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THEORETICAL REVIEW

Deconstructing and reconstructing cognitive performance in sleep deprivation

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SUMMARY

Mitigation of cognitive impairment due to sleep deprivation in operational settings is critical for safety and productivity. Achievements in this area are hampered by limited knowledge about the effects of sleep loss on actual job tasks. Sleep deprivation has different effects on different cognitive performance tasks, but the mechanisms behind this task-specificity are poorly understood. In this context it is important to recognize that cognitive performance is not a unitary process, but involves a number of component processes. There is emerging evidence that these component processes are differentially affected by sleep loss.

Experiments have been conducted to decompose sleep-deprived performance into underlying cognitive processes using cognitive-behavioral, neuroimaging and cognitive modeling techniques. Furthermore, computational modeling in cognitive architectures has been employed to simulate sleep-deprived cognitive performance on the basis of the constituent cognitive processes. These efforts are beginning to enable quantitative prediction of the effects of sleep deprivation across different task contexts.

This paper reviews a rapidly evolving area of research, and outlines a theoretical framework in which the effects of sleep loss on cognition may be understood from the deficits in the underlying neurobiology to the applied consequences in real-world job tasks.

Dissociating components of cognition in sleep deprivation research

Introduction

Sleep deprivation affects performance outcomes across a wide range of performance tasks and cognitive domains.[1–4] Sleep deprivation has been cited as a factor in many accidents in transportation and industrial settings,[5,6] and the costs to society are substantial in terms of property damage, lost productivity, personal injury, and death.[7] Significant research efforts are devoted to mitigating the adverse consequences of sleep deprivation in operational environments. These efforts are hampered, however, by limited knowledge about the effects of sleep loss as a function of job task. The literature is filled with evidence regarding differential effects of sleep deprivation on different performance tasks.[1–4,8,9] Furthermore, there is considerable trait inter-individual variability in responses to sleep loss,[10] which also differs from one performance task to another.[11] The mechanisms underlying these task-specific, differential responses are poorly understood. This makes it challenging, in general, to accurately predict the quantitative impact of sleep loss on performance for any specific job task.[12]

Against this background, it is important to recognize that cognitive performance is not a unitary concept. Rather, cognitive performance involves a number of component processes, such as stimulus detection, information encoding, working memory, motor action, etc. These component processes may be differentially affected by sleep deprivation. When examining the effects of sleep deprivation on performance, therefore, it is crucial to consider how

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Abbreviations

ACT-R	adaptive control of thought – rational
ATP	adenosine triphosphate
DLPFC	dorsolateral prefrontal cortex
EEG	electroencephalogram
fMRI	functional magnetic resonance imaging
GABA	gamma-aminobutyric acid
IGT	Iowa gambling task
IL1	interleukin-1
PVT	psychomotor vigilance test
RT	response time
SRSs	sleep regulatory substances
TNF	tumor necrosis factor
VLPO	ventrolateral preoptic nucleus
VMPFC	ventromedial prefrontal cortex

sleep loss affects each of the underlying cognitive processes and how those cognitive processes interact to affect overall performance. After disentangling the effects of sleep deprivation this way, computational modeling may be used to reconstruct and predict sleep-deprived performance across different task platforms. This paper reviews recent scientific advances that contribute to our understanding of cognitive impairment under conditions of sleep loss in new ways, with relevance both for the laboratory and for operational settings.

Task impurity

Studies investigating the effects of sleep deprivation on human cognition have traditionally focused on global task performance outcomes. This has yielded inconsistent findings regarding the effects of sleep loss on performance, both across and within different cognitive domains.[4,13] The heterogeneity of cognitive effects of sleep deprivation may be due at least partially to the "task impurity" problem,[14,15] which refers to the intertwined involvement of multiple cognitive processes during performance of a task.

The potential for task impurity to affect the ability to draw conclusions about sleep loss and cognition can be illustrated with currently popular research on decision-making using the Iowa gambling task (IGT).[16] Decision-making on the IGT involves the selection of cards from decks that are associated with variable gains and losses (shown in terms of hypothetical amounts of money), which are *a priori* not known. Good decision-making on this task requires subjects to learn to choose card decks that provide occasional small gains with infrequent small losses (good decks), instead of card decks that offer large gains with more frequent large losses (bad decks). However, sleep-deprived individuals choose higher-risk decks and exhibit reduced concern for negative consequences when performing tasks like the IGT.[17,18]

The IGT has been of particular interest for two reasons. First, performance on the task is diagnostic of decision-making problems experienced by patients with injury to the ventromedial prefrontal cortex (VMPFC).[19] Second, the IGT provides a context for analyzing decision-making based on the interplay of two distinct aspects of cognition: cold (i.e., rational) and hot (i.e., affective).[20,21] The general presumption has been that poor IGT performance reflects the absence or improper weighting of affective factors, and that this applies to a wide range of decision-making problems in everyday life.[22]

The IGT has become a popular general-purpose assay of decision-making. Global performance on the IGT, i.e., number of good and bad deck choices at asymptote, is often used as an index of the quality of decision-making in general. The variety of settings in which the IGT has been used includes the study of decision-making under conditions of sleep deprivation, where increased bad deck choices have been documented.[17,23] It is tempting to conclude that sleep deprivation produces poor IGT performance by interfering with affective factors biasing decision-making. However, the mechanisms at work during decision-making on the IGT turn out to be far more complex.[24,25]

Interplay of hot and cold cognition

Converging evidence indicates that IGT performance is based on multiple interacting circuits being employed to integrate hot and cold information in decision-making. Consistent with this notion, there is evidence that in healthy subjects the IGT measures two kinds of decision-making. The early trials assess decision-making about ambiguous risks, and later trials assess decision-making about known risks.[26,27] The influence of executive functions managed by the dorsolateral prefrontal cortex (DLPFC) becomes more pronounced as explicit knowledge is gained and risks become more precisely known. Thus, decision-making in the IGT involves continuous and dynamic processes, including the VMPFC and DLPFC circuits that support hot and cold cognition.[28–31]

