A dynamic attentional control framework for understanding sleep deprivation effects on cognition

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Abstract

The cognitive effects of sleep loss are often attributed to compromised functioning of the prefrontal cortex (PFC). However, compromised PFC functioning does not account for well-known effects of sleep deprivation on vigilance. Furthermore, the executive attentional control functions associated with the PFC show considerable variability in the effects of sleep deprivation. Evidence from neuroimaging suggests that sleep deprived people are sometimes able to maintain performance on cognitive tasks by increasing PFC activation of task-relevant circuits and by recruiting new circuits not typically involved in a particular cognitive operation. Still, little is known about how such compensatory processes work on a functional level, or what tradeoffs in processing they may entail. We propose a dynamic attentional control framework to bridge the gap between the evidence on sleep deprived neural circuits and cognitive task performance. We review evidence that shows that the pattern of preserved and compromised task performance can be understood in terms of sleep deprivation’s influence on frontostriatal circuitry such that the ability to maintain task-relevant information in the focus of attention is relatively spared but the ability to update task-relevant information in response to changing circumstances is more negatively affected. This framework helps account for why some tasks are more affected by SD than others, and why individual differences in the effects of sleep deprivation are task-specific.

Keywords

Cognitive control, Cognitive flexibility, Individual differences, Dopamine, Gene polymorphisms, Cognitive impairment, Sleep loss
1 Introduction

The impact of sleep deprivation (SD) on cognition is noteworthy not only because of its ability to compromise performance, but also because of the variability of these effects. Variability is manifest at two levels. First, some cognitive tasks are greatly impaired by SD while others remain relatively unaffected (e.g., Killgore, 2010; Lim and Dinges, 2010). Second, there are large individual differences in the extent to which tasks are susceptible to impairment by sleep loss (Van Dongen et al., 2004). Understanding both dimensions of the variability of SD effects is a challenging research problem, but one that is critical to efforts to mitigate the effects of sleep loss, which can result in catastrophic errors particularly in round-the-clock, high-pressure operational environments (e.g., Honn et al., 2018).

An apparent paradox has emerged from research on the task selectivity of SD effects. Numerous investigators have argued that the prefrontal cortex (PFC) is particularly vulnerable to sleep loss, and the executive attentional control functions often considered to be the province of the PFC provide a potential way to account for a wide range of operational errors (e.g., Harrison and Horne, 2000; Drummond et al., 2000, 2004). However, while the PFC is highly engaged in our most complex and sophisticated cognitive processes, several meta-analyses comparing the magnitude of the effects of SD on cognitive tasks have found that more complex cognitive tasks typically show less of an effect of SD than seemingly simple tasks that assess arousal and vigilant attention (e.g., Lim and Dinges, 2010; Wickens et al., 2015).

Several general approaches suggest ways to resolve inconsistencies between the PFC vulnerability hypothesis and the greater impact of SD on less complex tasks. One suggestion is that SD has a large effect on simple, sustained attention and the effects observed in more complex tasks are consequences of the downstream disruption of sustained attention (cf., Lim and Dinges, 2010; Ma et al., 2015). A second suggestion is that the PFC vulnerability hypothesis is largely correct, but smaller SD effects are observed on many complex tasks because these tasks are more arousing and people engage in compensatory strategies to make their performance under SD as resilient as possible (e.g., Drummond et al., 2004; Harrison and Horne, 2000). From the compensatory processing perspective, the greater influence of SD on many simple cognitive tasks stems from lower levels of arousal and higher level of boredom engendered by such tasks. Of course, a third possibility is that SD has direct and separate effects on both vigilance and PFC-related cognitive processing.

