

THE EFFECTS OF SLEEP LOSS ON DISSOCIATED COMPONENTS OF  
EXECUTIVE FUNCTIONING

By

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To the faculty of Washington State University:

The members of the committee appointed to examine the dissertation of  
ADRIENNE TUCKER find it satisfactory and recommend that it be accepted.

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Abstract

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The effects of sleep deprivation on executive functioning are poorly understood. A chief source of confusion is that although most executive functions tasks are a mixture of executive and non-executive components most researchers focus on a single global outcome score. Yet, a low score on an executive functions task does not necessarily mean a deficit in the executive function(s) of interest. Thus, the current study included a battery of executive functions tasks that each allowed for the dissociation of executive and non-executive components of performance. For contrast, a non-executive battery of tests known to be sensitive to sleep deprivation was also included. Twenty-three healthy adults (22-40 years; 12 females) spent 7 consecutive 24-hour days in a laboratory with continuous behavioral monitoring. 12 subjects were randomized to 62 hours of total sleep deprivation preceded and followed by two days with 10 hours time in bed; 12 controls had 10 hours time in bed each night. Executive functions testing was performed once at baseline, at 51 hours of total sleep deprivation, and after recovery; the non-executive functions battery was administered about every 2 hours throughout scheduled waking periods of the study. Well-known effects of sleep deprivation were replicated on

psychomotor vigilance, digit-symbol substitution, and subjective sleepiness. However, working memory scanning efficiency, dissociated using a modified Sternberg task, was maintained at baseline levels during sleep deprivation. The ability to overcome proactive interference, dissociated with the modified Sternberg task as well as with a probed recall task, was similarly maintained. These findings challenge a prevailing view that executive functions are specifically vulnerable to impairment due to sleep deprivation. Instead, the current results suggest that specific components of executive functioning are resistant to the effects of sleep loss, and that previous observations of performance impairment on executive functions tasks during sleep deprivation may have been due to deficits in non-executive aspects of cognition.

## Table of Contents

	Page
Acknowledgements.....	iii
Abstract.....	iv
List of Figures.....	viii
Dedication.....	ix
Introduction.....	1
Methods.....	9
<i>Subjects</i> .....	9
<i>Design</i> .....	9
<i>Executive Functions Task Battery</i> .....	12
<i>Modified Sternberg Task</i> .....	13
<i>Probed Recall Task</i> .....	14
<i>Letter Verbal Fluency</i> .....	14
<i>Non-executive Functions Task Battery</i> .....	15
<i>Psychomotor Vigilance Task</i> .....	15
<i>Digit Symbol Substitution Test</i> .....	16
<i>Positive and Negative Affect Schedule</i> .....	16
<i>Karolinska Sleepiness Scale</i> .....	16
Results.....	17
<i>Executive Functions Battery</i> .....	17
<i>Modified Sternberg Task</i> .....	17
<i>Global Aspects of Performance</i> .....	17

<i>Working Memory Scanning in Terms of Speed—Slope</i> .....	17
<i>Intercept</i> .....	19
<i>Overcoming Proactive Interference in Terms of Speed</i> .....	20
<i>Executive Function Components in Terms of Accuracy</i> .....	21
<i>Probed Recall</i> .....	22
<i>Letter Verbal Fluency</i> .....	25
<i>Non-executive functions battery</i> .....	29
Discussion .....	35
Appendix A .....	57

## List of Figures

	Page
<i>Figure 1.</i> Laboratory study design.....	2
<i>Figure 2.</i> Slope and intercept of RT across set size on the modified Sternberg task. ....	19
<i>Figure 3.</i> Cost of overcoming proactive interference on the modified Sternberg task.....	21
<i>Figure 4.</i> Cost of proactive interference (PI) for the probed recall task.....	24
<i>Figure 5.</i> Words generated during the letter verbal fluency task. ....	25
<i>Figure 6.</i> Mean phonemic cluster size and number of switches between phonemic clusters on the letter verbal fluency task.....	27
<i>Figure 7.</i> Lapses made on the PVT. ....	30
<i>Figure 8.</i> Ratings on the KSS. ....	31
<i>Figure 9.</i> Correct responses made on the DSST.....	32
<i>Figure 10.</i> Second method of analysis for the slope and intercept on the modified Sternberg task.....	58
<i>Figure 11.</i> Second method of analysis for the cost of overcoming proactive interference on the modified Sternberg task. ....	59



## Dedication

This dissertation is dedicated to my mother, for providing life-long support,  
and to Alan Munter, for encouraging me to go for it.

## Introduction

Sleep loss is a growing safety concern in modern industrialized societies, as both working hours and commute times are extended (Basner et al., 2007). Sleep loss has been documented to impair performance on simple cognitive tasks such as signal detection and reaction time tests (Durmer & Dinges, 2005). Modern occupational settings, however, increasingly involve complex tasks such as interpersonal communication and creative problem-solving (Overton, 2000) that require executive functions—an umbrella term referring to a family of higher cognitive functions thought to coordinate more basic cognitive functions in order to achieve goals. An important question is whether these executive functions are also impaired by sleep deprivation (Harrison & Horne, 2000). The real-world relevance of this question is illustrated by occupational disasters including the nuclear meltdown of Chernobyl, the grounding of the Exxon Valdez, and the Challenger space shuttle accident, all of which involved complex decision errors for which sleep loss has been cited to be a contributing factor.

Several laboratories have examined deficits in executive functions performance during sleep deprivation (e.g., Binks, Waters, & Hurry, 1999; Harrison, Horne, & Rothwell, 2000; Jennings, Monk, & van der Molen, 2003; Sagaspe, Charles, Taillard, Bioulac, & Philip, 2003; Habeck et al., 2004; Choo, Lee, Venkatraman, Sheu, & Chee, 2005; Heuer, Kohlisch, & Klein, 2005; Mu et al., 2005; Nilsson et al., 2005; Chuah, Venkatraman, Dinges, & Chee, 2006; Drummond, Paulus, & Tapert, 2006; Gottselig et al., 2006; Killgore, Balkin, & Wesensten, 2006; McKenna, Dickinson, Orff, & Drummond, 2007; Venkatraman, Chuah, Huettel, & Chee, 2007). Between studies there is considerable inconsistency as to whether and how executive functions are reported to

be impaired (see Jones & Harrison, 2001). Chuah et al. (2006) and Drummond et al. (2006), for example, found that sleep deprivation impaired the executive function of inhibiting a prepotent response using a go/no-go task, while Sagaspe et al. (2003) reported that inhibition was not impaired during sleep deprivation using a random letter generation task. Similarly, McKenna et al. (2007) reported that sleep deprivation changed behavioral decisions involving risk on a Lottery Choice Task; Venkatraman et al. (2007), using a different gambling task, reported no significant behavioral differences in choices made. Inconsistencies like these have made it difficult to derive a uniform theory of whether and how sleep deprivation affects executive functions.