Overall performance impairment on the IGT under conditions of sleep deprivation, or any other challenge, does not point unambiguously to the causal factors for the impairment. This is not to say that the IGT is an inferior decision-making task, but rather is a recognition that decision-making performance on the IGT must be analyzed in detail to understand why it is optimal in some circumstances and suboptimal in others. In offering the IGT as a clinical tool, Bechara defined its construct validity as an assessment of decision-making capacity without claiming that decisions in the task depend only on affective guidance.[32] Nevertheless, in both experimental and clinical uses of the IGT, the composite score over all trials is typically the only index used, and the pervasive assumption is that performance reflects hot cognition.[33–35] Yet, overall performance on the IGT, and other decision-making tasks, is based on multiple component processes involved in cognition, including cold cognition. It is important to recognize that these component processes may not respond to sleep deprivation in a unitary way.[15,36,37]

The complexity of IGT decision-making recommends caution in the interpretation of results from the diverse decision-making paradigms currently used in sleep research, particularly those that potentially involve complex interactions between cold and hot cognition. For example, decision-making in social exchange paradigms involving social–economic games such as the Ultimatum Game, the Dictator Game, or the Trust Game, have been widely used in cognitive neuroscience[38–40] and are presumed to have a significant affective component governing rejection of unfair offers.

A recent study found that following sleep deprivation, subjects increasingly rejected unfair offers in these games even when this resulted in greater personal losses of monetary distributions.[41] The inference was made that sleep loss increased the impact of negative affect, which in turn resulted in more rejections of offers perceived to be unfair. This inference is supported by findings of increased negative affect after sleep deprivation in other experimental paradigms.[42]

Although affective influences were invoked to account for decision-making changes after sleep deprivation, affect was not actually measured in the study.[41] Recent neuroimaging work has shown that judgment in social exchange games is not simply due to affective aversion to inequity. Instead, these decisions are highly context-dependent and involve brain structures, such as those in the DLPFC, that contribute to controlled, deliberative processing.[43,44] Therefore, independent measures of affect should be employed before invoking hot cognitive processes as an explanation for observed deficits.

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Ambiguity in underlying deficits

Other studies of economic decision-making paradigms in which affective processes are presumed to contribute to risk preferences have employed neuroimaging in the context of sleep deprivation.[45,46] One study, involving choices among risky decision-making gambles, showed that after sleep deprivation risk preference shifted from loss avoidance to gain seeking.[47] This shift in preference was accompanied by elevation in VMPFC activity, suggesting that sleep loss biased decision-making toward highest ranked outcomes. A second study examined social and economic exchange values using an experimental paradigm in which subjects could exchange money to view attractive faces.[48] Findings from this study were that sleep deprivation altered VMPFC activation, and that this activation reflected the valuation of social rewards. The alteration in value showed individual differences, in that sleep deprivation produced an increase in exchange value for some subjects and a decrease in exchange value for others, a finding consistent with prior reports of trait-like inter-individual differences in the effects of sleep deprivation.[10]

The overarching point is that a single global outcome measure from a decision-making task cannot unambiguously predict underlying cognitive deficits or performance in other domains after sleep loss. When using any measure of decision-making it is essential to measure both cold and hot cognitive component processes. Ideally, the global measures of decision-making performance should be accompanied by independent measures of cold and hot component cognitive processes. These independent measures of hot and cold cognition would also be valuable in clarifying results from neuroimaging studies.

Component cognitive processes

The problem of task impurity is in some respects compounded when different complex tasks that are assumed to assess the same domain of cognition may depend on somewhat different combinations of component processes. For example, executive functioning, as a cognitive domain, is multi-faceted, made up of a number of separate but associated processes.[49] Latent variable analyses of different executive function tasks have revealed that these tasks vary in the extent to which they load on inhibition, set shifting, and working memory updating abilities.[35,50] If sleep deprivation does not uniformly compromise these component processes, then it stands to reason that studies of sleep deprivation on executive functioning may come to different conclusions depending on the specific tasks used to assess executive function. Reviews of the literature confirm this notion.[4,51–53]

Evidence consistent with the idea that the relationship between sleep loss and executive functioning varies across component processes has been reported. Friedman and colleagues examined whether developmental patterns of sleep problems, recorded between the ages from 4 y to 16 y, predicted executive functioning in late adolescence.[50] Their study documented that children who had more of an improvement in sleep problems over the years displayed better executive functioning at age 17. Using a statistical model to dissociate components of executive function across several tasks, inhibition and updating but not shifting functions were found to be significantly related to prior sleep problems.

Decomposable performance tasks

A more theoretically driven approach to tackling the task impurity problem is the use of tasks that can be directly decomposed into separate components of interest. Cognitive tasks designed specifically to tease apart the effects of sleep deprivation on distinct cognitive processes are gradually being introduced into sleep research.[36,54–56]

Tucker and colleagues investigated the effects of sleep deprivation on distinct, experimentally isolated components of cognitive performance[36] by utilizing a modified Sternberg task.[57] This task allows for the dissociation of specific working memory processes (working memory scanning efficiency and resistance to proactive interference) from other components of cognition (e.g., encoding a stimulus, deciding on a response, and executing a motor response). In the task, subjects are shown a set of items to be held in working memory, and then a probe item. They respond by indicating whether or not the probe item is in the memory set. The number of items in the memory set varies, and thereby the working memory scanning requirement. Furthermore, when the probe item is not in the memory set, it may have been in the previously shown memory set, which induces proactive interference.

