task effect. Krueger and colleagues (Krueger et al., 2008) proposed that global pressure for sleep, while orchestrated by subcortical nuclei, is fundamentally driven by local processes at the level of cortical columns and other neuronal assemblies, which develop a metabolism- or plasticity-related need for sleep in response to prior use (e.g., task-related neuronal activity). Experiments in rats have yielded evidence that cortical columns can become locally unresponsive in a homeostatic manner in response to prior use, and that this phenomenon is associated with errors of omission and errors of commission on a licking task (Rector, Topchiy, Carter, & Rojas, 2005; Krueger et al., 2008) similar to lapses and false starts as observed in humans performing the PVT under conditions of sleep loss or prolonged time on task (Doran et al., 2001). This line of research has yielded evidence that local sleep at the level of cortical columns and other neuronal assemblies, through its pathway-specific and use-dependent properties, may be one mechanism by which the effects of time on task and time awake on performance may be jointly explained (Van Dongen et al., this volume).

To evaluate this conjecture requires more evidence regarding neurophysiology and cognitive functioning than is currently available. Biomathematical models of fatigue have not been developed to account for local changes in processing that may result from time on task. Thus, they cannot be used to understand the dynamics of performance on a task as a function of extended time on task. These dynamics operate at a short time scale (i.e., minutes), and require the specification of the underlying biological processes before they can be used to constrain the

dynamics of parameters associated with cognitive processes. In the current evaluation, a linear decline in alertness was sufficient to capture the general decline in performance across time on task that was observed in the human data, but this mapping was based upon observations in a limited context. Data from sleep deprivation studies using a 20-minute version of the PVT (Van Dongen, Baynard, Maislin, & Dinges, 2004) or longer vigilance tasks may shed further light on the linearity of the time on task effect.

In conclusion, the research presented here provides an initial exploration of computational mechanisms for fatigue associated with time on task and for fatigue associated with time awake and circadian rhythms. Our findings suggested that the short-term effects of time on task and the longer-term effects of sleep/wake homeostasis on cognitive performance may have shared underlying neurophysiological mechanisms. This has important implications in applied settings, where long work shifts and extended wakefulness combine to create situations where performance decrements are severe and may lead to disastrous consequences (e.g., Mitler et al., 1988; Åkerstedt, 2000; Dinges, 1995). For example, we have used the computational mechanisms described here to make quantitative predictions about changes in driver behavior resulting from extended time awake, showing how changes in sustained attention may impact driver performance (Gunzelmann, Moore, et al., in press). Such translational research has the potential to predict and quantify the risk of accidents associated with fatigue, and may ultimately lead to interventions that improve performance and safety in the 24x7 society.

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Table 1. Within-subject ANOVA results.

Measure	Source	df	Mean Square	F	Effect Size (η_G^2)
False Starts	M	1	0.0054	2.0	0.033
False Starts	A	1	0.2704	98.34**	0.627
False Starts	M x A	1	0.0133	4.83*	0.076
False Starts	Error	512	0.0028		
Median Alert RT	M	1	6866	3.67	0.027
Median Alert RT	A	1	42447	22.72**	0.147
Median Alert RT	M x A	1	427	0.23	0.002
Median Alert RT	Error	512	1868		
Lapses	M	1	0.1604	11.65**	0.055
Lapses	A	1	1.3233	96.10**	0.324
Lapses	M x A	1	0.0126	0.92	0.005
Lapses	Error	512	0.0138		
Non-Responses	M	1	0.0160	17.54**	0.246
Non-Responses	A	1	0.0659	72.09**	0.573
Non-Responses	M x A	1	0.0140	15.35**	0.222
Non-Responses	Error	512	0.0009		

Notes: *$p < .05$

**p<.001

RT=Reaction Time. M=Minute. A=Alertness.

Separate ANOVAs were computed for each dependent measure of interest, to assess the relationship between minute within task and mean daily predicted alertness from the Jewett and Kronauer (1999) model. The mean daily alertness values were calculated on the baseline day by averaging the biomathematically predicted values between 8:00am and 10:00pm (inclusive) at two-hour intervals. The remaining three days were calculated similarly but by averaging the values between 12:00am and 10:00pm (inclusive) at two-hour intervals.

Table 2. Correlation and root mean square deviation (RMSD) values for comparisons of the ACT-R model results to the human data.

	Correlation	RMSD	n	p
False Starts	0.89	0.021	40	$p < .001$
Median Alert Reaction Time	0.87	7.0 ms	40	$p < .001$
Lapses	0.94	0.026	40	$p < .001$
Non-Responses	0.75	0.020	40	$p < .001$

Note: The statistics aggregate across all days of total sleep deprivation. The RMSD values are proportions for false starts, lapses, and non-responses, and milliseconds for median reaction time.

