Sleep Less, Think Worse: The Effect of Sleep Deprivation on Working Memory

Steven J. Frenda
Department of Psychology, California State University, Los Angeles, United States

Kimberly M. Fenn*
Department of Psychology, Michigan State University, United States

Sleep deprivation is increasingly common and poses serious problems for optimal cognitive functioning. Here we review the empirical literature on the consequences of sleep loss for working memory. The bulk of the research suggests that sleep is essential for working memory. Sleep deprivation inhibits general attentional and mnemonic abilities and influences neural activation in frontal and parietal cortices, areas critical for working memory. Decreases in task performance are typically accompanied by decreased activation in task-relevant areas under conditions of sleep deprivation. However, there is some evidence that deprivation can lead to increases in activation as a compensatory mechanism allowing for maintained performance. We conclude by discussing what is known about the remediation of these impairments through the use of caffeine and other stimulants.

Keywords: Sleep deprivation, Working memory, Sleep restriction

Experts in sleep medicine recommend that adults obtain an average of at least 7 h of sleep per night for optimal health (Watson et al., 2015). Meanwhile, average sleep duration has decreased dramatically over the past fifty years (Bixler, 2009) and Americans are increasingly likely to maintain a chronic state of sleep debt (i.e., averaging 6 or fewer hours per night; Ford, Cunningham, & Croft, 2015). This is worrisome, in part because both total sleep deprivation (total sleep loss for 24 h or more) and partial sleep restriction (obtaining less than 7 h of sleep on multiple consecutive nights) are associated with a wide range of physiological and cognitive deficits. The physiological effects include impairments in immune function (Cohen, Doyle, Alper, Janicki-Deverts, & Turner, 2009; Prather et al., 2012) cardiovascular health (Meier-Ewert et al., 2004; Wang, Xi, Liu, Zhang, & Fu, 2012), and glucose metabolism (Spiegel, Leproult, & Van Cauter, 1999; Spiegel, Tasali, Penev, & Van Cauter, 2004), to name a few (see Alvarez & Ayas, 2004; Copinschi, 2005; Gallicchio & Kalesan, 2009; Knutson, Spiegel, Penev, & Van Cauter, 2007; Mullington, Haack, Toth, Serrador, & Meier-Ewert, 2009; Patel & Hu, 2008, for reviews). Here, we summarize what is known about the cognitive consequences of sleep deprivation, with an emphasis on working memory.

Sleep deprivation produces wide-ranging impairments in cognitive function, among which are deficits in executive function and attention (e.g., Durmer & Dinges, 2005; Harrison & Horne, 2000). Sleep deprivation increases response times and produces more errors in tasks that require sustained attention, especially under time pressure and as task duration lengthens (Alhola & Polo-Kantola, 2007; Lim & Dinges, 2010). Furthermore, individuals who are sleep deprived have difficulty learning new information and acquiring new skills, and they are less likely to revise and adapt their strategies in response to failures (e.g., Wimmer, Hoffman, Bonato, & Moffitt, 1992). Instead, they tend to perseverate on failed strategies (Harrison & Horne, 1999) and neglect behaviors that they deem nonessential or peripheral. Finally, impulsivity increases and inhibitory ability decreases (Harrison & Horne, 1998a). Taken together, the bulk of the literature makes it clear that adequate sleep is absolutely essential for cognitive functioning.

* Correspondence concerning this article should be addressed to Kimberly M. Fenn, Department of Psychology, Michigan State University, 316 Physics Rd, East Lansing, MI, United States. Contact: kfenn@msu.edu

Author Note

Please note that this paper was handled by the current editorial team of JARMAC.
Given that working memory (WM) is critical to optimal cognitive functioning, it is important to understand the specific WM deficits that are associated with sleep deprivation. To briefly summarize, WM is traditionally understood to have four interrelated components (Baddeley, 2000; Baddeley & Hitch, 1974): a “phonological loop” (for processing acoustic and auditory information), a “visuospatial sketchpad” (for rehearsing visual and spatial information), an “episodic buffer” (for combining various kinds of information into an integrated whole), and a “central executive” which presides over each of the other components. Executive control, specifically, is linked to sustained attentional abilities, sometimes referred to as vigilance.