Subjects were randomized to a total sleep deprivation condition or a control condition, and performance was assessed at baseline, after sleep deprivation (or no sleep deprivation in the control condition), and following two nights of recovery sleep. Overall performance on the modified Sternberg task showed impairment during sleep deprivation, as compared to baseline and recovery and compared to controls. However, two dissociated components of cognitive processing on this task – working memory scanning efficiency (see Fig. 1) and resistance to proactive interference – remained at baseline levels.[36] It appeared that only non-working memory components of the task were affected by sleep deprivation.[36,58]

This study demonstrated that deficits observed in overall performance on a working memory task are not necessarily due to specific impairments in working memory processes, but can be due to performance degradation in other components of cognitive processing. This finding challenges an influential theory stating that sleep deprivation is particularly detrimental for cognitive functions assumed to rely on the prefrontal cortex, such as working memory, executive functioning and decision-making.[59]

Resilience of working memory scanning efficiency to sleep deprivation was also observed in a study conducted by Tucker and co-workers in another laboratory,[60] where they additionally found that sleep deprivation degrades different components of cognition than does aging. Further evidence that sleep deprivation does not significantly impair resistance to proactive interference was reported by Cain and colleagues,[56] based on a decomposition of reaction times in the Stroop color-naming task.[61] Such findings leave unresolved whether latent variables statistically derived from multiple complex measures would be pure indices of distinct cognitive processes. The results of latent variable analyses depend not only on the underlying cognitive processes, but also on the selection of tasks and on the experimental challenge during which they are administered. It thus remains unclear whether the inhibition and updating abilities related to sleep problems in the study by Friedman and colleagues[50] effectively distinguished executive from non-executive contributions to cognitive processing.

Cognitive processes and brain function

Although cognitive processes are not necessarily mapped to brain activation along neuroanatomical lines,[62] neuroimaging techniques may be useful to disentangle the cognitive effects of sleep deprivation when combined with clever experimental designs. Some examples of this have already been discussed above.

Another noteworthy example is a functional magnetic resonance imaging (fMRI) study by Chee and Chuah, who used two different versions of a task to dissociate the contributions of attention and short-term memory capacity to performance decline

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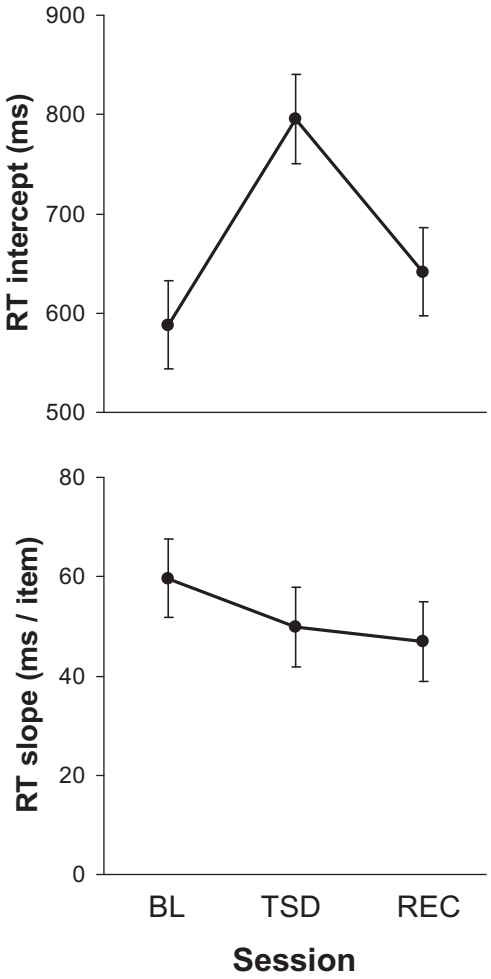

Fig. 1. Performance degradation on a modified Sternberg working memory task during sleep deprivation. The top panel shows the intercept of the linear relationship between memory set size and response time (RT), which measures overall cognitive performance with the exception of the working memory scanning efficiency component. The bottom panel shows the slope of this relationship, which isolates the working memory scanning efficiency component of performance on the task. Means ± standard errors are shown for twelve healthy adults tested in a laboratory during a baseline session (BL), after 51 h of total sleep deprivation (TSD), and following two nights of recovery sleep (REC), at fixed time of day (11:00 h). The results show that performance on the Sternberg task was adversely affected by sleep deprivation, but this was not attributable to impairment of working memory scanning efficiency. Figure adapted from Tucker et al.[36] with permission.

in working memory following sleep deprivation.[63] In a visual short-term memory version of the task, subjects were instructed to remember an array of blocks with between one and eight colors, and then recall whether a probe color was present or absent from the memory array. In a visual attention version of the task, subjects simply responded if a colored block was presented in the center of the array. Sleep deprivation resulted in a significant decline of performance on the visual short-term memory version of the task, with accuracy declining both with increasing array size and sleep deprivation.

The findings from this task version alone would seem to indicate that sleep deprivation caused a reduction in visual short-term memory capacity. However, a performance decline, accompanied by reduced brain activation in the visual pathway, was also observed in the visual attention version of the task, with no effect of array size. This suggested that degraded attention or perceptual processing, rather than reduced short-term memory capacity, may have been the primary cause of the overall decline in working memory performance observed following sleep deprivation.

Further dissociation of perceptual processing is possible by examining event-related potentials in the electroencephalogram (EEG) during task performance.[64] Jackson and colleagues conducted a sleep deprivation study in which performance on a visual reaction time task was measured. Slowing of reaction times and increased errors of omission were observed, accompanied by decreased amplitude of the P300 components of the visual event-related potential over the parietal cortex.[65] The P300 is associated with stimulus evaluation processes such as item categorization and stimulus discrimination.[66,67] Thus, the study results suggested that sleep deprivation selectively affected post-detection aspects of perception, rather than earlier sensory processes.