One potential source of confusion is that there is mounting evidence for the existence of multiple independent executive functions (e.g., MacDonald, Cohen, Stenger, & Carter, 2000; Miyake, Friedman, Emerson, Witzki, & Howerter; 2000). Moreover, as executive functions, by definition, operate on other cognitive processes, any task that targets executive functions also implicates non-executive cognitive processes (i.e., the task impurity problem). For this reason, a low score on an executive functions test does not necessarily mean impairment of the target executive function, but could arise instead from impairment of other processes involved in the task (see Whitney, Jameson, & Hinson, 2004).

One reason, then, that opposing views exist as to whether and how executive functions performance is degraded during sleep deprivation is that the tasks commonly used to measure executive functions do not allow for the dissociation of the various independent executive and non-executive aspects of cognition that contribute to global performance (as remarked by Dinges & Kribbs, 1991; Jennings et al., 2003; and Turner et

al., 2007). The chief contribution of the current study was the selection of a battery of executive functions tasks that each allow for the dissociation of some of these intertwined components of performance. These dissociated components of performance were used to challenge three current theories, discussed below, of how sleep deprivation affects executive functioning.

Our first task was a Sternberg working memory task modified after an adapted version developed by Whitney et al. (2004) in order to allow for the dissociation of two components of executive functioning (cf. Bunge, Ochsner, Desmond, Glover, & Gabrieli, 2001). In the standard Sternberg task (Sternberg, 1966) participants are asked whether a probe item was in a set of items held in working memory. The classic finding on this task is that there is a linear relationship between the number of items that a participant has to remember (set size) and the length of time it takes to respond (reaction time, or RT). Conceptually, the RT at two different set sizes should share all component cognitive processes except for the amount of time needed to scan the additional items in working memory. As such, the slope of the line of RT across set size is thought to be a relatively pure measure of the executive functions component of working memory scanning efficiency. The intercept of this line reflects the amount of time taken for all other component processes involved in this task, which include encoding the probe, deciding whether or not it was a member of the memory set, and executing a motor response (keypress).

In the current modified version of this task, a second executive functions component was assessed. In the classic task, half of the trials are negative, in that the probe item is not a member of the to-be-remembered set, and requires a negative

response. In the adapted version (Whitney et al., 2004), half of these negative trials were manipulated such that although the probe was not a member of the current set, it was a member of the previous set. Because the probe was a member of the previous set, its familiarity results in proactive interference that requires the ability to inhibit a 'yes' response to the negative probe (Jonides et al., 1998). Correctly responding 'no' on these trials requires the ability to inhibit the irrelevant information, that is, to overcome the proactive interference from the irrelevant information. The difference in RT between manipulated and non-manipulated trials provides an index of the ability to resolve proactive interference. As such, our modified Sternberg task allowed for the dissociation of two components of executive functioning: working memory scanning efficiency and the ability to overcome proactive interference.

The probed recall task originated by Bunting (2006) constitutes a second task that allows for the dissociation of the ability to overcome proactive interference. In this task, participants are shown 12 items, one at a time, and are then asked to recall either the first four items, middle four items, or last four items in the correct order. Half of the trials involve lists consisting solely of digits or solely of words. In these homogenous trials, proactive interference builds throughout the list and is highest for the last four items. Recall of these last four items, where interference is maximum, provides a useful index of the ability to remember items in the face of proactive interference (Bunting, 2006). The other half of the trials are heterogeneous such that the last four items of the word trials are replaced by digits, and the last four items of the digits trials are replaced by words. This manipulation releases proactive interference for these last four items, improving recall (Bunting, 2006). Recall of the last items on these heterogeneous lists, then, reflects

all components of the task besides resolving proactive interference. The executive functions component representing the ability to overcome proactive interference can be dissociated by calculating the difference in the accuracy of recall of the last items between homogeneous and heterogeneous lists.

The third and final task in our executive functions task battery was a phonemic, or letter, verbal fluency task. I assessed verbal fluency with the Controlled Oral Word Association Test (COWAT; Benton, Hamsher, & Sivan, 1976), which has been associated with cognitive flexibility and mental set shifting. Participants are given a letter as a prompt and are asked to generate as many words as possible in one minute that begin with that letter. This is repeated for two additional letters. Often the outcome variable used for this task is the total number of words generated for the three letters administered. However, two dissociable components of performance can also be obtained (Troyer et al., 1997). Words on this task are generated in phonemic clusters, or groups of words that are similar in sound. After a cluster of words has been exhausted, participants must switch to a new cluster. Mean phonemic cluster size was used as a measure of non-executive processing while the number of switches between phonemic clusters was used as a measure of executive processing associated with mental set shifting.

Dissociated executive and non-executive components of performance were used to evaluate three current theories of how sleep deprivation affects cognitive performance. The first and most widely-cited view (Harrison et al., 2000) is that sleep deprivation may impair executive functions performance because these tasks selectively engage the prefrontal cortex. A basis for this theory is provided by evidence that sleep loss differentially affects frontal lobe functions. For instance, studies have shown that sleep

pressure, as operationalized by increased theta power density in the electroencephalogram, is most evident in frontal areas during sleep deprivation (e.g., Cajochen, Knoblauch, Kräuchi, Renz, & Wirz-Justice, 2001; Finelli, Baumann, Borbély, & Achermann, 2000). Also, a neuroimaging study using positron emission tomography, which allows greater anatomical specificity, found that sleep deprivation decreases metabolism specifically in the prefrontal cortex (Thomas et al., 2000). Thus, it seems that sleep loss might differentially affect executive functions and the tasks that rely on them. Indeed, Harrison et al. (2000) state that even brief executive functions tests will show impairment during conditions of sleep deprivation if these tests selectively involve the prefrontal cortex.