Our goal here is to review current evidence on PFC vulnerability to SD and its effects on task performance and to offer a theoretical framework that holds promise for organizing the relevant literature. Our framework suggests new approaches to understanding both task variability and individual differences in susceptibility to the effects of SD. We focus mainly on the effects of short-term, total SD because the majority of relevant laboratory research uses a total SD manipulation. However, concepts in this approach should be useful for synthesizing research on changes in cognitive functions resulting from sleep restriction and circadian phase. Our theoretical approach, a dynamic attentional control framework, draws on insights from both
the PFC vulnerability hypothesis and the view that complex task performance can break down as a function of the downstream effects of early attentional processes. In addition, we integrate recent data on cognitive control and flexibility to provide a better means of characterizing why some tasks are more affected by SD than others and some reasons why this task variability is manifest differentially at the individual level.

2 PFC functions and SD

In a review examining a broad range of complex tasks, Wickens et al. (2015) found that total SD, sleep restriction, and performance during the circadian night all impaired complex task performance, but these tasks were generally less degraded than simple task performance. However, a major difficulty with interpreting the finding that complex tasks generally have smaller SD effects than tests of simple attention is that describing tasks as complex, or even as PFC-related tasks, is a very coarse level of analysis. By their nature, tasks high in complexity depend on the integration of multiple brain circuits and the coordination of multiple potentially dissociable cognitive processes. Thus, it is unsurprising that data on the vulnerability of complex tasks to SD present many inconsistencies. In this regard, while Lim and Dinges (2010) showed that SD effects on complex tasks are generally smaller than the effects on tests of vigilance and simple attention, their review also demonstrated that the effect sizes in tasks of complex cognition varied substantially across studies depending on the specific task used.

Can inconsistencies in the results on complex task performance be explained by downstream effects of lapses of attention? It is likely that some declines in higher level cognition during SD occur as a result of attentional lapses on the information available for reasoning or complex judgments (cf., Lim and Dinges, 2010; Sturm and Willmes, 2001). In addition, SD reduces activation in the salience network (medial frontal cortex and insula), which is critical to attentional engagement of task critical stimuli (Chen et al., 2016; Ma et al., 2015). However, there is convincing evidence that disruption of early attentional processing is not the whole story. For example, studies using tasks that require complex attentional control have shown large behavioral effects of SD that are independent of sustained attention, as measured by the PVT (Venkatraman et al., 2011; Whitney et al., 2015, 2017). In addition, neuroimaging data across a variety of cognitive tasks have shown that sleep loss alters patterns of activation of the PFC and its connections to the parietal cortex (e.g., Chee et al., 2010; Drummond et al., 2000; Krause et al., 2017; Ma et al., 2015).

Processing in circuits that connect the frontal lobe regions to the basal ganglia, i.e., the frontostriatal pathways, is of particular interest in attempts to understand how SD affects cognition because several lines of neuroimaging evidence suggest that these circuits are involved in compensatory efforts to maintain performance under SD (e.g., Chuah et al., 2006; Drummond et al., 2004; Krause et al., 2017; Nakashima et al., 2018; Schmidt, 2014). Similar claims have been made for
relatively preserved cognitive performance by people with chronic insomnia (e.g., Ballesio et al., 2018). For example, using a go/no go task as a measure of response inhibition, Chuah et al. (2006) found that SD produced tonic decreases in activation of the medial ventrolateral PFC and insula, but individuals who showed phasic increases in the right ventrolateral PFC were resilient to the effects of SD on the task. Another relevant example of evidence for compensatory activation was demonstrated by Drummond et al. (2004) using Baddeley’s grammatical reasoning test. This test of logical reasoning is highly dependent on PFC-related working memory processes, but is frequently cited as an example of a complex cognitive task that is relatively unaffected by SD (Harrison and Horne, 2000; Lim and Dinges, 2010). Drummond and colleagues parametrically manipulated the difficulty of the grammatical transformations used and found several brain areas unique to the SD group in which activation increased with task difficulty. These areas included portions of the PFC already recruited to perform the test as well as frontal and midbrain language centers, which would suggest use of a verbal strategy to assist in task performance.