Working memory performance and its related cognitive operations are typically measured by tasks designed to tap various aspects of memory and attention, including (but not limited to) digit span and word list recall tasks (Quigley, Green, Morgan, Idzikowski, & King, 2000), the N-back task (Choo, Lee, Venkatraman, Sheu, & Chee, 2005), Sternberg-type verbal working memory tasks (Mu, Mishory, et al., 2005; Mu, Nahas, et al., 2005), random generation tasks (Heuer, Kohlisch, & Klein, 2005), the Flanker task (Tsai, Young, Hsieh, & Lee, 2005), and the psychomotor vigilance test (Van Dongen, Baynard, Maislin, & Dinges, 2004). Across hundreds of studies using these and other measures, sleep deprivation has been linked to slowed reaction times in simple attention tasks (Karakorpi et al., 2006), decreased auditory vigilance and visuospatial attention (Bocca & Denise, 2006; Johnsen, Laberg, Eid, & Hugdahl, 2002), and impaired verbal working memory (Chee et al., 2006; Karakorpi et al., 2006). Sleep deprivation has been shown to compromise performance on tasks in terms of both speed and accuracy. For instance, it may simply slow a person’s ability to accurately complete a task (Chee & Choo, 2004; De Gennaro, Ferrara, Cucio, & Bertini, 2001) or compromise accuracy (but not speed; Gosselin, De Koninck, & Campbell, 2005; Kim et al., 2001), or impair both speed and accuracy (Smith, McEvoy, & Gevins, 2002; Yeniad et al., 2013). Thus, sleep deprivation impairs all facets of working memory.

A succinct summary and comparison of the effects of sleep deprivation across tasks is no easy feat, in part because studies vary considerably in terms of sample characteristics, experimental controls, and the duration of sleep deprivation or sleep restriction, to name a few. To illustrate, a number of studies have compared the performance of rested participants to participants who had endured anywhere between 21 h of sleep deprivation (Smith et al., 2002) to upwards of 75 h (Magill et al., 2003). Still others have merely restricted the sleep of participants, as in a study that compared performance in participants who slept either 3, 5, 7 or 9 h every night for a week (Belenky et al., 2003).

Across studies, one general finding is that several nights of sleep restriction results in similar performance impairments as one (or more) nights of total sleep deprivation. For example, one ambitious study compared attention and working memory performance following 14 days of restricted sleep (either 4 or 6 h per night) with performance after either 1, 2, or 3 nights of total sleep deprivation (Van Dongen, Maislin, Mullington, & Dinges, 2003). Participants who endured 3 nights of total sleep deprivation showed significantly worse performance on all tasks than any other group. However, the authors also found a dose response curve in the sleep restricted groups. Participants who obtained only 4 h of sleep per night for 14 days showed levels of impairment roughly equivalent to participants who had endured two nights of total sleep deprivation whereas participants who obtained 6 h of sleep per night for 14 days exhibited impairment on par with those who had endured just one night of total sleep deprivation. An implication of these findings is that the deleterious effects of chronic sleep restriction accumulate over time, eventually matching the more immediate effects of total sleep deprivation. That said, because there are relatively few studies comparing the effects of sleep restriction with the effects of total sleep deprivation, the precise relationship between the amount of sleep loss and the degree of cognitive impairment remains somewhat unclear. It seems, for instance, that sudden total sleep loss may be marginally more deleterious than the same amount of sleep lost gradually over a period of time, though the cognitive consequences of partial and/or chronic deprivation are serious in themselves.

Across these and other studies, extended wakefulness affects a number of cognitive operations, but seems to wreak particular havoc on sustained attention and vigilance, as well as working memory. A key concern emerging from these patterns of findings has to do with the relatively similar, albeit slightly smaller effects of chronic partial sleep deprivation. Of note, although total sleep deprivation can occur in the general population, chronic partial sleep deprivation closely mirrors the kind of sleep loss that is increasingly common in today’s society. This is significant because of the profound real-world costs associated with the loss of these cognitive functions across a variety of high stakes domains. Whether this loss involves the speed with which a professional may complete an important task, or their ability to complete it accurately (or both), these findings warrant significant concern. Unfortunately, there have been significant challenges in summarizing across this vast literature due to a striking heterogeneity of study characteristics and methodologies. Further systematic comparisons of the relative impact of chronic and acute sleep loss are needed, ideally using a variety of methods in a variety of contexts and real-world situations.

