Two Ways to Model the Effects of Sleep Fatigue on Cognition

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Abstract

We compare how the same cognitive model completes a task within two alternative modifications to a cognitive architecture to represent sleep deprivation. One modification (ACT-R/F) has a module that uses a biomathematical model of the effects of sleep deprivation on performance to drive parameter changes in the architecture that impact behavior and performance. The second, new, modification (ACT-R/Φ) represents the effects of sleep deprivation on physiological systems and has these systems modulate cognition. The model completes the psychomotor vigilance task (PVT) within both ACT-R/Φ and ACT-R/F. We found that the two implementations produced similar response times (means) in simulated days one and two. However, the distribution of the response times across the two days of sleep deprivation varied between models. The ACT-R/Φ model shows a wider distribution in both days 1 and 2 due to an increased and modulating production utility noise that affects its ability to select the correct rules consistently. Though they represent sleep deprivation in different ways, and on different levels, both of these implementations lead us towards a more unified understanding of how sleep deprivation affects our bodies, how we think and behave over time, and how to represent these effects.

Keywords: ACT-R, sleep deprivation, behavioral moderators, HumMod.

Introduction

Extensive empirical research has demonstrated that performance varies in systematic ways over time as a result of time awake, time on task, circadian rhythms, and a variety of other factors that impact the effectiveness and efficiency of cognitive processing (e.g., Gluck & Gunzelmann, 2013). Despite the obvious importance of these factors, collectively referred to as cognitive moderators (e.g., Ritter et al., 2007; Ritter et al., 2003; Silverman et al., 2006), their roles in human cognition, are rarely considered in cognitive science research. Instead, nearly all computational and mathematical models in the literature treat the cognitive system as an optimally functioning information processing machine, which does not waver in its performance over seconds, minutes, hours, or days of performance. As increasingly sophisticated models of various cognitive processes are developed, it is critical to improve the fidelity of moderating functions to capture human performance across the broad range of situations being modeled.

This research focuses on one of these factors—the impact of fatigue brought on by extended time awake. Sleep and circadian rhythms are features of nearly all life on earth, yet their function and impact on cognitive functioning and performance remain poorly understood and infrequently modeled. We examine two ways to model these features.

ACT-R/F

In recent years, some research using computational modeling has begun to expose how human information processing is impacted by fatigue and related factors (e.g., Gunzelmann et al., 2009). This research manipulates parameters in a cognitive architecture, ACT-R, to capture performance changes associated with time awake and circadian rhythms (e.g., Gunzelmann et al., 2012; Gunzelmann et al., 2009), as well as time on task (e.g.,Gartenberg et al., 2014; Gunzelmann et al., 2010). At a theoretical level, the approach integrates a theory of the dynamics of alertness into the ACT-R architecture, creating the ACT-R/F (ACT-R/Fatigue) system. At its core, it demonstrates how fluctuations in alertness can influence performance by impacting the functioning of information processing mechanisms within the cognitive system.

The primary component of the theory is a mechanism associated with fatigue that disrupts ongoing cognitive processing. In the model, the disruptions are implemented as micro lapses, which are small gaps in the information processing in central cognition. These gaps lead to small delays in performance (10's of ms). However, their probability increases with fatigue, which can lead to substantial impairments in performance. In conjunction with this mechanism, there is a compensation mechanism that reduces the likelihood of microlapses, but also increases the likelihood of executing inappropriate, or less useful, cognitive actions.

The mechanisms are implemented in ACT-R's central cognitive module, a production system that coordinates the activity of the other modules to maintain goal-directed cognitive activity (Anderson, 2007). This system operates in cycles, each lasting about 50 ms each. On each cycle, where appropriate actions are identified, one is selected based on a utility calculation, and then the action is taken, provided the utility surpasses the utility threshold. Microlapses occur when the threshold is not reached. In the traditional version

of ACT-R, the model run is terminated when actions fail to exceed the utility threshold. When this situation occurs in ACT-R/F, the cognitive cycle is "skipped," producing a gap of about 50 ms in the goal-directed processing of the model. Because there is noise in the utility calculation mechanism, it is possible that a production will exceed the utility threshold on a subsequent cycle. Compensation is represented in the architecture by reducing the utility threshold. Although this decreases the likelihood of a microlapse, it also increases the probability that actions with a lower utility will be selected and executed in the model.