Given the evidence for compensatory processing while performing cognitive tasks under SD, explaining the variability in SD effects requires an understanding of how people make dynamic adjustments to their information processing in order to preserve complex task performance to the extent possible. While the existing physiological data make a good case that compensatory processing is taking place under SD, the data offer few clues as to how such compensatory mechanisms operate and affect task performance (cf., Giacobbo et al., 2016). As a foundation for bridging between the physiological evidence for compensatory processing and a functional understanding of how compensatory processing operates, we next provide a brief overview of key concepts from the current literature on how attention is managed during cognitive tasks and how adjustments in processing are made dynamically as conditions change.

3 A dynamic attentional control framework

Findings from both animal models and humans, spanning studies of dopaminergic genetic polymorphisms, functional neuroimaging, and brain disorders, support the idea that the frontostriatal circuitry is fundamental to attentional control, decision making, and sleep-wake regulation (Cools and D’Esposito, 2011; Eisenberg and Berman, 2010; Ernst and Steinhauser, 2017; Holst et al., 2017; Qiu et al., 2016). Although we do not fully understand the processes of cognitive control that are implemented in the frontostriatal circuitry, it is clear that a fundamental issue in managing our cognitive system is the tradeoff between cognitive stability and cognitive flexibility. That is, we need stable maintenance of current goals and task relevant information in working memory to preserve information from distraction, but we must also be able to destabilize information in working memory to flexibly shift...
goals and strategies as conditions in the environment change (e.g., Armbruster et al., 2012; Braver et al., 2009; Whitney et al., 2017).

An illustration of the fundamental balancing act of cognitive control appears in the context of real-world operational tasks that are especially prone to errors due to sleep loss. Consider an operator in a power system control room when a major weather event or cyberattack begins. In this situation the operator is flooded with information from different monitoring systems, and the operator is under severe time pressure to make correct decisions about how and where to intervene (Mijović et al., 2019). Initial goals and plans for corrective action must be maintained in working memory in the face of distracting, lower priority information from sensors that are relevant to other, conflicting goals. When prior goals and other information become irrelevant as conditions change, cognitive flexibility is needed. If the operator cannot adapt to the new conditions errors will result from perseveration on previously relevant strategies. Such high-demand contexts are challenging to performance under the best of circumstances, but people who have had inadequate sleep are especially prone to errors when there is high information throughput, time pressure, and the need to process feedback to determine if conditions are changing dynamically over time (Wesensten and Balkin, 2013; Whitney et al., 2015).

The cognitive control processes and neural circuitry fundamental to the stability-flexibility tradeoff are summarized in Fig. 1. These processes have been

![Diagram of OFC and striatal network](image)

**Fig. 1**

The orbitofrontal cortex (OFC) and striatal network play complementary roles in making a series of choices between two response alternatives (r1 and r2). After sufficient feedback, expected outcomes are generated in working memory. Current goals, strategies, and expectations are maintained by dopamine (DA) dependent processing in the OFC. Feedback contrary to expectations serves as a trigger to destabilize working memory and shift responding on future choices. This flexibility is dependent on DA circuits in the striatal network.
studied in the laboratory using a variety of working memory, inhibitory control, and cognitive flexibility tasks that are sensitive to information maintenance, attentional distraction, and flexible updating. The maintenance and updating neural circuits are dissociable, and performance depends on the correct circuit taking precedence in the correct context (see Barkley, 2012; Braver, 2012; Nigg, 2017 for reviews).

The dorsolateral PFC is especially important in the working memory processes of maintaining goal relevant information in the focus of attention and preserving the information from interference by distracting information. Decision making based on choices maintained in working memory is guided by representations of the value of choices for which the ventromedial PFC and related connections are critical. These processes are often studied in the laboratory using simulated gambling tasks (e.g., Hartstra et al., 2010; Lawrence et al., 2009). For example, subjects might be presented with two options on each trial, one of which delivers positive outcomes at a higher probability than the other. Feedback over trials allows subjects to aggregate outcomes from each choice and anticipate the likely outcome of a choice (e.g., Frank and Claus, 2006). This guidance of choice processing and decision making based on expectations is known as top-down attentional control.