In this vein, Harrison et al. (2000) hypothesized a parallel between the cognitive impairments seen in sleep deprivation and those seen in aging, as both seem to selectively involve the prefrontal cortex. They considered some performance tasks that specifically involve this area of the brain and reported that performance was similarly impaired in both older participants and sleep-deprived younger participants. The authors thus posited that executive functions performance is selectively impaired in sleep deprivation because of deficits in the functioning of the prefrontal cortex. If this theory is correct, global performance should be impaired during sleep deprivation on all three of the executive functions tasks in our study. Additionally, the executive components of each task should be selectively impaired as compared to the non-executive components during sleep deprivation.

Another theory is that of Doran, Van Dongen, and Dinges (2001), who hypothesized that sleep deprivation affects cognitive performance through impairment of

sustained attention. These authors established that sustained attention is impaired during sleep deprivation as measured by response variability on a simple reaction time task, the psychomotor vigilance task (PVT). They then introduced state instability theory (Doran et al., 2001), which posits that performance impairment during sleep deprivation is caused by the “fundamental increase in moment-to-moment variability of attention brought about by the interaction of the homeostatic drive for sleep, the endogenous circadian promotion of wakefulness, and the compensatory effort exerted by subjects to perform” (p. 261). The authors further stated that this variability in performance due to difficulty sustaining attention would transfer to many other tasks since “attention is a requirement of many goal-directed activities” (p. 264).

The key to detecting performance impairment during sleep deprivation, according to wake state instability theory, is in examining *variability* in performance sampled over a period that requires sustained attention. Two of our tasks, the probed recall task and the letter verbal fluency task, are relatively brief (less than 10 minutes) and according to state instability theory, performance may be intact for these two tasks during sleep deprivation because they do not tax sustained attention sufficiently to observe performance decrements. The modified Sternberg, by contrast, is longer and yields more than double the samples of performance. The intercept of the line of RT as a function of set size, which includes attentional components of performance, should be impaired during sleep deprivation if this theory is correct. The theory does not address executive functioning components per se, and thus can not be used to predict how sleep deprivation will affect performance on the executive functions components of our tasks.



Finally, we considered the controlled attention model proposed by Pilcher, Band, Odle-Dusseau, & Muth (2007), which attempts to unite the two previous theories. This model posits that dysfunction of the prefrontal cortex during sleep loss leads to decrements in performance through impairment of controlled attention. The authors adopt the definition of controlled attention put forth by Kane & Engle (2002) as the “ability to prevent attentional focus from being captured by mental or environmental distractors” (p. 638). The ability to overcome proactive interference is one key determinant of controlled attention (Kane & Engle, 2002). Proactive interference is caused by previously presented information, and thus constitutes a mental (as opposed to environmental) distractor. If the controlled attention model is correct, then one of the current dissociated executive functioning components, the ability to overcome proactive interference, should be impaired during sleep deprivation.

In short, in the current study we used a battery of executive functions tasks that allowed us to isolate components of executive functioning. We investigated whether working memory scanning efficiency, the ability to resolve proactive interference, the ability to shift efficiently between linguistic subcategories, and other elements of task performance are differentially affected by sleep deprivation. Taken together, the changes we observed in distinct cognitive processes challenge prevailing views of how sleep loss affects cognitive functioning. This has important implications for our understanding of the cognitive and behavioral consequences of sleep loss from the laboratory to operational settings and many aspects of daily life.

## Methods

### *Subjects*

In order to be eligible for participation in the study, subjects met the following criteria: age 22–40 years; physically and psychologically healthy, as assessed by physical examination and history; no clinically significant abnormalities in blood chemistry; free of traces of drugs, as assessed by urine screen and breathalyzer; not pregnant; not a current smoker; and no history of moderate to severe brain injury. In addition, participants had to have good habitual sleep, between 6 and 10 hours in duration daily; regular bedtimes, getting up between 06:00 and 09:00; no sleep or circadian disorder, as assessed by questionnaires; and no recent shift work or travel across time zones. Furthermore, participants had to have normal or corrected to normal vision and hearing; and be a native English speaker.

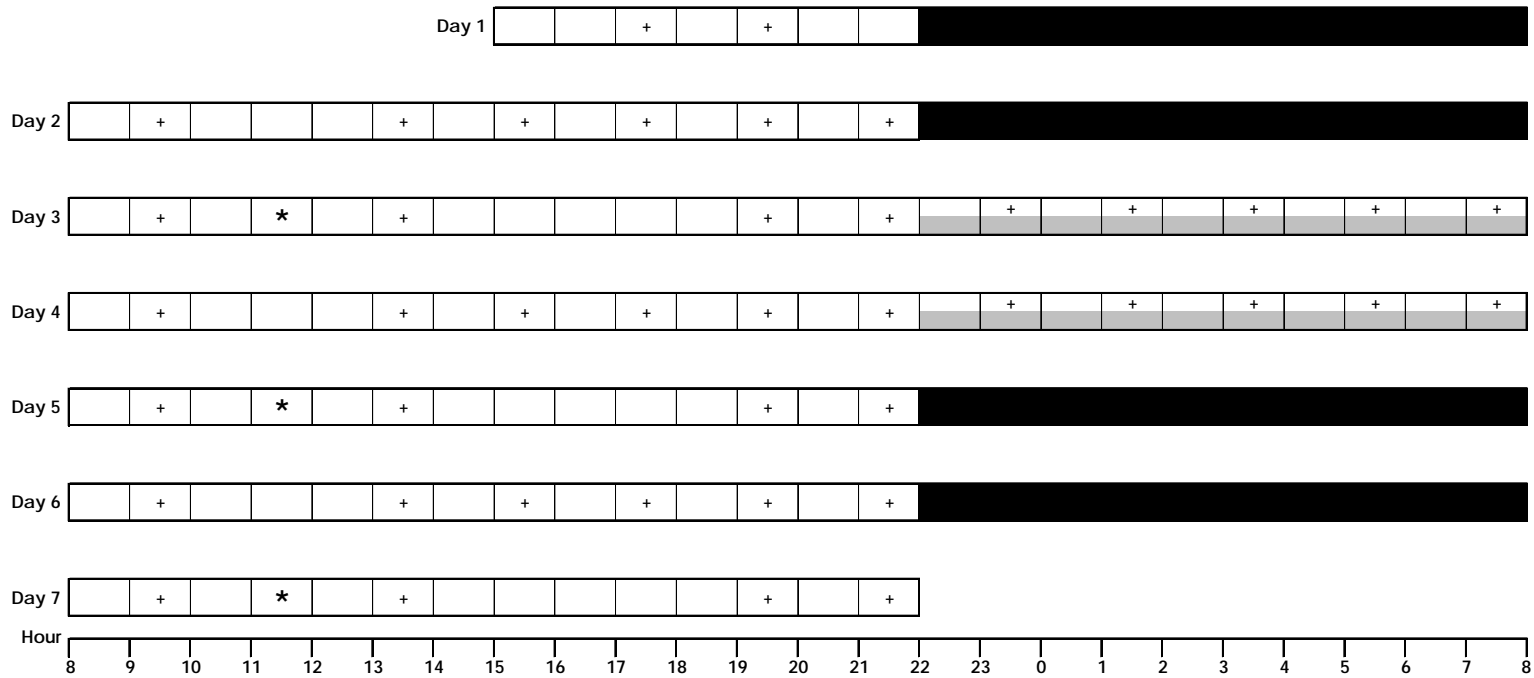
A total of 26 participants passed screening and completed the study. After inspection of polysomnographic recordings of the first night in the laboratory, three participants were removed from the database because of clinically relevant abnormalities in baseline sleep. Thus, data are reported here for a total of 23 subjects (21 Whites: 9 men, 11 women, age range 22–38 years; 1 Black male, age 26; 1 Asian male, age 32). A power calculation performed in advance of the study indicated that this sample size should suffice to detect typical effects of sleep deprivation on cognitive performance.