To control the dynamics associated with fluctuations in alertness, a biomathematical model of alertness was integrated into ACT-R as a new module. The biomathematical model is described in detail in McCauley et al. (2013). Generally, the McCauley model accounts for changes in overall cognitive functioning stemming from time awake and circadian rhythms, incorporating the two-process theory of alertness (Achermann & Borbély, 1992).

The biomathematical model produces a numerical estimate of fatigue. The function of the module is to connect the numerical output of the biomathematical model to parameters in the ACT-R architecture related to the information processing mechanisms that are hypothesized to be affected by fatigue. For instance, within central cognition biomathematical model outputs, F, influence the utilities of candidate actions and the utility threshold. This is achieved by computing scaling factors, FP and FT, for the utility of productions and the threshold as follows:

Eq. 1 $FP = 1 - a_{FP}F$
Eq. 2 $FT = 1 - a_{FT}F$

In these equations, a_{FP} and a_{FT} are parameters that define the slope of a linear mapping of fatigue values to the utility and threshold scalars, respectively. FP and FT are constrained to be between 0 and 1. The scaling factors, in turn, influence the utility values and threshold in ACT-R:

Eq. 3 $U_i' = a_{FP}FU_i$
Eq. 4 $T_i' = a_{FP}FT_i$

Here, Ui' is the computed utility value for production (i), and T_i' is the utility threshold used to determine if the selected production is executed. These mechanisms have been demonstrated to capture in detail changes in human performance on a sustained attention task with sleep deprivation. The model predicts the response time distribution of individual participants, at a level of precision that is equivalent to the detail provided by a diffusion model of the same task (Walsh et al., 2014).

ACT-R/Φ

As more models of cognition and information processing moderators are developed, it will also be important to find a way to tie these separate models together. However, understanding the interactions between moderators can be difficult as the models are often developed in isolation.

One way to make the modeling of the interactions between moderators and the effects of these interactions on information processing more straightforward and tractable is to model these effects on the physiological, as well as the cognitive, level. Common physiological systems involved in changes in cognitive mechanisms can be used as a basis for understanding the interactions between moderators.

The ACT-R/Φ architecture (Dancy et al., In Press) combines a cognitive architecture (ACT-R) and an integrative computational model of physiology (HumMod; Hester et al., 2011) so that the bidirectional connections between physiological and cognitive systems can be simulated. HumMod is a computational modeling and simulation system that provides an integrative computational model of human physiology, to simulate the interaction between physiological, affective, and cognitive change. The ACT-R/Φ architecture has been used to model the dynamic effects of physiological change due to a psychological stressor (Dancy et al., In Press) and due to affective thirst (Dancy & Kaulakis, 2013). These moderators affect some of the same basic cognitive mechanisms in the architecturesImportantly, because these models have been developed within a single unified architecture, their interactions can also be modeled.

The stress-related pathways between physiological and cognitive processes in ACT-R/Φ are also important for modeling the effects of sleep loss due to the involvement of the Locus Coeruleus (LC) System and Hypothalamic-Pituitary-Adrenal (HPA) axis, which are important in circadian components of sleep (Saper et al., 2005).

The ACT-R/Φ architecture uses variables from the physiological (using HumMod) and affective systems (using theory from affective neuroscience and emotion research) to determine a level of memory-based arousal. Arousal is determined using cortisol, epinephrine, corticotrophin releasing hormone (CRH), and a FEAR value (e.g., Panksepp et al., 2011) as shown in equation 5.

Eq. 5 $Arousal = f(cort) * [\alpha * g(crh) + \beta * h(epi)]$

The equation reflects evidence that cortisol seems to serve more of a multiplicative than additive role in memory-based arousal due to the LC system (e.g., Roozendaal & McGaugh, 2011; Roozendaal et al., 2006). In Equation 5, α and β are parameters that determine the slope of the linear relation between deviation from the normal physiological state; $f(cort)$, $g(crh)$, and $h(epi)$ is a function of the change in cortisol, CRH, and epinephrine (respectively) from the baseline state.