Cognitive modeling

Dissociation of distinct components of cognition in the context of sleep deprivation has also been pursued with cognitive modeling.[68,69] One promising approach involves the diffusion model (see Fig. 2), which aims to disentangle the cognitive processes involved in making simple two-choice decisions. The diffusion model separates the quality of evidence entering a decision from the decision criteria, as well as from non-decision processes.[70]

As part of the experiment conducted by Tucker and colleagues,[36] subjects performed a two-choice numerosity discrimination task at baseline, after sleep deprivation (or no sleep deprivation in the control condition), and following two nights of recovery sleep. In each trial of the task, between 31 and 70 asterisks were placed in random locations in a 10 by 10 array on a computer screen, and subjects were instructed to decide whether the number of asterisks was greater or less than 50. The diffusion model was fitted to the response times (RTs)[69] to dissociate three distinct aspects of cognitive processing: drift rates (accumulation of evidence entering the decision), boundary separation (evidence thresholds that trigger the decision), and non-decision processes (e.g., information encoding and response execution). Following sleep deprivation, best-fitting model parameters indicated longer drift rates, pointing to reduced ability to effectively extract information from the stimuli presented. In addition, the non-decision components of cognition

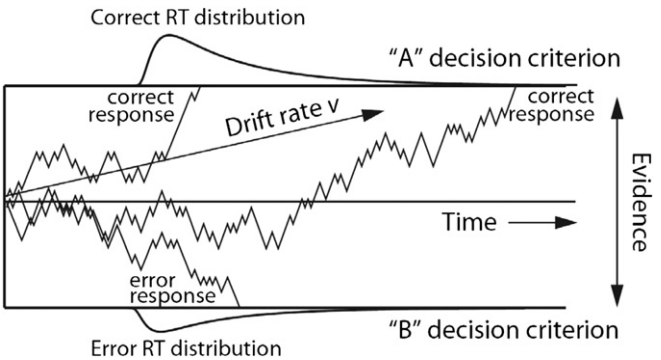

Fig. 2. The diffusion model for two-choice decision-making tasks. The figure shows three sample paths of evidence accumulation following stimulus presentation. These reach the criterion threshold for a correct decision ("A") or error ("B") with different drift rates (v), representing varying ability to effectively extract information from the stimulus. This illustrates variability within the decision process, leading to probability distributions for correct and error response time (RT). The mathematical equations of the diffusion model disentangle evidence accumulation (drift rate) from the criteria triggering the decision, and from non-decision processes such as information encoding and response execution. Figure adapted from Ratcliff and Van Dongen[69] with permission.

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were found to be impaired, which could reflect degraded information encoding and/or response output processes.[69]

During the same experiment,[36] subjects additionally performed a one-choice decision, simple reaction time task called the psychomotor vigilance test (PVT).[71] Ratcliff and Van Dongen[72] demonstrated that the principles of the diffusion model for two-choice decisions also applied to this one-choice task, and that drift rates are affected by sleep deprivation in the same manner for the PVT[72] as seen for the numerosity discrimination task.[69] This suggests that dissociating components of cognition through cognitive modeling is a fruitful approach toward better understanding the effect of sleep loss on cognition across different performance tasks.

Integrating components of cognition through cognitive architectures

Reconstructing task-specific performance

To enable prediction of the effects of sleep deprivation on cognitive performance across different task platforms, a critical further step is to determine how the distinct components of cognition integrate to produce cognitive impairment in sleep-deprived individuals. By carefully tracing the throughput of information from stimulus input to performance output, the various ways that sleep loss can lead to degraded performance may become better understood both qualitatively and quantitatively. Computational models of cognitive performance have been employed to understand and predict task performance as reconstructed from the constituent cognitive processes and the effects of sleep deprivation thereon.[73]

In the context of a research program aimed at minimizing the impact of performance impairment due to sleep deprivation by redesigning task environments, Gunzelmann and colleagues argued that four features are needed to be able to use a computational model to make task-specific performance predictions at an appropriate level of detail.[73] These features are a quantitative theory of the components of cognition and their interactions; an understanding of how the components of cognition are involved in a particular task context; a model of the temporal dynamics of the cognitive effects of sleep deprivation overall; and an understanding of how these temporal dynamics impact on information processing mechanisms in the components of cognition brought to bear on the task context.

Cognitive architectures

Models of the components of cognition and their interactions have been developed in the form of cognitive architectures, which can be seen as formalized, unified conceptualizations of human cognition.[74] Cognitive architectures are computational instantiations of theories regarding the components of cognition and how they interact to drive cognitive performance. One such cognitive architecture is called adaptive control of thought – rational (ACT-R).[75]

ACT-R represents cognition as a set of modules characterizing components of cognition, which contribute to the emergence of human behavior by interacting through a central cognition unit. There are modules in ACT-R that represent visual perception processes, acquisition and use of declarative knowledge, motor actions, etc. Each module contains mechanisms determining the quantitative details of that module's performance. For instance, the declarative knowledge module contains mechanisms to determine the activation (availability) of a chunk of information based on how frequently the chunk has been used in the past, how recently it has last been used, and its relevance to the current task context. Activation levels are used to determine what chunks of knowledge are retrieved from declarative memory, and how long it takes to execute that retrieval. The quantitative mechanisms in ACT-R were developed and validated using empirical evidence from the literature.[75–77]

Central cognition in ACT-R is implemented as a production system. This aspect of the cognitive architecture represents procedural knowledge as condition–action pairs or "productions". The selection and execution of productions is determined by a utility mechanism, which evaluates and compares the usefulness of alternative productions that are applicable in the current context. Utility is defined here in terms of the history of each action leading to desirable outcomes (i.e., achieving goals), reflected in a reinforcement learning algorithm.[78] This mechanism allows utility to evolve over time, which leads to the emergence of adaptive, effective action as experience is gained in a particular task environment. To anticipate the discussion below, we focus on this component of the architecture to demonstrate the ability to capture changes in cognitive processing resulting from sleep loss. (We have also explored the impact of sleep loss in the context of other components of the information processing system, such as declarative memory.[79,80]) See Fig. 3.