### *Design*

In the seven days leading up to the laboratory experiment, subjects were not allowed to use caffeine, alcohol, or tobacco products. They were required to keep their regular bedtimes and refrain from napping. Compliance was assessed by wrist actigraphy,

sleep diary, and a time-stamped voice recorder which subjects called at bedtime and upon awakening.

For the laboratory portion of the experiment, participants were randomized to either a condition that involved sleep deprivation (11 Whites: 6 men, 5 women, age range 22–37; 1 Asian male, age 32) or a control condition (10 Whites: 4 men, 6 women, age range 22–38; 1 Black male, age 26). In the condition that involved sleep deprivation, participants first received two baseline nights with 10 hours time in bed for sleep; then were kept awake for 62 hours of total sleep deprivation, which entailed missing the next two nights of sleep; and were finally allowed two recovery nights with 10 hours time in bed for sleep. In the control condition, participants received 10 hours time in bed for sleep each night. All scheduled sleep periods were from 22:00 to 08:00. See Figure 1.



**Figure 1. Laboratory study design.** Each row represents a day in the experiment; each cell represents an hour. Solid black bars represent time in bed for sleep for both sleep deprivation and control groups—these periods are always 10 hours in duration (22:00 to 08:00). During the nights following day 3 and day 4, the sleep deprivation group remained awake (shown in white) while the control group was given time for sleep on both nights (shown in gray). The executive functions battery, designated here by an asterisk, occurred at 11:00 on days 3, 5, and 7; these three measurements constituted baseline, sleep deprivation, and recovery, respectively. The non-executive functions test bouts, indicated by plus signs, occurred at 2-hour intervals throughout most of the scheduled waking periods. The sleep deprivation group continued to take the non-executive functions test battery during the two nights of sleep deprivation.

The experiment was conducted in a controlled laboratory environment in the Sleep and Performance Research Center at Washington State University, Spokane. Up to four participants were in the laboratory simultaneously; each participant had their own bedroom for sleep and performance testing. The laboratory environment was sound-attenuated and temperature-controlled ( $21.3^{\circ}\text{C} \pm 1.1^{\circ}\text{C}$ ), and light levels were fixed ( $\leq 100$  lux) during waking periods. No visitors and no phone calls were allowed. Subjects were only permitted nonvigorous activities within the laboratory. Meals were provided every four hours.

The primary focus of the experiment was to investigate in detail the effects of sleep deprivation on cognitive processes. To this end, cognitive performance was tested repeatedly on an executive functions task battery and a non-executive functions task battery.

#### *Executive Functions Task Battery*

The executive functions task battery was administered during baseline (day 3), total sleep deprivation (day 5), and recovery (day 7). The baseline measurement was scheduled on day 3 to ensure that participants were fully rested and acclimated to the laboratory. The sleep deprivation measurement was scheduled at 11:00 after 51 hours awake to be certain that participants had both accrued a high homeostatic drive for sleepiness and would be close to the circadian nadir in alertness during sleep deprivation (Van Dongen & Dinges, 2005a). Finally, the recovery measurement was scheduled for day 7 to allow two 10-hour sleep opportunities for performance to recover completely (Van Dongen & Dinges, 2005b). The battery was administered at the same time of day (11:00) for both groups at all three sessions in order to avoid circadian confounds.

The executive functions battery consisted of a modified Sternberg task, a probed recall task, and a letter verbal fluency task. To minimize practice effects, the participants received three different, equivalent versions of each test in randomized, counterbalanced order over the three sessions. The order of the tests within the battery was also randomized for every participant to help control for any carryover effects from one test to the next. The executive functions battery took approximately 50 minutes to complete.

*Modified Sternberg Task* Our version of the Sternberg task combined features of the original task (Sternberg, 1966) and the modified version of the task (Whitney et al., 2004; cf. Bunge et al., 2001) so that we could separate out two dissociated executive functions components (outlined below). Each test bout contained 128 trials. The test items were letters (excluding vowels and the letter y). Our version contained memory sets of two items and of four items (50% of each). The linear relationship of RT across set size (two versus four items) was parsed into its slope and intercept. The slope of RT across set size was used to assess the executive functions component of working memory scanning efficiency. The intercept of RT across set size captures the other, largely non-executive component processes involved in task performance—encoding the probe, deciding yes or no, and executing the motor response. Additionally, our version contained negative probes that were either recent (seen in the previous trial) or non-recent (50% of each). The difference in RTs between recent and non-recent negative probes was used to assess the executive functions component of the ability to overcome proactive interference. In addition to examining speed of response (i.e., RT), accuracy was also examined as an outcome variable, using parallel procedures.

*Probed Recall Task* We used the probed recall task originated by Bunting (2006). In this task, participants are shown 12 items, one at a time, and are then asked to recall either the first four items, middle four items, or last four items in the correct order. Half of the trials were interference-maximum—for these trials the item type was either all words or all digits throughout the list. The other half of the trials were interference-release—for these trials the item type switched for the last four items from words to digits or vice versa. The difference in recall scores for the last four items between interference-maximum and interference-release trials provided a measure of the ability to overcome proactive interference.