The systems involved in stress and arousal (e.g., the LC system and the HPA axis) have also been shown to modulate both declarative and procedural memory (e.g., see Sara & Bouret, 2012; Schwabe & Wolf, 2013). Thus, this arousal factor affects both declarative and procedural memory in the ACT-R/Φ architecture by affecting related noise parameters,

that is, $:ans$ (declarative memory noise) and $:egs$ (procedural memory utility noise) are both modulated using Equation 6 (A stands for *Arousal*).

Eq. 6 $$noise = \begin{cases} \frac{1-A}{0.5} - 1 \; \forall \; A \leq 0.5 \\ \frac{A}{0.5} - 1 \; \forall \; A > 0.5 \end{cases}$$

Both low arousal (below a nominal value) and high arousal cause an increase in noise, making it more difficult to retrieve chunks (declarative memory) and to select the correct productions (procedural memory). In addition to its effects on procedural memory noise, *arousal* also modulates utility threshold of matched rules when it goes below the nominal arousal value. We chose to alter both noise and threshold in this case because of existing evidence that as neural arousal decreases below basal values (as measured by activity in the LC-system), distractibility tends to increase (e.g., Aston-Jones & Cohen, 2005). One way to interpret this result is that decision utilities are more affected by a noise as neural arousal lowers.

Implementing Biomathematical Models of Fatigue in HumMod

We implemented a mathematical model of the HPA-axis in HumMod to simulate circadian and sleep homeostatic changes in adrenocorticotropic hormone (ACTH) and cortisol. We modified the effect of CRH on ACTH so that ACTH levels show circadian fluctuations; this causes related circadian fluctuations in cortisol levels. A sleep homeostatic variable was also added that directly affects cortisol. This variable represents the direct modulatory effects the SCN can have on cortisol outside of the HPA-axis (e.g., see Saper et al., 2005).

The arousal representation was modified (Equation 7a) to include a neural sleep homeostatic variable that decreases (and has an accelerating decline) as time awake increases. H_{S_N} is a neural sleep homeostatic variable that causes arousal to decrease as the time awake increases. As with equation 5, the parameters α and β are parameters that determine the slope of the linear relation between deviation from the normal physiological state.

Eq. 7

A $$Arousal = H_{S\_N} * f(cort) * [\alpha * g(crh) + \beta * h(epi)]$$

B $$cort = H_{S\_C} + Secretion_{Constant} * ACTHEffect + Degradation$$

C $$H_{S\_C} = \begin{cases} SA_{Mag} * \left(1 - {SA_{Rate}}^{T_S}\right) \; [while \; asleep] \\ WA_{Mag} * \left(1 - {WA_{Rate}}^{Tw}\right) \; [while \; awake] \end{cases}$$

D $$ACTH = Secretion_{constant} * CRHEffect * \sum_{i=1}^{4}\left[\rho_i * \left(Sin\left[\frac{i*\pi*t}{720} - \theta\right]\right)\right] + 1$$

Cortisol (Equation 7-B) fluctuates over the course of the day due to circadian rhythms and a sleep homeostatic parameter (Equation 7-C). ACTH (Equation 7-D) has circadian fluctuations, and this variable directly modulates cortisol secretion via the *ACTHEffect* variable, though the proportion of cortisol secretion that is caused due to ACTH varies by time of day and sleep-wake transition time[1]. In this equation, t (for current time of the day) is represented at minutes.

These equations in the HumMod physiological model create a fluctuating HPA-axis, governed by time of day (assuming a stable entrained normal sleep and wake time) and homeostatic pressure created by time spent asleep or awake. Figure 1 shows changing cortisol levels over the course of two days in the updated model. The model displays a peak in cortisol levels at the point of waking (6am in this case) and a trough at 12am.

