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Decreased Arousal as a Result of Sleep Deprivation

The Unraveling of Cognitive Control

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This chapter discusses recent efforts at developing mechanisms for capturing the effects of fatigue on human performance. We describe a computational cognitive model, developed in ACT-R, that performs a sustained attentional task called the psychomotor vigilance task (PVT). We use neurobehavioral evidence from research on sleep deprivation, in addition to previous research from within the ACT-R community, to select and to evaluate a mechanism for producing fatigue effects in the model. Fatigue is represented by decrementing a parameter associated with arousal in ACT-R, while also reducing a threshold value in the architecture to capture attempts at compensating for the negative effects of decreased arousal. These parameters are associated with the production utility computation in ACT-R, which controls the selection/execution cycle to determine which production (if any) to execute on each cognitive cycle. In ACT-R, this mechanism is linked to the basal ganglia and the thalamus. In turn, portions of the thalamus show heightened activation in attentional tasks under conditions of sleep deprivation. The model we describe closely captures the performance of human participants on the PVT, as observed in a laboratory experiment involving 88 hours of total sleep deprivation.

Until recently, computational cognitive models of human performance were developed with little consideration of how factors such as emotions and alertness influence cognition. However, with increased sophistication in models of cognitive systems, advances in computer technology, and pressure for ever more realistic representations of human performance, cognitive moderators are emerging as an important area of research within the field of computational modeling (e.g., Gratch & Marsella, 2004; Hudlicka, 2003; Ritter, Reifers, Klein, Quigley, & Schoelles, 2004). There is a sense in which this development is both premature and long overdue. Evidence for its prematurity can be found in many of the other chapters in this volume. Cognitive science has yet to unravel many of the intricacies of "normal" human cognition. Therefore, adding additional complexity by including cognitive moderators that influence those thought processes constitutes a substantial challenge. However, cognitive moderators are pervasive in human cognition. It seems essential, therefore, that they be considered in attempts to understand human cognitive functions. If cognitive architectures are to be viewed as "unified theories of cognition" (Newell, 1990), then they must include mechanisms to represent those factors that have substantial modulatory effects on cognitive performance.

This chapter describes an effort to introduce a theory of degraded cognitive functioning into the adaptive control of thought–rational, or ACT-R, cognitive architecture. In this case, the degradation arises from the combined effect of sleep deprivation and endogenous circadian variation. We describe a computational cognitive model that incorporates mechanisms to represent decreased alertness and describe the impact of those mechanisms on the model's performance on the psychomotor vigilance task (PVT), a sustained attention task that has been extensively validated to be sensitive to variation in sleep homeostatic and circadian dynamics, while being relatively immune to the effects of aptitude and learning (Dorrian, Rogers, & Dinges, 2005). Our modeling effort draws on recent research on partial and total sleep deprivation (e.g., Van Dongen

et al., 2003), and leverages recent advances in understanding how sleep deprivation impacts neurobehavioral and brain functioning (e.g., Drummond et al., 1999, 2000; Drummond, Gillin, & Brown 2001; Habeck et al., 2004; Portas et al., 1998).

In the sections that follow, we describe relevant research related to sleep loss. This is followed by a description of the PVT and then the ACT-R model we have developed to perform it. We use the model to demonstrate the effectiveness of our approach for capturing performance decrements as a function of sleep deprivation. In describing the model, we suggest some alternative mechanisms to illustrate how the effects of sleep deprivation can be seen as resulting from impacts to either central control (Type 1 control) or the internal control of functional processes (Type 2 control), which includes processes like memory retrieval or programming motor movements. This distinction constitutes a major theme of this book. Although the mechanistic explanation for the effects of sleep deprivation we have developed is not explicitly defined in terms of Type 1 or Type 2 control, the discussion illustrates how the modeling effort is improved through consideration of this distinction.

Neuropsychological Research on Sleep Deprivation

Unquestionably, sleep deprivation has a negative effect on human performance across a wide array of tasks and situations. Determining the particular impacts of sleep deprivation, both behaviorally and physiologically, has been a significant topic of study in psychological and medical research for quite some time (e.g., Patrick & Gilbert, 1896; von Economo, 1930). Research originally focused on identifying the nature of neurobehavioral incapacitation but shifted to changes in cognitive performance when early studies did not provide conclusive evidence that sleep loss eliminated the ability to perform specific tasks (e.g., Kleitman, 1923; Lee & Kleitman, 1923). Current research directions have been motivated by the desire to uncover the neurophysiologic mechanisms that produce diminished alertness and decrements in cognitive performance, as well as any compensatory mechanisms. Research evaluating behavioral, pharmacological, and technological countermeasures to offset deficits of sleep deprivation has also been a long-standing focus of research (e.g., Bonnet et al., 2005; Caldwell, Caldwell, & Darlington 2003; Caldwell, Caldwell, Smith, & Brown, 2004; Dinges & Broughton, 1989).

At the cortical level, studies have shown inconsistent patterns of regional activation responses to sleep deprivation, depending on the type of cognitive task, its difficulty, and the method used to measure activation (e.g., Chee & Choo, 2004; Drummond et al., 1999, 2001; Habeck et al., 2004). At the subcortical level, a main area that consistently shows sensitivity to sleep deprivation is the thalamus (Chee & Choo, 2004; Habeck et al., 2004; Lin, 2000; Portas et al., 1998). The thalamus typically shows an increase in activation when individuals are asked to perform a task while sleep deprived, relative to performing the task when well rested. For instance, Portas et al. (1998) asked participants to perform a short-duration attention task while activity was measured using fMRI. They found that the thalamus showed increased activation while performing the attention task under conditions of sleep loss, while overall performance (response time) was not significantly different from baseline. From these results, they concluded, "This process may represent a sort of compensatory mechanism. . . . We speculate that the thalamus has to 'work harder' in conditions of low arousal to achieve a performance that is equal to that obtained during normal arousal" (p. 8987). The possibility of such a compensatory mechanism involving the thalamus is discussed further in the section on the computational model later in this chapter.

Biomathematical Models of Sleep Deprivation

In addition to the significant progress that has been made in understanding the neurobehavioral mechanisms of sleep deprivation, researchers studying fatigue have also developed biomathematical models that reflect the influence of sleep history and circadian rhythms on overall cognitive performance, or alertness (Mallis, Mejdal, Nguyen, & Dinges, 2004). Such models provide a means for describing the dynamic interaction of these factors. For instance, Figure 17.1 shows the predictions for one of these models, the circadian neurobehavioral performance and alertness (CNPA) model (Jewett & Kronauer, 1999), for a protocol involving 88 hr of total sleep deprivation. The circadian rhythm component of the model is responsible for the cyclic nature of the predictions and increased sleep loss is responsible for the overall decline across days.

Although there is room for improvement in all current biomathematical models of performance (Van Dongen, 2004), the models have potential value for predicting global changes in alertness over time in a