How does sleep deprivation produce these deficits? One view suggests that sleep deprivation impairs performance by generally inhibiting the ability to sustain attention and alertness, especially in tasks that do not demand a high level of concentration and vigilant attention (see Durmer & Dinges, 2005; Pilcher, Band, Odle-Dusseau, & Muth, 2007). In part, this view is supported by research suggesting that simple attentional tasks are most strongly compromised by sleep deprivation, whereas more complex and intrinsically engaging tasks are less affected (e.g., Lim & Dinges, 2010). Furthermore, sleep deprivation causes “microsleeps,” or brief, fleeting lapses in brain activity that appear to resemble patterns of activity observed during normal sleep (Priest, Brichard, Aubert, Liistro, & Rodenstein, 2001). Microsleeps can explain some of the functional declines among sleep-deprived participants. However, there is also evidence that impairment occurs even in the periods of time between these lapses (e.g., Dorrian, Rogers, & Dinges, 2005). One key factor that seems to contribute to performance
deficits observed during deprivation is changes in neural function, an area that has received considerable attention in the literature.

**Neural Changes Associated With Sleep Deprivation**

Across many studies utilizing a wide range of tasks, the most consistent neural consequence of sleep deprivation is reduced activity in the frontal cortices (Drummond et al., 1999, 2000; Harrison & Horne, 1998b; Mu, Mishory, et al., 2005; Mu, Nahas, et al., 2005; Thomas et al., 2006; see Harrison & Horne, 2000; for reviews). Consistent with this view, a number of studies have shown that sleep deprivation impairs temporal memory, verbal fluency, and response inhibition (all of which are linked to the prefrontal cortex) but does not lead to impaired performance on tasks that are linked to other brain regions (e.g., recognition memory; see Harrison, Horne, & Rothwell, 2000).

However, in studies using tasks specifically related to working memory, sleep deprivation seems to affect a larger network of neural activity, including both frontal and parietal cortices (Chee & Choo, 2004; Chee et al., 2006). Indeed, there is evidence that basic maintenance of information, which is critical to working memory, is related to changes in parietal activation. For example, studies measuring performance in the Sternberg Working Memory Task (Sternberg, 1966) have shown that performance declines following sleep deprivation were accompanied by a reduction in activation in posterior parietal cortices, bilaterally. Although other areas also show reduced activation during deprivation (including bilateral dorsolateral prefrontal cortex, DLPFC), reductions in these other areas were smaller than the reductions in parietal cortices (Mu, Nahas, et al., 2005). Indeed, several other studies have shown that maintenance of information in working memory is associated with a reduction in parietal activation during conditions of sleep deprivation (Bell-McGinty et al., 2004; Chee & Choo, 2004; Chee et al., 2006; Habeck et al., 2004; Mu, Mishory, et al., 2005).

Maintenance of information is one aspect of working memory, but working memory also involves more complex processes such as the manipulation or updating of information. Other work has investigated the effects of sleep deprivation using more complex tasks that test both maintenance and manipulation in working memory, but results have been somewhat equivocal. Two studies used multiple tasks to investigate the difference in neural activation between simple maintenance of information and maintenance and manipulation of information. Both studies found a reduction in bilateral parietal regions for both types of tasks (Chee & Choo, 2004; Chee et al., 2006). In both studies, the authors used the same task to measure maintenance and manipulation, but there were contrasting effects in frontal regions. The authors evaluated maintenance and manipulation using a task they designed (the PLUS task) in which participants were presented with two letters and asked to think of the letter after the present letter and to maintain that information in memory. Thus, if they present the participants with the letters “B” and “J” then participants should remember “C” and “K” and these letters would later be matched with a probe stimulus (Chee & Choo, 2004, p. 4561). The latter study, Chee et al. (2006) found reduced activity in left prefrontal regions after sleep deprivation. These reductions in activation were not statistically meaningful but were significantly correlated with reductions in accuracy during sleep deprivation. In contrast, the earlier study (Chee & Choo, 2004) found an increase in activation in left DLPFC (Chee & Choo, 2004) after deprivation, possibly reflecting compensatory effort.

Although results vary somewhat across studies, it is clear that the brain is in a very different state during sleep deprivation. Performance in working memory tasks, specifically, has been consistently linked with changes in a fronto-parietal network. The most consistent findings suggest a reduction in activation in parietal areas which is correlated with performance deficits seen during deprivation. The results regarding changes in frontal activation during working memory performance are somewhat mixed. Several studies have found reductions in frontal areas (Chee et al., 2006; Mu, Nahas, et al., 2005), however, other studies have found either no change in frontal activity (Habeck et al., 2004) or an increase in activity (Chee & Choo, 2004) when participants are sleep-deprived. These differences may reflect compensatory effort, a concept that is discussed in the next section. Regardless of specific patterns, it is clear that during working memory tasks, sleep deprivation is associated with changes in frontal and parietal activation and even reduced functional connectivity between areas (De Havas, Parimal, Soon, & Chee, 2012).