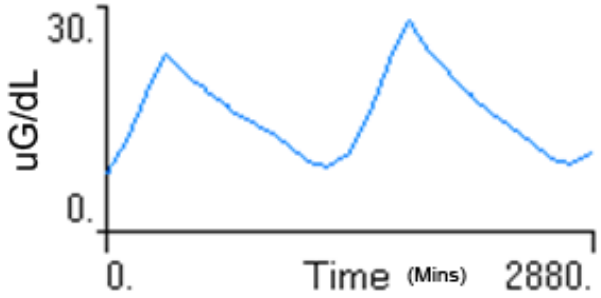

Figure 1. Cortisol levels over the course of two days ([uG/dL]/minutes).

If we cause the model to go two days without sleeping, cortisol in HumMod shows a different, but still circadian, rhythm (Figure 2). The peaks and troughs are roughly at the same positions, but Figure 1 shows a higher minimum and maximum that occur near sleep-wake transitions. The cortisol profile of the sleep deprived model shows a steady increase in peak and trough across days as sleep deprivation time increases.

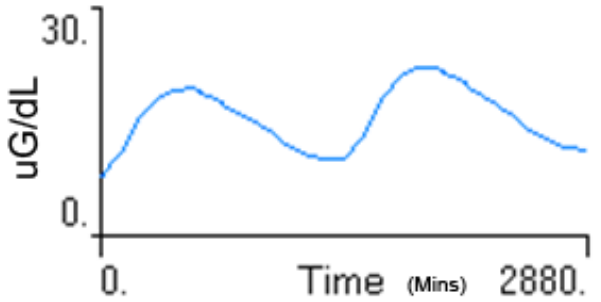

Figure 2. Cortisol levels over the course of two days with the model sleep deprived ([uG/dL]/minutes).

ACT-R/F, ACT-R/Φ, and the PVT

To get a further understanding of how model behavior may change when using these alternative implementations, we implemented a model of the psychomotor vigilance task

[1] Equations 7-C & -D are modified from Thorsley et al. (2012)

(PVT) within both ACT-R/F and ACT-R/Φ. In the task, a millisecond counter is presented at the center of a monitor at a random delay of 2-10 seconds from the previous response. The task is to respond to the appearance of the counter by pressing a response button.

The pervasive use of this task in sleep research makes it an important task for theories of fatigue to address. In addition, the subtle changes to response time distributions of fatigued individuals in the task imposes a critical test of the capacity of a computational theory to make detailed, quantitative predictions about human performance.

The model includes three rules—one each for waiting, attending to a stimulus, and responding to the stimulus. Partial-matching is enabled in the model to allow rules that match some, but not necessarily all, of the rule conditions. Thus, when the rules are affected more by noise, whether by increasing the actual noise (ACT-R/Φ) or lowering the utility values of all of the rules (ACT-R/F), false starts (responding before a stimulus is presented) can occur.

Overall means (and std. dev.) of response times were similar between models for both days 1 and 2: 237.4 (14.84) and 235.1 (15.26) for the ACT-R/ Φ model, and 238.3 (12.85) and 238.4 (11.48) for the ACT-R/F model. Despite the overall similarity, the distributions of means for days 1 and 2 differed between models (Figure 3 and 4).

Figure 4 shows slightly different mean response time distribution between days 1 and 2 with day 1 showing a more uniform density distribution. The increased noise due to physiological change in the ACT-R/Φ implementation caused a wider distribution of response times as compared to the ACT-R/F implementation.

Discussion and Conclusions

Both of the implementations discussed provide a novel way of modeling and simulating the effects of sleep deprivation on cognition, albeit in different ways and on different levels of representation. The ACT-R/F implementation takes a tested biomathematical performance model and applies it to procedural memory in the ACT-R architecture (see also Gunzelmann et al., 2012). Implementing sleep deprivation in ACT-R/Φ required adding circadian rhythms and sleep homeostatic modulation to physiological variables and having these variables modulate cognitive systems.

Comparison of the two architectures

We found that there are similarities and differences between the two approaches. Both approaches include aspects of sleep behavior, and the resulting predictions are similar in how they predict that there are increases and decreases in performance across a day.