*Letter Verbal Fluency* We used a letter verbal fluency task called the Controlled Oral Word Association Test (Benton et al., 1976). This test has previously been administered in the context of sleep deprivation (Harrison & Horne, 1997; Binks et al., 1999). In the present study, trained research assistants read instructions following a standard script (Spren & Strauss, 1998). Participants are given a letter as a prompt and are asked to generate as many words as possible in one minute that begin with that letter. This is repeated for two additional letters. We used the standard versions of this test with start letters of F-A-S, P-R-W, and C-F-L (Ross, Furr, Carter, & Weinberg, 2006).

As in previous research, we examined the total number of words generated. In addition, we examined two variables representing dissociable components of fluency performance: mean phonemic cluster size, which is believed to represent automatic (non-executive) processing; and number of switches between phonemic clusters, believed to represent executive processing related to cognitive flexibility and mental set shifting (Troyer et al., 1997). We also examined two types of errors: perseverative errors, i.e., the

number of times that a participant repeated the same word; and non-perseverative errors, which included the number of non-words, the number of proper nouns, the number of words repeated with a different ending, and the number of words that began with a different (i.e., wrong) letter.

#### *Non-executive Functions Task Battery*

A battery of tests not involving executive functions was administered at regular 2-hour intervals throughout most of the waking periods (see Figure 1). This battery consisted of the following tests known to be sensitive to sleep deprivation (e.g., Belenky et al., 2003; Van Dongen, Maislin, Mullington, & Dinges, 2003): the Psychomotor Vigilance Task (Dinges & Powell, 1985), the Digit Symbol Substitution Test (Wechsler, 1981), and the Karolinska Sleepiness Scale (Åkerstedt & Gillberg, 1990). The Karolinska Sleepiness Scale was administered both at the beginning and at the end of each test bout. We additionally included a scale that has not been widely used in studies of sleep deprivation, the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988); results from this scale are not reported here. The entire battery took approximately 18 minutes to complete.

*Psychomotor Vigilance Task* We used the 10-minute Psychomotor Vigilance Task (Dinges & Powell, 1985), which is considered the gold-standard measure of the effects of sleep deprivation on behavioral alertness (e.g., Rupp, Acebo, Seifer, & Carskadon, 2007). For this task subjects are required to respond as quickly as possible to a visual stimulus; presentations of this stimulus occurred randomly every 2 to 10 seconds. Our main variable was the total number of lapses, defined as number of reaction times



$\geq 500$ ms, which is a standard outcome measure of this task (Dorrian, Rogers, & Dinges, 2005).

*Digit Symbol Substitution Test* We used a 3-minute computerized version of the cognitive performance test of the same name in the Wechsler Adult Intelligence Scale (Wechsler, 1981). In this task, participants are shown a key whereby symbols are associated with numbers. Then symbols are presented, one a time, and the participant types in the corresponding number as quickly and as accurately as possible. To reduce practice effects, at each administration of this task a random pairing of symbols with numbers is generated. Still, this task displays a well-known practice effect across days of test bouts (e.g., Van Dongen et al., 2003). The total number of correct responses, which is thought to be a measure of cognitive throughput, was used as the outcome measure for this task.

*Positive and Negative Affect Schedule* To measure affect, subjects were asked to rate on a scale of 1 to 5 to what extent their feelings matched each of 10 words related to positive feelings and emotions and each of 10 words related to negative feelings and emotions (Watson et al., 1988). Results of this test are not reported here.

*Karolinska Sleepiness Scale* To measure subjective sleepiness, we used the Karolinska Sleepiness Scale (Åkerstedt & Gillberg, 1990). This is a Likert-type rating scale on which participants rate their subjective sleepiness using a scale from 1 (very alert) to 9 (very sleepy).

## Results

### *Executive Functions Battery*

#### *Modified Sternberg Task*

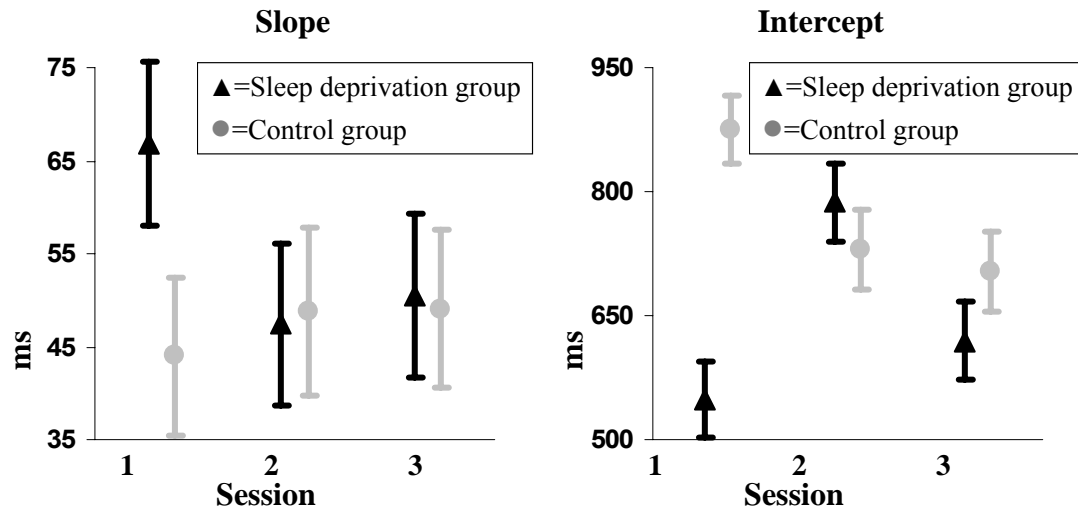
For the Modified Sternberg task, we eliminated RTs that were less than 150ms; these constituted less than 2% of the data. RTs that were more than 2000ms were automatically eliminated as participants only had a 2s window to respond. As per standard Sternberg task procedure, only data from trials where a correct response was made were used when examining RT. Data were analyzed using two different statistical methods: one method is reported below and the second method is reported in Appendix A.

*Global Aspects of Performance* RT and accuracy were each used in a mixed-effects model with a two-way interaction of group and session. Both version of the task and the order of the task within the overall battery were added as covariates. As expected, sleep deprivation significantly slowed global RT ( $F(2, 8278)=69.7, p<0.001$ ), and significantly decreased accuracy ( $F(2, 8641)=10.4, p<0.001$ ). The sleep deprivation group was 207 ms slower and 3% less accurate at the sleep deprivation session relative to baseline. Other studies have also reported speed and accuracy decrements during sleep deprivation for related working memory tasks. To interpret the cause of performance impairment on this executive functions task, we now turn to the component processes of cognition.