**Compensatory Responses**

Although there is widespread evidence for reductions in cortical activation during sleep deprivation, there is also some evidence that in certain tasks, the brain can compensate for impairments brought on by sleep deprivation. Several working memory tasks provide a unique way to investigate this question because the difficulty of the task can be parametrically manipulated such that performance is measured at increasing levels of cognitive load. Under normal conditions, neural activation in task-specific areas increases as the complexity or difficulty of the task increases. However, the results of several studies suggest that sleep deprivation can complicate this relationship. For example, two studies showed decreased accuracy on an n-back task following sleep deprivation (Choo et al., 2005; Lythe, Williams, Anderson, Libri, & Mehta, 2012) and found that sleep deprivation and task difficulty interacted to produce differential activation patterns in certain regions of the brain. Specifically, sleep deprivation prevented (or attenuated) the expected increase in activation due to increasing task difficulty. Seemingly at odds with this finding, one study that used Baddeley’s logical reasoning task found that increasing task difficulty produced even greater increases in activation during sleep deprivation, relative to rested controls (Drummond, Brown, Salamat, & Gillin, 2004). However, in that study, performance was maintained during deprivation. Taken together, these findings suggest that during sleep deprivation, the brain must work harder if it is to maintain the same level of performance.

Consistent with this, several studies have found increased activation in task-related areas when working memory
performance levels are maintained (Drummond, Gillin, & Brown, 2001; Drummond et al., 2004; Drummond, Meloy, Yanagi, Orff, & Brown, 2005) Furthermore, the magnitude of the increase in activation after sleep deprivation has been associated with the stability of performance (Drummond et al., 2005). For example, Drummond and colleagues (2000) found that sleep deprivation led to increases in left prefrontal cortex activity during encoding of a basic verbal memory task (recall and recognition of a list of words), suggesting a compensatory response to sleep deprivation in task-related areas. Moreover, the authors found that sleep deprivation led to additional recruitment of other areas that did not appear active while participants were in a rested state (Drummond et al., 2000). Thus, even though behavioral measures may not show differences between sleep-deprived and rested individuals, the neural mechanisms underlying similar performance may differ considerably.

Individual Differences in Response to Sleep Deprivation

The changes in performance and neural function associated with sleep deprivation are well documented, but it is important to understand whether some individuals are particularly vulnerable or resistant to these effects. A number of studies have investigated individual differences in vulnerability to the cognitive deficits associated with sleep deprivation, finding differences that are stable over time and reliable (Lim, Choo, & Chee, 2007; Rupp, Wesensten, & Balkin, 2012; Van Dongen, Baynard, Maislin, et al., 2004; Van Dongen, Maislin, & Dinges, 2004). In other words, a person’s vulnerability (or resilience) to the effects of sleep deprivation may be determined in part by individual-level traits, and there is some evidence that this may be related to genetic factors (Kuna et al., 2012).

Importantly, although virtually all individuals show some cognitive deficits associated with sleep deprivation, Van Dongen and colleagues (2004) found that sleep-deprived individuals who showed deficits in some aspects of cognitive function did not necessarily show equivalent deficits in others. The authors argue that there are at least three dimensions that may vary independently or orthogonally under conditions of sleep deprivation: subjective evaluation of sleepiness and mood, attention and vigilance, and general cognitive processing (similar to working memory). This distinction is critical because it suggests that performance deficits in certain tasks may not be predicted by performance in other tasks or even by subjective assessments of sleepiness.

Not only are there stable individual differences in performance deficits related to sleep deprivation, but there are also stable changes in neural activation during deprivation. For instance, one study found that during a working memory task, reductions in parietal activations (which correspond to impaired task performance) were consistent across two separate nights of sleep deprivation (Lim et al., 2007). Moreover, individual differences in vulnerability to sleep deprivation may be predicted by neural activation during a rested, waking, state. For example, overall neural activation (Mu, Mishory, et al., 2005) and functional connectivity between frontal and parietal areas (Cui et al., 2015; but see also Yeo, Tandi, & Chee, 2015) during rest all predict vulnerability to deficits associated with sleep loss. These studies all suggest that greater activation and stronger connectivity at baseline predict resilience to the effects of sleep deprivation (i.e., less impairment than those with lower activation or weaker connectivity).