They are different in the initial quality of their predictions and their extendibility. ACT-R/F is more accurate in its predictions. ACT-R/Φ is more extendable, in that it would be very feasible to represent in a plausible way how other factors will interact with sleep, such as caffeine.

Including HumMod in ACT-R/Φ raises the question of usability, however. HumMod, while a useful system, is another large system that has to be run and understood. It

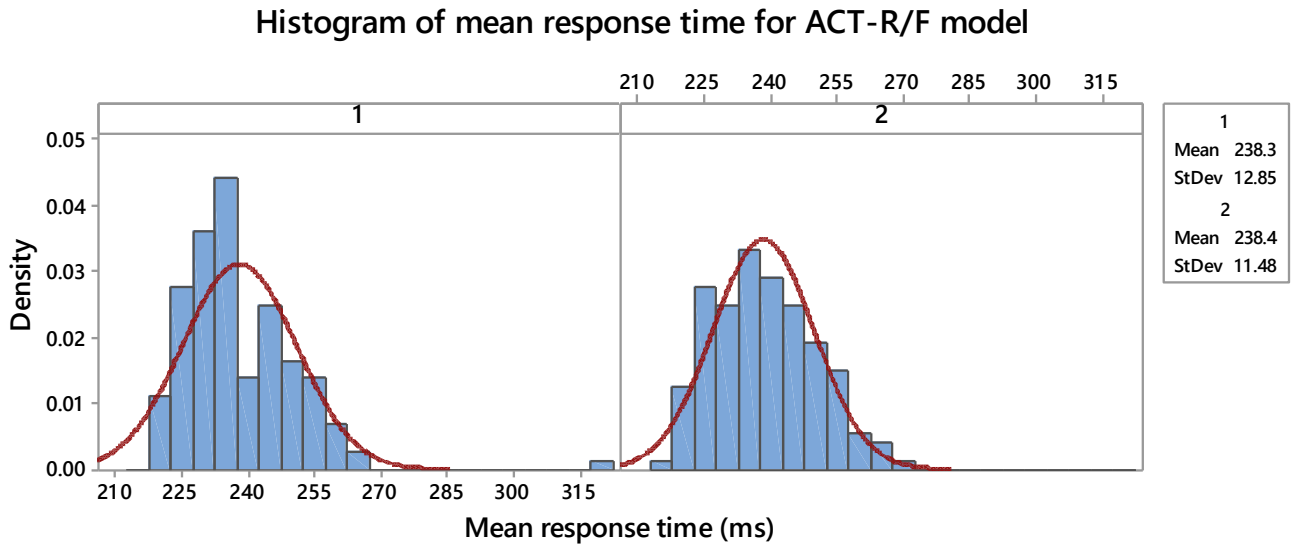

Figure 3. Mean response times of the PVT model used in the ACT-R/F extension. Distribution density shape of show a different pattern between day 1 and 2 than the model using ACT-R/Φ extensions (Figure 4).