Next, consider what happens if the outcome probabilities associated with each choice are reversed, so the formerly superior choice becomes the inferior choice and vice versa. To switch response patterns after this change in contingencies requires updating of information in working memory to avoid perseveration on previously superior choices. A key to quickly adapting to the change in contingencies, i.e., to displaying cognitive flexibility, is that the top-down expectations about choice outcomes allow the subject to quickly recognize the reversal because of the conflict between expected outcomes and actual outcomes (e.g., Klanker et al., 2013). Whereas the original aggregation of outcomes to form an expectation is heavily dependent on working memory and orbitofrontal cortex circuits, the ability to show cognitive flexibility in response to the change in outcome probabilities is dependent on the striatum and basal ganglia circuits (e.g., Izquierdo et al., 2017; Klanker et al., 2013, 2015). Dopamine plays a key role in both orbitofrontal cortex maintenance processes and the flexible response to changing outcome probabilities, with D1 receptors of primary importance in the OFC circuits and D2 receptors in the striatum and associated pathways of primary importance to flexibility (e.g., Klanker et al., 2013, 2015).

Not only are the neurological underpinnings of these cognitive functions distinct, but they appear to depend on a delicate neurotransmitter balancing act. High tonic dopamine (DA) levels are beneficial for stable cognitive functions, such as basic working memory maintenance and applying a repetitive rule. However, higher tonic levels can restrict phasic DA alterations, and phasic DA signaling is beneficial for flexible cognitive functions (Grace, 2000). Thus, high amounts of consistent DA activity can lower transient DA activity. This tradeoff extends to DA relationships in the PFC and striatum, where increases in striatal DA lead to restrictions in prefrontal DA, and vice-versa (Cools, 2015).
Studies of single nucleotide polymorphisms (SNPs) related to dopaminergic activity in the OFC and striatum provide important evidence for dopamine’s role in attentional stability and flexibility. For example, COMT Val158Met codes for the amount of the catechol-O-methyltransferase enzyme in the PFC. This enzyme alters the levels of DA breakdown, with the Val allele resulting in higher amounts of enzymatic activity, translating into lower levels of baseline DA, and the Met allele resulting in lower amounts of enzymatic activity, translating into higher levels of baseline DA (Chen et al., 2004). Studies routinely find that the allele that offers an advantage for working memory and other tasks requiring stable maintenance of information the Met allele, is related to poorer performance on tasks that require cognitive flexibility. Bruder et al. (2005) found a Met advantage on a letter-number sequencing task, while Goldberg et al. (2003) found a Met advantage on an n-back task in the 1-and-2-back conditions, both suggesting that Met alleles are related to enhanced working memory abilities. However, Colzato et al. (2010) found that Met carriers had increased switch costs on a task switching task and Krugel et al. (2009) showed that Val homozygotes have enhanced performance on a reversal learning task, in addition to increased striatal fMRI responses to both positive and negative prediction errors. Thus, the benefits of Met alleles for working memory performance may serve to limit cognitive flexibility.

With regard to the effects of SD on these cognitive control processes, the emerging pattern of evidence supports the hypothesis that SD has a greater effect on cognitive flexibility than on the maintenance of goal relevant information. The more pronounced effect of SD on cognitive flexibility has important implications for understanding both task variability and individual differences in the study of SD. Evidence is beginning to emerge that variations in dopaminergic genes expressed in the frontostriatal circuitry may help explain individual differences in the cognitive effects of SD (e.g., Holst et al., 2017; see chapter “Unraveling the genetic underpinnings of sleep deprivation-induced impairments in human cognition” by Van Dongen, this volume). Studies of several SNPs related to dopamine expression, including BDNF, COMT, and DRD2 C957T, have found evidence of alleles that show resilience to the effects of SD specific to tasks that involve cognitive flexibility (Grant et al., 2018; Satterfield et al., 2018; Whitney et al., 2017).