Figure Captions

Figure 1. Best-fitting parameter values and best-fitting linear regression for each parameter for the model compared to human data on each of 4 days of total sleep deprivation (TSD) and each minute of performance in the Psychomotor Vigilance Test (PVT). Solid lines represent unconstrained best-fitting parameter values, while dashed lines show the regression predictions.

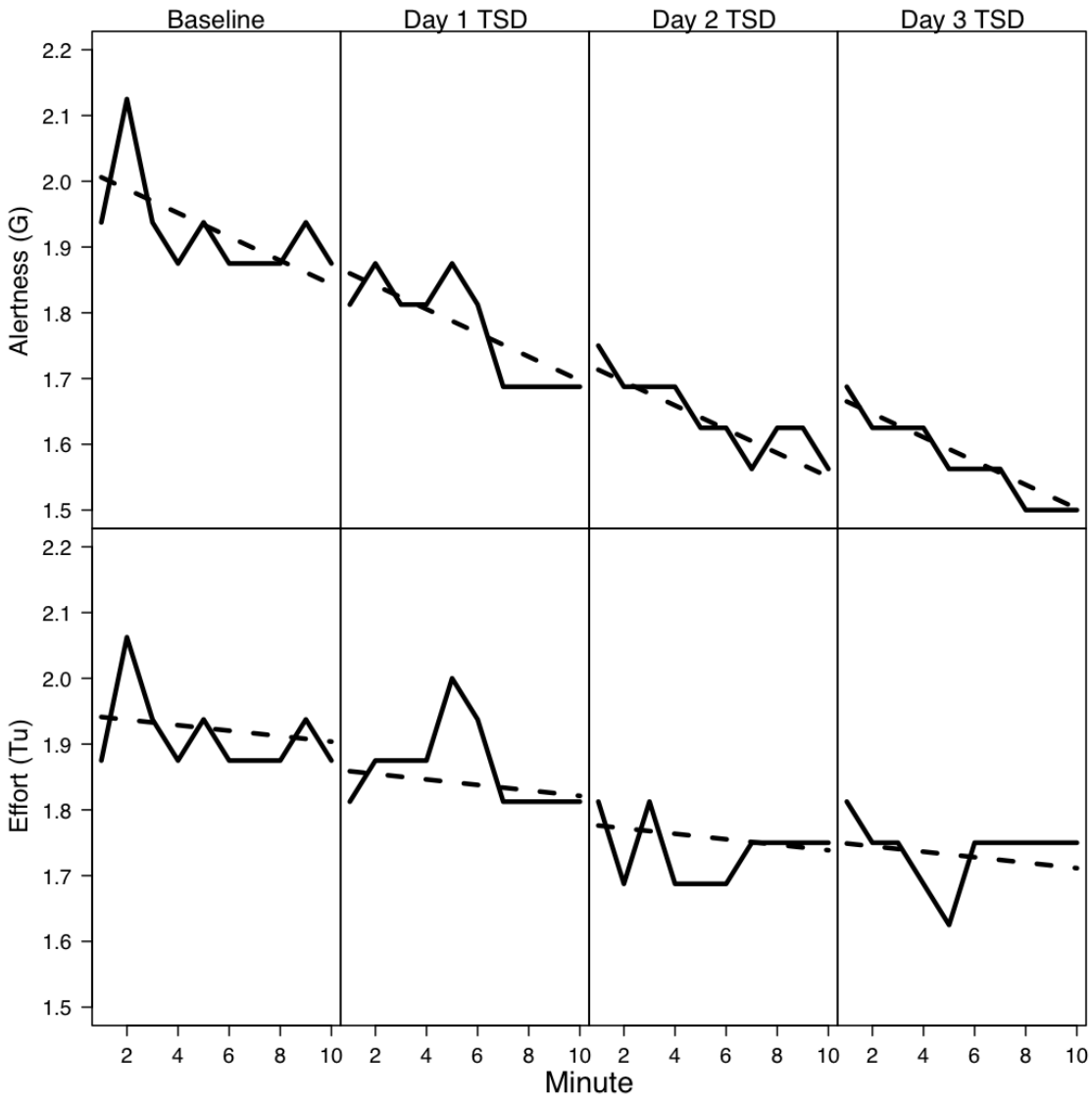

Figure 2. Comparison of model performance to the human data averaged over days of total sleep deprivation (TSD), divided into 1-minute segments within sessions. Solid lines are human data and dashed lines are model data. Data show percentage of responses for false starts, lapses, and non-responses, and median response time in milliseconds (ms) for alert responses.