It is important to note that even highly skilled individuals show deficits related to sleep loss and may also show systematic individual differences. In one study, Air Force F-117 pilots were subjected to 38 h of sleep deprivation, which led to significant deficits in their performance in a flight simulation (Van Dongen, Caldwell, & Caldwell, 2006). In contrast to findings described above, here performance ability at rested baseline did not predict the pilots’ performance while sleep deprived. In other words, even highly skilled individuals may be vulnerable to deficits associated with sleep loss, and these deficits can impair performance even on extensively practiced complex skills. Lastly, there remain important unanswered questions about why certain individuals are especially vulnerable (or resilient) to these effects.

Remediation of Deficits Associated With Sleep Deprivation

Given the drastic changes in working memory performance and neural function associated with sleep loss, it is important to understand how these deficits can be overcome. Several studies have investigated the extent to which performance deficits associated with sleep deprivation could be remediated. There is some evidence that bright light (particularly in the morning) can reduce sleepiness during sleep deprivation (Horne, Donlon, & Arendt, 1991; Leproult, Van Reeth, Byrne, Sturis, & Van Cauter, 1997) and remediate performance on both vigilance (Chellappa et al., 2011) and working memory tasks (Gabel et al., 2015). Additionally, a number of studies have tested sleep-deprived participants following the administration of various stimulants—including caffeine, amphetamines, or modafinil (a dopamine reuptake inhibitor that is commonly prescribed for sleep disorders such as narcolepsy). Across a range of working memory and attention tasks, stimulants tend to improve performance during deprivation in rodents (Pierard et al., 2007) and humans (Kamimori et al., 2015; Killgore, Kahn-Greene, Grugle, Killgore, & Balkin, 2009; Lieberman, Tharion, Shukitt-Hale, Speckman, & Tulley, 2002; Wesensten et al., 2002; Wesensten, Killgore, & Balkin, 2005; for review see Bonnet et al., 2005).

Attempts to determine the optimal treatment during deprivation have largely found null results. For example, studies have repeatedly shown that for vigilance tasks, comparisons between caffeine, modafinil or dextroamphetamine (a stimulant that increases dopamine, norepinephrine, and serotonin levels, Killgore et al., 2009; Wesensten et al., 2005) or caffeine and modafinil alone (Wesensten et al., 2002) found no difference between the treatment groups, although all groups performed above placebo. In executive function tasks, there is some

---

1 Although this study did not test working memory directly, it is likely that participants rehearsed the words in an effort to remember them. Also of note, the researchers observed declines in recall performance following deprivation, but no such declines in recognition performance.
disagreement about the relative effectiveness of various stimulants. For example, one study found that in category fluency tests, the stroop task, and the Wisconsin Card Sorting task, there was no difference in performance between participants who were given caffeine, modafinil or dextroamphetamine (Wesenen et al., 2005; but see Killgore et al., 2009 for a more nuanced view).

Given that there is no strong evidence that commercial stimulants have a marked benefit over caffeine, the logical recommendation might be to recommend caffeine administration during periods of sleep deprivation due to more severe side effects and propensity of addiction of stimulants. However, although caffeine may provide immediate remediation from cognitive deficits, it can disrupt subsequent sleep if it is ingested at sleep onset (Landolt, Dijk, Gaus, & Borbely, 1995a; see Roehrs & Roth, 2008 for review) and even if it is ingested in the morning (Landolt, Werth, Borbely, & Dijk, 1995b). Furthermore, routine caffeine ingestion can lead to dependency. This is important because caffeine withdrawal has been shown to result in impaired cognitive performance, reduced arousal, and headaches (James, 1998; Rogers et al., 2005; see Juliano & Griffiths, 2004 for review).

Summary

Working memory performance declines under conditions of sleep deprivation. These performance decrements are associated with a change in neural activation throughout the brain, particularly in the frontal and parietal areas. In certain tasks, performance may be maintained during sleep deprivation but the neural mechanisms underlying similar performance show marked differences when an individual is sleep deprived. Finally caffeine and various stimulants can be used to remEDIATE performance decrements during deprivation. However, these stimulants are not recommended because of the adverse effects they have on subsequent sleep and propensity for dependency. In short, there is no substitute for adequate sleep and a full 7–9 h of sleep must be obtained each night to maintain optimal working memory performance.

Conflict of Interest Statement

The authors declare that they have no conflict of interest.

Acknowledgements

This work was supported by Office of Naval Research grant N00014-16-1-2841 to KM2.

References


Received 11 May 2016; received in revised form 1 October 2016; accepted 3 October 2016

Available online 9 November 2016