The conditions triggering productions in ACT-R are related to information made available by the different modules. For instance, when a chunk of information is retrieved from declarative knowledge, this information is available to central cognition to influence the selection of an appropriate action and promote triggering of the corresponding production. ACT-R also has an "intentional" module that constrains action to a particular goal and an "imaginal" module that can maintain an internal representation of critical aspects of the task context. These parts of the architecture handle information processing capacities that can be considered executive functioning. Acting together, ACT-R's components provide a general account of human cognition (see Fig. 3), which has been used to simulate human performance across a wide variety of tasks and domains.[75,77]

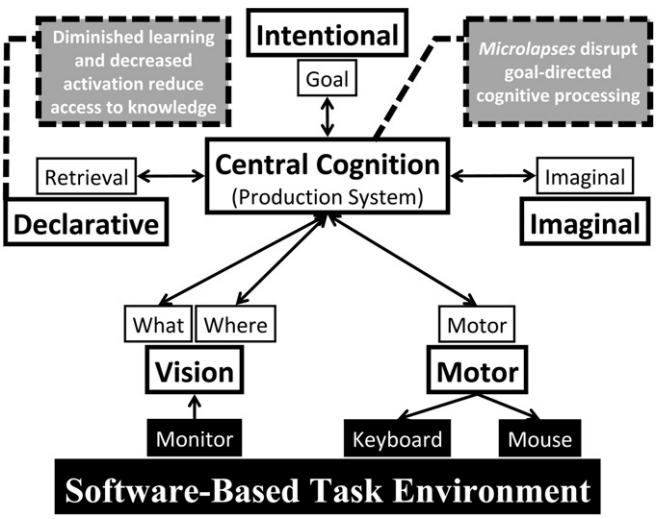

Fig. 3. Representation of the adaptive control of thought – rational (ACT-R) cognitive architecture, showing central cognition and some of the other components of cognition implemented in the system. Modules are shown in large font, with associated information buffers adjacent in smaller font. The external task environment is represented with black boxes and white text. The effects of sleep loss are implemented as changes to the parameters of the underlying mechanisms (see gray boxes), reducing the efficiency and the effectiveness of the information processing system. In the research discussed in the main text, we focus specifically on changes to the parameters associated with central cognition.[91]

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Simulation of task performance

For ACT-R to interact with a task environment, there needs to be an understanding of how the components of cognition represented in ACT-R are used in the task context. Concretely, it is necessary for ACT-R to have appropriate knowledge about the task, which is applied through the operation of the information processing mechanisms in the architecture. The combination of knowledge and mechanisms, which constitutes a "model" in ACT-R, determines how the components of cognition are utilized in performing a particular task. Because ACT-R models are software-based, they can actually be situated in task environments and simulate human performance. That is, ACT-R models produce simulated actions in software-based tasks, leading to moment-to-moment predictions of behavior and performance that can be compared directly to human data.

To simulate task performance under conditions of sleep deprivation in ACT-R, mathematical models quantifying the temporal dynamics of the global cognitive effects of sleep deprivation have been used. A number of such "fatigue models" are available,[81,82] all of which are based at least in part on the seminal two-process model of sleep regulation.[83,84] This model posits that sleep timing and duration are determined by two key neurobiological processes: a homeostatic process aiming to balance time spent awake with time spent asleep, and a circadian process promoting wakefulness during the day and sleep during the night.

The homeostatic process can be seen as providing a drive for sleep that increases across time awake and decreases across time asleep; the circadian process can be seen as a drive for wakefulness that increases during the day and decreases during the night. During a normal schedule of daytime wake and nighttime sleep, the two processes work synergistically to produce alert wakefulness during the day and consolidated sleep at night.[85] Under conditions of extended or nighttime wakefulness and/or travel across time zones, the two processes become misaligned and waking alertness is compromised.[82] The consequences for global performance outcomes are well characterized and can be quantitatively predicted based on equations for the homeostatic and circadian processes.[86–90]

Impact of fatigue on cognitive processes

Current research focuses on how to harness the dynamics of the global cognitive effects of sleep deprivation, as captured in fatigue models, to drive appropriate temporal changes in the effectiveness or efficiency of cognitive processes in the modules of ACT-R. An underlying assumption is that relative changes in global performance over time are present on some scale in one or more of the component cognitive processes, and therefore should also be reflected in the efficacy of particular information processing mechanisms in ACT-R.[91] If this assumption holds and the right ways to connect a fatigue model to the ACT-R cognitive architecture can be identified, then it should be possible to conduct simulations of performance during sleep deprivation that generalize well across task platforms.[73,92]

An illustrative example is computational modeling of the effects of sleep loss on sustained attention performance on the PVT. ACT-R was enabled to perform this simple reaction time task through the implementation of four productions: 1) waiting for the stimulus to appear, 2) shifting visual attention to the stimulus when it appears, 3) executing a response when the stimulus has been encoded, and 4) executing a response independent of the presence of the stimulus. The fourth production allowed the model to produce false starts, and was intended as a stand-in for deficits in processes like motor inhibition, which are not currently part of the ACT-R architecture.