Wide ranging evidence supports the notion that maintenance of task relevant information in the focus of attention is largely unimpaired by SD, at least within the number of hours of wakefulness typically employed in studies of total SD (e.g., Giacobbo et al., 2016; Tucker et al., 2010; Whitney et al., 2015). Note that while many tasks of working memory show a modest effect of SD on overall performance (Wickens et al., 2015), performance on tests of working memory include both executive and non-executive components. For example, SD produces slower mean reaction times on tests of working memory scanning in which people hold a memory set in the focus of attention and respond whether a probe item is in the memory set (Casement et al., 2006; Cui et al., 2015; Mu et al., 2005; Tucker et al., 2010). However, if working memory scanning is isolated from encoding and response speed by computing the slope of reaction time over memory set size, we see that the process
of scanning working memory is not affected by SD (Tucker et al., 2010). It is the non-working memory components of performance that are affected by SD. Converging evidence for the preservation of working memory capacity under SD was obtained by Giacobbo et al. (2016) who found that SD did not impair performance on the backward digit span test. Similar results have been obtained across other measures of the ability to maintain stable representations in the focus of attention (Casement et al., 2006; Cui et al., 2015; Tucker et al., 2011).

When interpreting the evidence for preserved working memory under SD it is important to bear in mind the evidence for compensatory processing noted above. In some cases, working memory and related executive processes may be preserved though greater activation of relevant circuitry or recruiting additional areas (e.g., Chuah et al., 2006; Drummond et al., 2004). If cognitive stability is preserved, in part through compensatory processing, does the compensatory activation produce tradeoffs that limit other cognitive processing? We do not know the answer to this question, but some interesting possibilities are raised by recent research on the neuroeconomics of cognitive effort (e.g., Westbrook and Braver, 2015). Attentional effort appears to be allocated based on a cost-benefit analysis of resources needed to complete current goals versus the potential benefit of having resources available for flexible use of resources as the context changes. If so, under SD the preservation of cognitive stability could come at the cost of cognitive flexibility.

What is clear from existing evidence is that cognitive flexibility declines substantially when people are sleep deprived. Two types of measures are commonly used to study cognitive flexibility: reversal learning and task switching (e.g., Dajani and Uddin, 2015). The power system control room example provided above to illustrate the distinction between cognitive stability and cognitive flexibility is representative of reversal learning in a natural context. Reversal learning tasks used in the laboratory typically involve learning novel stimulus-response pairings that must be updated based on choice feedback. For example, subjects might learn that choice A in a gambling game often leads to winning money, while choice B more often leads to losing money. These contingencies then change so that choice B is now the better option and subjects must update their choice patterns in order to be successful (e.g., Waltz and Gold, 2007).

When cognitive flexibility is studied with task switching, an individual is required to alternate between multiple stimulus-response sets based on cues that are associated with “rules.” One example of such a task switching procedure is a digit classification task in which subjects are told that one trial cue (e.g., a blue star) means they should report whether a number is odd/even and a different trial cue (e.g., a red circle) means they should report whether a number is greater or less than five (e.g., Wang et al., 2017). Subjects must switch their response patterns based on which cue is present on a given trial. The typical finding is that there is a switch cost manifest as slower or less accurate task performance on trials after a switch in task compared to trials in which the preceding trial involved the same task (e.g., Braver et al., 2003; Logan, 2003).

Whitney et al. (2015) investigated cognitive flexibility during SD using a reversal learning task in which subjects learned from feedback which four digits, out of eight,
they should respond to (go set) and which four digits that they should avoid respond-
ing to (no go set) under strict deadlines. The go and no sets were reversed without
warning after subjects had mastered the original stimulus-response patterns. Rested
subjects were able to detect and adapt to the reversal after 8–12 trials, but SD subjects
had not adapted to the reversal after 40 trials of the reversal phase. Using the digit
classification task switching procedure described above, Couyoumdjian et al. (2010)
found evidence for substantial impairment of cognitive flexibility in sleep deprived
subjects. Under SD, subjects had significantly larger switch costs than rested sub-
jects. Similarly, Slama et al. (2018) found detrimental effects of SD on task switching
but no effect on working memory.