*Working Memory Scanning in Terms of Speed—Slope* The average magnitude of the slope was 50 ms, this magnitude is in line with that reported in previous literature. The effect of set size, or difference in RT between the two set sizes, is statistically

equivalent to the slope of the relationship between RT and set size, which represents working memory scanning efficiency (Sternberg, 1966). Thus, to examine the effect of sleep deprivation on this effect, the statistical test of primary interest was the three-way interaction of group, session and set size. In addition to the version of the task and the order of the task within the overall battery, positive versus negative (i.e., whether the probe was or was not a member of the current memory set) was included as a covariate. Only trials where the probe was non-recent (i.e., not manipulated to have high proactive interference) and for which a correct response was made were used.

As expected, there was a main effect of set size ( $F(1, 4096)=206.5, p<0.001$ ); it took participants longer to respond to a set size of four than to a set size of two and thus we replicated the finding of a linear relationship between set size and RT. There was no significant three-way interaction between group, session and set size, however ( $F(2, 4096)=1.2, p=0.31$ ). Thus, sleep deprivation had no specific effect on working memory scanning efficiency. Planned contrasts confirmed that there was no significant difference in the effect of set size in the experimental group from session 1 to session 2 ( $t(4096)=2.5, p=0.12$ ), and that there was no significant difference in the effect of set size between the sleep deprivation and control groups at session 2 ( $t(4096)=0.01, p=0.91$ ). See left hand panel of Figure 2, which displays the slopes in the sleep deprivation and control groups by session.



**Figure 2.** Slope and intercept of RT across set size on the modified Sternberg task. On the left, slopes are shown for the sleep deprivation and control groups at each session. On the right, the corresponding intercepts are shown. Symbols indicate the mean  $\pm$  standard error.

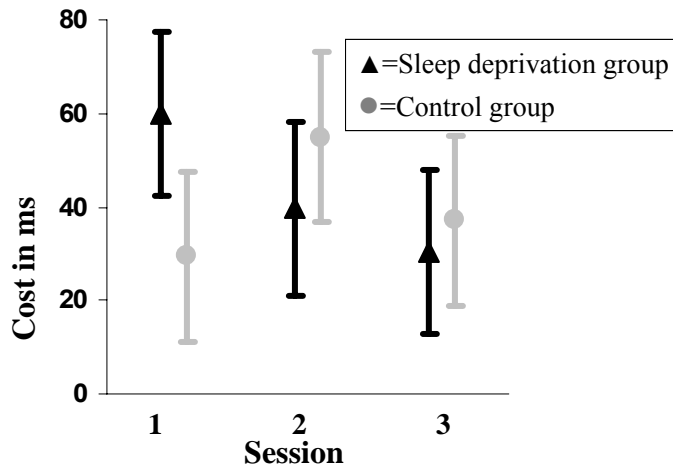
*Intercept* If RT increases aren't attributable to the slope, then they logically should be reflected by the intercept, which is thought to capture all other cognitive component processes needed to perform the task. The average value of the intercept was 693 ms. The slope and the intercept were negatively correlated ( $r=-0.45$ ,  $p<0.001$ ). Statistically, the intercept was estimated as follows: the RT at set size 2 was determined and then from that was subtracted the difference in RT between the set sizes (representing the slope). Planned contrasts confirmed that there was a significant difference in the RT intercept in the experimental group from session 1 to session 2 ( $t(4096)=37.2$ ,  $p<0.001$ ), and that there was no significant difference in the intercept between the two groups at session 2 ( $t(4096)=0.7$ ,  $p=0.39$ ). This lack of a significant difference, however, should be interpreted in light of the fact that the groups had different intercepts at baseline. An additional contrast was performed that revealed that the two groups were significantly different from each other at session 2 relative to baseline ( $t(4096)=60.6$ ,  $p<0.001$ ). Thus, the slowing in RT during sleep deprivation is reflected in a higher RT intercept, while the

slope, representing working memory scanning efficiency, is unchanged. See right hand panel of Figure 2.

*Overcoming Proactive Interference in Terms of Speed* For the ability to overcome proactive interference on the modified Sternberg task, the primary analysis for the effect of sleep deprivation on RT focused on the main effects and interaction of group, session and recency. A different subset of trials was used for examining this second executive functions component. Whereas for working memory scanning efficiency we looked at both positive and negative trials that were non-recent and excluded all recent trials, thus approximating the conditions of the original Sternberg task; here we look at both recent and non-recent trials and exclude all positive trials, as it is the negative trials that induce proactive interference through recency (Monsell, 1978). In addition to the version of the task and the order of the task within the overall battery, set size (i.e., two versus four) was included as a covariate.

As expected, there was a main effect of recency ( $F(1, 4137)=32.1, p<0.001$ ). The average cost of a recent probe was 60 ms, this is in line with published literature and we were thus successful in inducing the expected effect of proactive interference. There was no three-way interaction between group, session and recency, however ( $F(2, 4137)=0.9, p=0.40$ ). Thus, while sleep deprivation lengthened RTs, it had no specific effect of the ability to overcome proactive interference. Planned contrasts confirmed that there was no significant difference in the effect of recency in the experimental group from session 1 to session 2 ( $t(4137)=0.6, p=0.43$ ), and that there was no significant difference in the effect of recency between groups at session 2 ( $t(4137)=0.3, p=0.56$ ). See Figure 3, which

displays the cost of overcoming proactive interference in the sleep deprivation and control groups by session.



**Figure 3.** Cost of overcoming proactive interference on the modified Sternberg task. The difference in RT between non-recent and recent probes is shown for the sleep deprivation and control groups. Symbols and error bars indicate mean  $\pm$  standard error.

*Executive Function Components in Terms of Accuracy* It is possible that although our executive functions components were preserved during sleep deprivation in terms of speed, deficits in one or both of these components of cognition may have shown up in accuracy (reflecting a speed-accuracy tradeoff). Accordingly, analyses were repeated with accuracy instead of RT as the outcome variable.