The execution of these productions depended on the task context. The "wait" production only fired when there was nothing present on the screen; attention was shifted only when the stimulus appeared; and a response was made only when attention had been shifted and the stimulus had been encoded. In the context of Fig. 3, when a stimulus appeared, its location became available in the *where* buffer of the vision module. Once this happened, the second production was executed, sending a request to the vision module to move attention to the stimulus. Once the vision module executed the action, a representation of the stimulus became available in the *what* buffer. This then enabled the third production to send a request to the motor module to execute a key press to make a response. The fourth production mentioned above would make the same request, but did not depend on having information encoded, so it could be selected and executed at any time. This happened rarely, however, because that production had a low nominal utility (selectable only through stochasticity in the utility mechanism).

To model the effects of sleep deprivation on PVT performance in ACT-R, a mechanism causing interruptions of cognitive processing in central cognition was implemented. These interruptions were brief (tens of milliseconds), but if they occurred frequently, their combined effects manifested as cognitive lapses (i.e., significantly slowed response times). The likelihood of the interruptions depended on the utility values of the productions in the model – when falling below a given threshold, no actions were taken – and the utility values were linearly coupled to the output of a fatigue model. This made the frequency of the brief interruptions dependent on the predicted level of fatigue. A second mechanism was included to reflect compensatory effort exerted to overcome the effects of sleep loss.[91] This way, the computational model produced simulated performance patterns, characterized as response time distributions, that closely matched human data observed at baseline and through up to 88 h of total sleep deprivation.[93] See Fig. 4.

Generalization to other task platforms

The mechanism involving brief interruptions of cognitive processing in central cognition, coupled to the output of a fatigue model, turned out to also be useful for predicting how sleep loss impacts human performance in other tasks that utilize the same component cognitive processes, such as dual-task performance[94] and automobile driving.[73] In an ACT-R model of driving,[73] brief interruptions of cognitive processing can have a substantial impact on the model's monitoring of lane position and steering, that is, on the model's success (in simulations) to control a vehicle.[95] This ecologically valid behavior is achieved without changing any of the parameters for the cognitive processes instantiated in the ACT-R modules or for the coupling with the fatigue model, demonstrating the potential of the approach of reconstructing task performance from decomposed cognitive processes to predict the effects of sleep loss across different tasks.

Because the ACT-R model of driving (with the cognitive interruption mechanism included)[73] actually drives the car in the simulated environment, its behavior is a reflection of the interaction between information processing mechanisms in the cognitive architecture and characteristics of the task environment. Fig. 5 illustrates this interaction with predictions of the proportion of time spent in a lane violation state during a 10-min driving scenario, as a function of selected driving speed. The predictions were generated using the ACT-R model of driving[73] with all model

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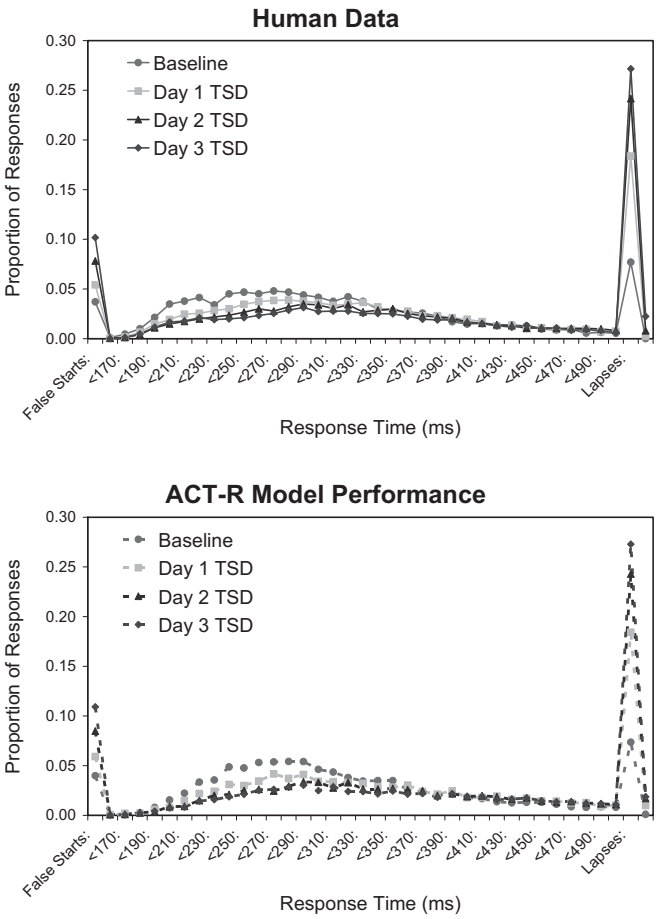

Fig. 4. Psychomotor vigilance test (PVT) performance as observed in an experiment and as predicted with computational modeling. The top panel shows response time distributions, aggregated over thirteen healthy human subjects, observed during 10-min PVT sessions performed at baseline and across three days of total sleep deprivation (TSD) in a laboratory.[93] The data are represented as proportions of responses falling into discrete response time categories. The first (left-most) point in each graph represents the proportion of false starts (i.e., responses made before the stimulus was presented or within 150 ms of stimulus onset). The next set of points captures responses between 150 and 500 ms after stimulus onset – aggregated into 10 ms bins – which are considered to be alert responses. The second-to-last point represents the proportion of responses greater than 500 ms, which by convention are called "lapses".[116] The last (right-most) point (not labeled) shows non-responses, which are trials when no response was made within 30,000 ms of stimulus onset (time-outs). Note the shift to the right of the response time distribution that occurred as a consequence of sleep deprivation, which is indicative of increasing state instability.[93] The bottom panel shows adaptive control of thought – rational (ACT-R) model predictions of the response time distributions as a function of increasing amounts of sleep deprivation. The model predictions are based upon simulating human performance in the task – actual aggregations of response times for individual trials over 100 simulated 10-min task sessions. Note the high degree of correspondence between the human observations in the top panel and the computational predictions in the bottom panel. Figure adapted from Gunzelmann et al.[91] with permission.

parameters held constant, varying only to the speed at which the car was driven. The results were intuitively reasonable, illustrating that computational models of performance developed on the basis of standard performance tasks have the potential to scale up to real-world job tasks and applications.