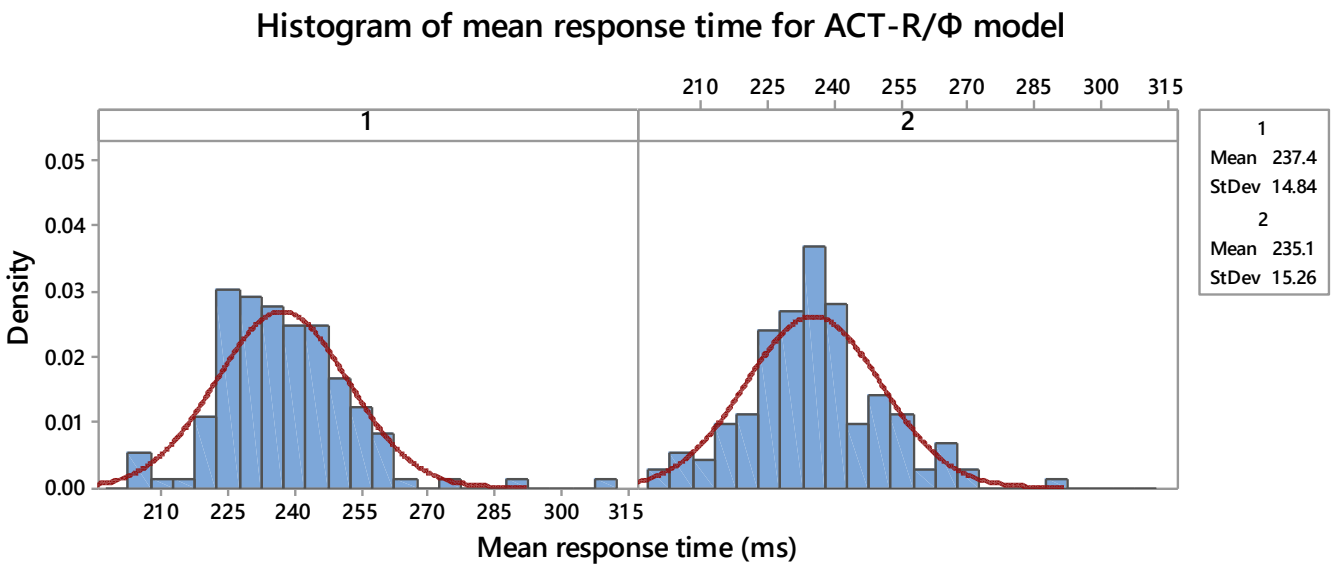

Figure 4. Mean response times of the PVT model used in the ACT-R/ Φ extensions. Distribution density shape of mean values changes between day 1 and day 2.

takes ACT-R/Φ longer to run, and it takes a little more interpretation. Future work will need to explore potential software optimization methods and implementations that can be used with high performance computing (e.g., Harris et al., 2009). It will also be useful to explore possible combinations of the approaches as some mathematical variables in the performance model used in ACT-R/F have been connected to neural representations (McCauley et al., 2013). At this point, we are not able to make recommendations about which is better, but the two approaches are at least different.

This work will also raise new problems about understanding architectures. The combined architecture, that is, ACT-R/Φ, will have further variables and will require further validation, crossing between cognitive psychology and physiology. This will raise new challenges.

Future Work

We will expand upon the new ACT-R/Φ implementation by performing parameter sweeps so that the most realistic model predictions can be found. The performance costs inherent in running the HumMod and expanded ACT-R systems in tandem means that using existing work and theory related to the parameters/variables for performance optimization will be especially important. In addition to continuing to solidify and validate components of these implementations, there are two particular research directions in which this work can expanded: the effects of caffeine on cognition and interactions between sleep deprivation and stress.

As caffeine continues to play a significant role in modern society, it will be important to have a quantifiable understanding of its modulation of cognitive performance over time and to have the same understanding of the ways time of day may interact with this modulation. More recent work in modeling the effects of caffeine on vigilance (Ramakrishnan et al., 2014) and on declarative memory (e.g., Ritter et al., 2009) provide a useful roadmap for continued expansion of the work presented here. Working within HumMod to represent the effects of caffeine on physiology and then and thus on cognition provides a principled way to combine the effects of moderators.

There also exist several parallels between work on sleep deprivation and work on stress systems. It has even been suggested that sleep deprivation can be seen as a form of stress, causing allostatic physiological and behavioral change (McEwen, 2006). Many of the neural systems implicated in the behavioral change due to sleep deprivation and stress systems overlap and are influenced by one another (e.g., the LC system, basal ganglia, and hippocampus). Thus, the generalization of these implementations to the study of stress would be a fairly natural evolution of the work.

Summary

As we continue to study the ways behavioral moderators affect the way we think, feel, and perform during daily activities, it will be vital to keep in mind the effects of these moderators across time, and during different parts of the day. In addition, it will be important that the models and architectures we develop to describe and predict these behavior are generalizable and can be understood in the context of separate, but related cognitive, affective, and physiological behavior. Both of these implementations lead us towards a more unified understanding of how sleep deprivation affects our bodies, as well as the way we think and behave over time.

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