4 “Complex cognition” and SD reconsidered

Although neurophysiological evidence has provided support for the general idea that
higher-level decision making may be preserved under SD in part through compen-
satory processing, the mechanisms, benefits, and costs of such processing have been
left largely unspecified. The dynamic attentional control framework described here
may shed light on how and why some aspects of higher level cognition are more
affected by SD than others. For example, individual economic decision making, i.e.,
decisions involving risk, uncertainty, and relative and absolute value of monetary
outcomes, relies on the neurocognitive processes that are dependent on the PFC
(e.g., Lowe et al., 2017). Because of the presumed vulnerability of the PFC to sleep
loss (Killgore, 2010; Womack et al., 2013), it therefore follows that economic deci-
sion making should be impaired by SD. Nevertheless, laboratory studies have shown
that some decision making tasks that rely heavily on the PFC do not show consistent
SD effects, while other PFC dependent decision making tasks do show SD effects
(Womack et al., 2013).

One illustration of a decision making task that shows a surprising lack of compro-
mise by SD is delay discounting (Green and Myerson, 2004, 2010). Delay discounting
requires individuals to evaluate choices based on differences in delay and magnitude of
options. For example, choice alternatives that are offered typically provide an imme-
diate payoff with a small amount of money versus a delayed payoff with a large
amount of money. A shift in preference for immediate options over more valuable
delayed options is a hallmark of impulsivity in decision making, where people fail
to adequately weigh future consequences of a choice, and it is predictive of risk taking
behavior in real life settings (Dalley and Robbins, 2017; Evenden, 1999; Madden and
Bickel, 2010). Yet, despite the fact that delay discounting tasks engage PFC processes
that are often presumed to be compromised by sleep loss, studies of SD report no
consistent alteration of delay discounting (Acheson et al., 2007; Demos et al., 2016;
Libedinsky et al., 2013). By this well-established laboratory measure, SD individuals
are not more impulsive or risk prone than they are when rested.

In contrast, studies employing the Iowa Gambling Task (IGT; Bechara et al.,
1997; Dunn et al., 2006), which also engages PFC processes, have consistently
reported impaired decision making during SD (Killgore et al., 2006, 2007). The IGT involves gambling with hypothetical gains and losses from four simulated decks of cards. Choices from two of the decks, designated as bad decks, produce frequent large gains, but these gains are offset by occasional larger losses. Choices from the other two decks, designated as good decks, produce frequent small gains, along with infrequent smaller losses. To maximize gains over the course of the task, individuals must learn to choose the smaller, but more reliable, gains provided by the good decks. The IGT shares the probabilistic uncertainty of many behavioral economic tasks, i.e., a good choice does not always result in a good outcome, but it also requires that individuals learn through outcome feedback which choices are advantageous and which choices are disadvantageous. When performing the IGT, SD subjects continue to make choices from bad decks rather than learning to make choices that are advantageous in the long run (Killgore et al., 2006, 2007). This pattern of choices is similar to that found when functions in some PFC regions, such as the ventromedial PFC, are compromised (Bechara et al., 1997).

Why should two different decision making tasks that engage the PFC produce such different patterns of performance impairment during SD? An initial suggestion was that the IGT might activate affective processes that are also compromised by SD and those affective processes are responsible for impaired decision making (Killgore, 2010). This suggestion gained support from research using economic decision making tasks manipulating the risk or uncertainty of choice options. One study examining choices of hypothetical monetary lotteries that contrasted different levels of risk found that SD subjects showed increased preference for risks involving gains, while also showing decreased preference for risks involving losses (McKenna et al., 2007). The change in risk preference was consistent with another study using risky economic decision making and fMRI which reported higher neural activation for gains and lower neural activation for losses (Venkatraman et al., 2007). However, subsequent research has cast doubt on the notion that affective biasing of value is responsible for the compromise of decision making during SD. These recent studies have found that risk preference, including the loss aversion bias that is characteristic of most well-rested people, is not affected by SD (Maric et al., 2017; Mullette-Gillman et al., 2015). Furthermore, it is not clear why delay discounting, which is unaffected by sleep loss, would not engage affective processes that are operative in other economic decision making tasks, as delay discounting is often used as assessment of impulsivity (Madden and Bickel, 2010).