A challenge for this approach to reconstructing cognitive performance are the complex interactions of mechanisms and potentially large numbers of parameters relating sleep deprivation effects to different processing mechanisms. The research described here illustrates the promise of the approach, but there are still significant questions to be addressed. This will require focused

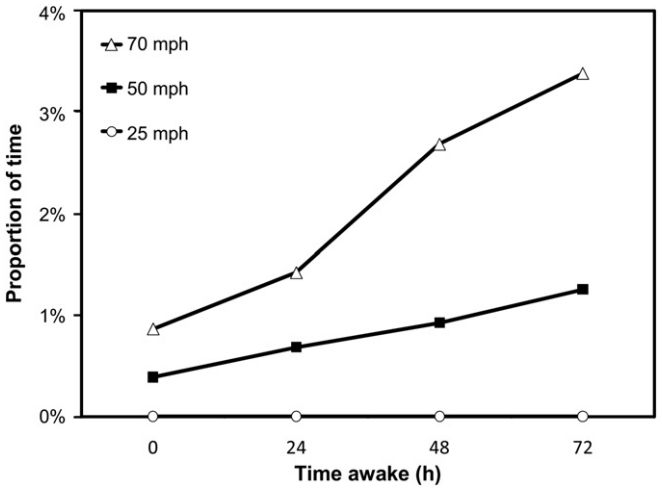

Fig. 5. Predicted probability of lane violations as a function of driving speed and duration of wakefulness. The figure shows the average percentage of time that a portion of a motor vehicle is outside its lane boundary during a 10-min driving scenario, simulated with our adaptive control of thought – rational (ACT-R) model for three different speeds and four different times awake. The ACT-R model's knowledge and all model parameters were held identical to those in the published model[73] for all simulations. The only difference was the speed at which the model drove. Because the model generates data by interacting with the simulated driving environment, its performance predictions vary in accordance with the driving context.

empirical and modeling research to quantitatively understand the temporal dynamics of fatigue, the cognitive processes affected, and the resulting changes in performance and behavior.

A theory on the neurobiological mechanisms underlying task-specific performance deficits

Top-down control

Neuroimaging studies have revealed that the functional connectivity normally observed between cognitive control regions of the parietal lobe and the parahippocampal region is attenuated following sleep deprivation, suggesting a loss of top-down control.[96,97] There is increased thalamic activation in response to visual attention performance during sleep deprivation,[98] which has been interpreted as an attempt of the brain to augment arousal levels in the face of increasing sleep pressure.[99] On the other hand, cognitive (attentional) lapses have been found to be associated with reduced thalamic activation and attenuated frontoparietal activation[100,101] as well as increased activity of the extrastriate cortex and hippocampus[101] and of the frontal and posterior midline regions of the "default mode network",[102] which may collectively reflect a break-down in the ability of top-down control mechanisms to modulate attention and to encode stimuli.[103]

These findings are consistent with the state instability hypothesis[93] – a conceptual framework for explaining the occurrence of cognitive lapses and the shifting of response time distributions in sustained attention performance following sleep deprivation (see Fig. 4). Within this framework, response times become more variable from moment to moment during extended wakefulness due to the penetration of sleep-initiating mechanisms in the face of reduced top-down control of brain state. A reduction of top-down control over attention due to sleep deprivation would seem to suggest that executive control of cognitive functioning is especially sensitive to sleep deprivation. However, as discussed above, it is not clear that degraded performance during sleep deprivation results from problems

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with executive control over attention. There is growing evidence that degraded performance during sleep deprivation may also results from inefficiencies in stimulus detection and encoding[36,101,104] and the bottom-up flow of information,[72,100] which could leave executive control circuits working with an impoverished information flow.

Bottom-up emerging property

The state instability hypothesis does not state specifically what drives the assumed penetrating sleep-initiating mechanisms. Addressing that issue may be important for understanding the effects of sleep deprivation on cognitive performance more comprehensively. One theory for which evidence is accumulating[105,106] posits that sleep/wake regulation takes place locally at the level of cortical columns and other neuronal assemblies,[107,108] making sleep initiation fundamentally a bottom-up emerging (rather than a top-down controlled) property of brain.[109]

In experiments with rats,[110] a whisker barrel (i.e., cortical column) was shown to respond to intensive whisker twitching displaying evoked responses in the EEG that were characteristic of sleep. Yet, neighboring whisker barrels simultaneously exhibited evoked responses characteristic of wakefulness, and the whole organism was functionally awake. The incidence of the whisker barrel in question showing evoked responses indicative of the local sleep state was probabilistic, and increased with the time it had been in the wake state and the intensity of the whisker stimulation. These results indicated that local sleep initiation is a homeostatic, use-dependent, stochastic process.

Preliminary data showed that on a rat-research variant of the PVT,[111] rats trained to lick for a drop of liquid in response to stimulation of a specific whisker and tested while functionally awake showed more performance errors (i.e., failures to lick) when the corresponding whisker barrel displayed evoked responses indicative of local sleep.[109] This observation provided the first experimental evidence that local, stochastic sleep initiation at the level of cortical columns causes performance impairment, and suggested a new explanation for why the effects of sleep deprivation on cognition manifest as performance instability.[112] The data from these experiments further indicated that local sleep initiation occurs in connection with stimulus-driven – and therefore task-related and use-dependent – synaptic transmission. This may explain why sleep deprivation-induced cognitive impairment is task-specific, as the impairment would be expected to depend on which neuronal assemblies are specifically associated with the task context and on the intensity by which they are stimulated by the task.[113] See Fig. 6.