Accuracy was  $>90\%$  during sleep deprivation for both set sizes. There was no three-way interaction between group, session and set size on accuracy ( $F(2, 4289)=0.2$ ,  $p=0.81$ ). As such, performance on working memory scanning efficiency was preserved during sleep deprivation in terms of accuracy as it was preserved in terms of speed. A planned contrast also confirmed that there was no significant difference in the effect of set size on accuracy in the experimental group from session 1 to session 2 ( $t(4289)=1.3$ ,

$p=0.25$ ), and that there was no significant difference in the effect of set size on accuracy between the two groups at session 2 ( $t(4289)=2.5, p=0.11$ ).

Accuracy was  $>90\%$  during sleep deprivation for both recent and non-recent probes. Similarly, there was no three-way interaction between group, session and recency on accuracy ( $F(2, 4295)=0.4, p=0.65$ ). Thus, the ability to overcome proactive interference was preserved during sleep deprivation in terms of accuracy as it was preserved in terms of speed. Planned contrasts confirmed that there was no significant difference in the effect of recency on accuracy in the experimental group from session 1 to session 2 ( $t(4295)=2.2, p=0.14$ ), and that there was no significant difference in the effect of recency on accuracy between groups at session 2 ( $t(4295)=0.3, p=0.59$ ).

#### *Probed Recall*

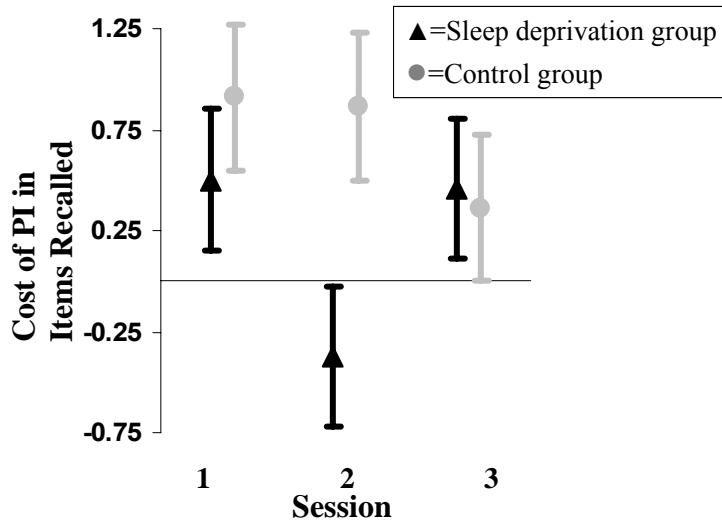
For this task we were interested in recall of the last four items. There was a main effect of list type ( $F(1, 241)=9.7, p=0.002$ ); as expected, participants recalled more of the last four items from the interference-release than from the interference-maximum trials and thus our manipulation was successful. Recall between interference-release and interference-maximum trials was slightly correlated ( $r=0.28, p=0.02$ ). Participants remembered on average 0.44 items more out of the four for interference-release trials than for interference-maximum trials.

There was no group by session interaction on recall of the last four items ( $F(2, 241)=0.6, p=0.56$ ). Planned contrasts revealed that there was no significant difference between sleep deprivation participants from baseline to sleep deprivation ( $t(241)=1.3, p=0.21$ ) or between the groups at the second sleep deprivation session

( $t(241)=-1.1, p=0.26$ ). Thus, sleep deprivation did not affect the ability to recall these last four items.

There was no three-way interaction between group, session and list type ( $F(2, 241)=1.8, p=0.17$ ), suggesting that sleep deprivation had no effect on the ability to overcome proactive interference. Planned contrasts revealed a trend in the effect of list type in the experimental group from session 1 to session 2 ( $t(241)=3.1, p=0.08$ ). This trend occurred as the direction of the means changed: during sleep deprivation, the sleep deprived participants did not show a cost of proactive interference but recalled more instead of fewer words at the interference-maximum trials. See Figure 4. At all other sessions in this sleep deprived group and at all other sessions in the other control group, participants, as expected, recalled fewer words at these interference-maximum trials. We can thus safely conclude that sleep deprivation did not impair the ability to overcome proactive interference: if anything sleep deprived participants were improved, not impaired, on this component of executive functioning. A planned contrast found that there was a significant difference in the effect of list type on recall accuracy between groups at session 2 ( $t(241)=6.02, p=0.01$ ). Thus, an additional contrast was performed that revealed that the two groups were not significantly different from each other at session 2 relative to baseline ( $t(241)=1.35, p=0.25$ ). See Figure 4.





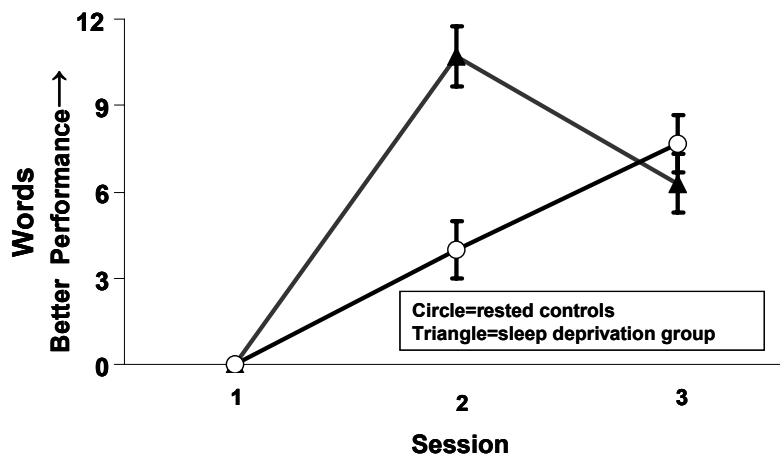
**Figure 4.** Cost of proactive interference (PI) for the probed recall task. Graphs show the cost of proactive interference in terms of number of items recalled for the sleep deprivation and control groups at each of the three sessions. Symbols and error bars indicate mean  $\pm$  standard error.

In addition to the above analysis, the difference between recall of the last items of PI-maximum and PI-release lists was calculated directly for each subject at each session and entered into a mixed-effects ANOVA. There was no group by session interaction, ( $F(2, 40)=1.75, p=0.19$ ), suggesting that sleep deprivation did not affect the ability to overcome proactive interference. A planned contrast revealed a trend towards significance in PI-diff between experimental subjects from session 1 to session 2 ( $t(40)=3.05, p=0.09$ ). The direction of the mean was that participants showed less of a cost (showed a gain) with proactive interference, however. Thus, there was no evidence that sleep deprivation significantly affected the ability to overcome proactive interference. A second planned contrast revealed a significant difference in PI-diff between experimental and control subjects at session 2 ( $t(40)=6.0, p=0.019$ ). A third comparison ( $t(40)=1.5, p=0.24$ ) revealed that the two groups were not significantly

different from each other at session 2 relative to baseline, however. In any case, the direction of the difference is that sleep deprived participants were *better* able to overcome proactive interference rather than worse. Thus, the two methods of analysis converge to suggest that sleep deprivation does not impair the ability to overcome proactive interference as measured by this test of probed recall.