If biasing of the neural valuation signals does not explain when decision making is compromised by SD, perhaps the influence of attentional control processes does. Delay discounting may be less susceptible to SD because it places lower demands on attentional control. Delay discounting tasks fully describe choice options, and do not require updating of information based on past choices. Thus, there is no need for a tradeoff between the stable maintenance of choice information and flexible updating. On the other hand, the IGT requires continual updating of choice information based on choice outcomes. In addition, the IGT has an implicit reversal learning element that requires flexibility in attentional control for good performance. On early IGT
trials subjects are given a long series of good outcomes from what is ultimately a bad deck (Dunn et al., 2006). Almost all subjects develop an immediate preference for this bad deck option based on these early trials. But subjects must overcome this newly developed preference to maximize overall outcomes. The early reversal in contingencies for the bad deck may be essential to group performance differences obtained from the IGT (Fellows and Farah, 2003). Thus, the attentional control requirements of the IGT, requiring a balance of stability and flexibility, may explain why its performance is more readily compromised by sleep loss than is performance on delay discounting, and a variety of other decision making tasks that do place great demands on attentional control. This is not to say, that affective processes and valuation of outcomes do not play in role in SD effects on decisions about risk. Clearly, affective processes are a part of risky decisions, and an important goal for future research is to further understand how affective processes and cognitive control processes mutually influence SD effects on decision making.

The dynamic attentional control explanation is also supported by more recent research on risk and uncertainty in economic decision making. For example, although risk preference may not be changed by SD, the means by which risk are evaluated may change (Mullette-Gillman et al., 2015). More specifically, SD may contribute to use of a satisficing strategy, which reduces demands on attentional resources, instead of relying on a more resource intensive maximizing strategy that is likely to be used when people are well rested (Mullette-Gillman et al., 2015). For example, the attentional control framework predicts that delay discounting during SD could be impaired by imposing a restriction on the throughput of information used to make decisions, such as by a decision deadline, or by otherwise challenging the allocation of attentional resources by dual task or divided attention requirements. The effect of sleep loss could be one of the multiple factors that influence the malleability of risk preference in judgment (Hinson et al., 2003; Lempert and Phelps, 2016).

5 Conclusions

The pattern of variability in SD effects across multiple cognitive tasks is consistent with an asymmetry in the fundamental cognitive control balance between stability and flexibility of information in the focus of attention. People who have experienced significant sleep loss have particular difficulty with flexible updating of task sets and processing strategies especially under fast-paced and dynamically changing conditions. This conclusion is consistent with behavioral evidence, neuroimaging, and dopaminergic genetic data related to the functioning of the frontostriatal circuitry.

In addition to accounting for the pattern of evidence on variability in SD effects, the attentional control framework proposed here suggests that a task whose performance does not appear to be compromised by sleep loss may be made more susceptible to the effects of sleep loss by adding or increasing the attentional control demands of the task. Taking the view that sleep loss is one of multiple situational challenges that can trigger compensatory processes situates SD research firmly
within a long tradition of research in cognitive neuroscience. We contend that research on the selectivity of SD effects on cognition can help us understand how the cognitive system is functionally organized and implemented in neural circuits, just as the selectivity of the effects of brain lesions or other challenges have helped us understand functional and neurophysiological aspects cognition in general (e.g., Miller and Cohen, 2001). The dynamic attentional control framework presented here makes use of recent research on the cognitive neuroscience of attentional control to better understand sleep deprivation effects on cognition. As we learn more about the implications of sleep deprivation’s effect on the attentional stability-flexibility trade-off, we believe that sleep research can become a major contributor to development and testing of general theories of cognitive control.

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