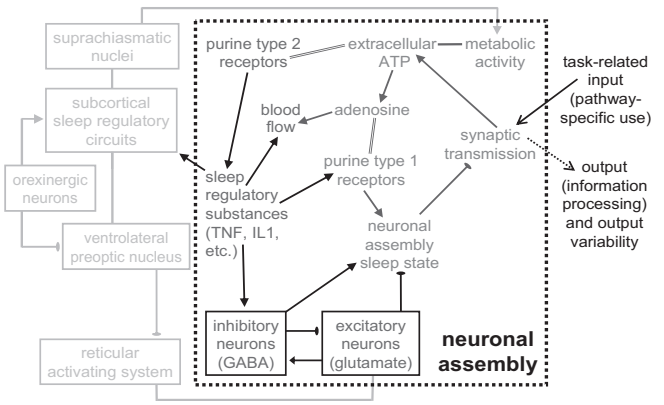

Fig. 6. Simplified representation of a theory on mechanisms of local, use-dependent sleep initiation underlying task-specific cognitive impairment during sleep deprivation. a) Information processing in neuronal assemblies (such as cortical columns) triggers a metabolic, biochemical cascade that promotes the local sleep state (medium gray schematic). When the neuronal assembly is in the wake state and stimulated by input stemming from the cognitive task at hand, it responds with synaptic transmission to process the input signal and generate corresponding output. This triggers release of adenosine triphosphate (ATP) into the extracellular space and increases local metabolic activity. Rapid breakdown of extracellular ATP results in accumulation of adenosine. Binding of adenosine at purine type 1 receptors (adenosine receptors) promotes the neuronal assembly sleep state, during which there is hyperpolarization (changing the evoked potential triggered by the input stimulus) and synaptic transmission is fundamentally altered. This effectively removes the assembly from the coordinated response of the neuronal assemblies involved in the task at hand, resulting in a lapse of information processing. Thus, the local sleep state causes output variability which, at the behavioral level, leads to task-specific performance instability. b) ATP induces release of sleep regulatory substances (SRSs) such as tumor necrosis factor (TNF) and interleukin-1 (IL1) through binding at purine type 2 receptors (dark gray schematic). Continued stimulation of the neuronal assembly causes these SRSs to accumulate and effect an increase in the density of post-synaptic receptors binding adenosine, thereby use-dependently increasing the probability of entering the sleep state. The SRSs also promote the neuronal assembly sleep state through activation of GABAergic inhibitory neurons. The GABAergic neurons inhibit glutamatergic excitatory neurons, which prevents these latter neurons from promoting the local wake state. The SRSs together with metabolic products such as adenosine also influence regional blood flow and thereby oxygen and metabolic nutrient supply. c) Subcortical sleep regulatory circuits coordination and consolidation sleep/wake states across the whole brain, as influenced by the collective neuronal assembly states integrated across the brain through mechanisms involving the SRSs (light gray schematic). Key subcortical sleep regulatory circuits include the ventrolateral preoptic nucleus (VLPO), which can shut down the wake-promoting (e.g., glutamatergic) neurons of the reticular activating system and other systems such as the cholinergic networks of the basal forebrain; orexinergic (hypocretinergic) neurons, through which it has been suggested that compensatory effort to stay awake prevents whole-brain sleep by inhibition of the VLPO; and the circadian pacemaker in the suprachiasmatic nuclei of the hypothalamus, which drives circadian rhythms in background metabolic activity. Whole-brain induction of sleep by the VLPO allows SRS concentrations and receptor densities to be restored, and prevents behavioral interaction with the environment when too many neuronal assemblies are in the local sleep state. Figure adapted from Van Dongen et al.[112] with permission.

Conclusion

The deleterious effects of sleep deprivation on cognitive performance appear to emerge from interaction between the task environment and specific degradations in components of cognitive functioning.[72,73,114] The moment-to-moment variability associated with these degradations and the dynamic nature of most task environments make this interaction complex, resulting in sleep-deprived operational task performance where errors and accidents tend to be rare but their consequences may be severe.[115]

Understanding the nature and underlying mechanisms of task-specific deficits in cognitive performance when sleep-deprived may make it possible to design task environments that inherently mitigate these deficits. In the framework of the theory outlined above, this might be possible, for instance, by strategically distributing task demands over multiple cognitive pathways. This could lessen neuronal assembly use and thereby reduce the likelihood of local, use-dependent sleep initiation that would result in disrupted performance.

In conclusion, it is important to dissociate the component processes of cognitive tasks in order to better understand the effects of sleep loss on performance. Research on the deconstruction and reconstruction of cognitive performance under conditions of sleep deprivation will help to bridge the gap between laboratory research and field application. It may lead to the development of countermeasures targeting the components of cognition most affected by sleep deprivation, which will be invaluable in the many safety- and performance-critical operational environments that offer insufficient opportunity for sleep.

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Practice points

- The effects of sleep deprivation on cognitive performance are stochastic in nature and task-specific;
- To understand the effects of sleep deprivation on cognitive functioning, it is essential to focus not only on overall performance outcomes but also on dissociated cognitive processes underlying task performance;
- Computational models in cognitive architectures are beginning to be used to simulate and predict moment-to-moment performance when sleep-deprived, in a way that generalizes to real-world operational tasks.

Research agenda

- To discern components of cognition underlying task performance in order to better explain cognitive deficits due to sleep loss;
- To further develop performance tasks and cognitive models that allow dissociation of components of cold and hot cognition;
- To investigate inter-individual differences in the degradation of specific components of cognition under conditions of sleep deprivation.

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