### *Letter Verbal Fluency*

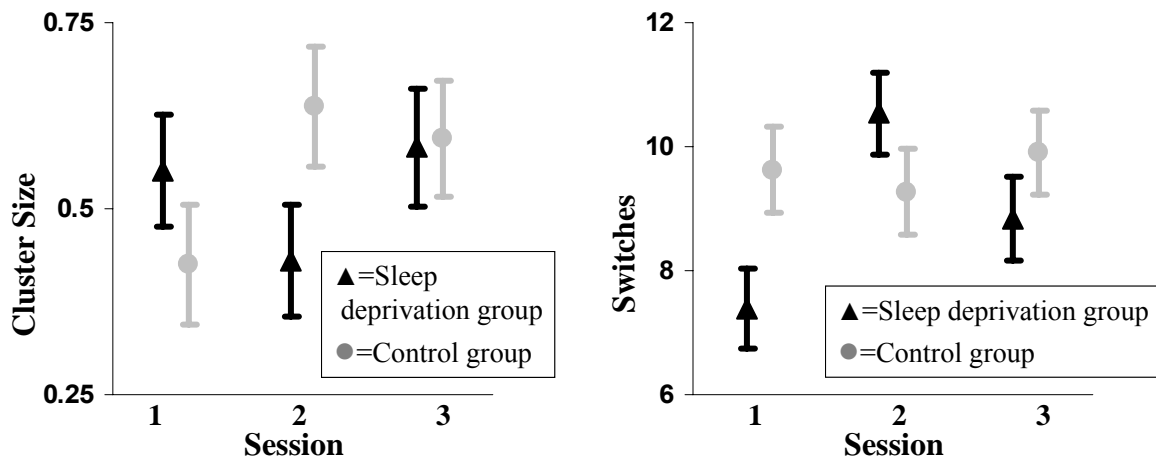
On average, participants generated 42 words per test bout, this is in line with published reports (e.g., Troyer et al., 1997). For words generated, there was an interaction of group and session ( $F(2, 173)=5.2, p=0.007$ ); on average, sleep deprived participants generated an additional 10.8 words than they did during their baseline session ( $t(173)=-5.8, p<0.001$ ) and an additional 6.3 words than they did during their recovery session ( $t(173)=-3.3, p=0.001$ ). See Figure 5.



**Figure 5.** Words generated during the letter verbal fluency task. Difference from baseline in the total number of words generated is shown during sessions 2 and 3 for the sleep deprivation and control groups. Symbols and error bars indicate mean  $\pm$  standard error.

We now turn to the two component processes contributing to global performance on this test: mean cluster size and number of switches between clusters. The two component processes were modestly negatively correlated ( $r=-0.46, p<0.001$ ). Cluster size showed a slight but significant correlation with total words generated ( $r=0.22, p=0.002$ ) while switches showed a substantial and significant correlation with total words generated ( $r=0.71, p<0.001$ ). Average phonemic cluster size was  $0.53 \pm 0.42$  while average number of switches was  $27.6 \pm 9.6$ . These results mirror those in the published literature, and thus we replicated the basic effects on this task (e.g., Troyer et al., 1997).

For average phonemic cluster size there was an interaction of group and session ( $F(2, 173)=3.1, p=0.05$ ). While controls had significantly bigger clusters during the second session ( $t(173)=-2.2, p=0.03$ ), experimental participants had smaller clusters during this second sleep deprivation session, although this difference was not statistically significant from their baseline score ( $t(173)=1.32, p=0.19$ ) or from their recovery score ( $t(173)=-0.34, p=0.74$ ). Thus, sleep deprivation did not significantly affect the non-executive component of this test. See Figure 6.



**Figure 6.** Mean phonemic cluster size and number of switches between phonemic clusters on the letter verbal fluency task. On the left, mean cluster size is shown for the sleep deprivation and control groups at each session. On the right, the number of switches between clusters at each of these sessions are shown. Symbols and error bars indicate mean  $\pm$  standard error.

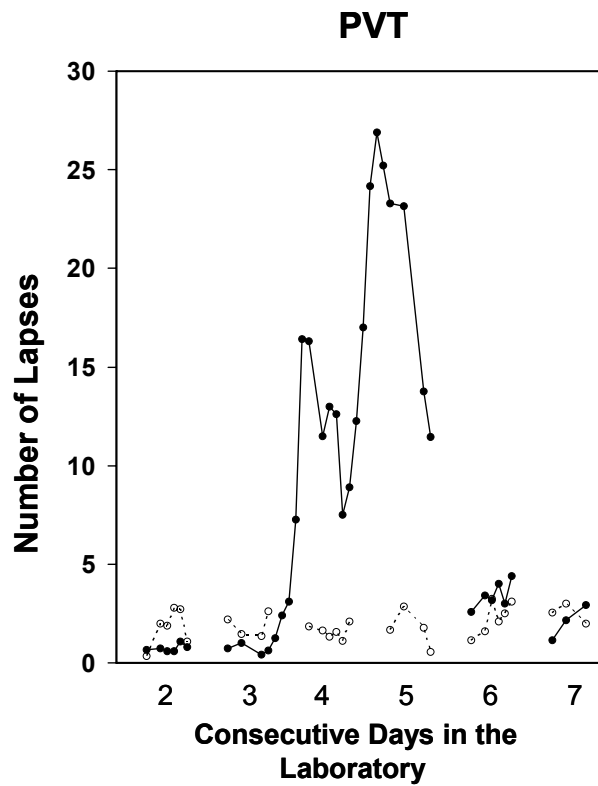
For switches between phonemic clusters there was a significant interaction of group by session ( $F(2, 173)=8.3, p<0.001$ ); sleep deprived participants generated on average 9.4 more switches between phonemic clusters when sleep deprived than baseline ( $t(173)=-5.3, p<0.001$ ) and 4.4 more switches when sleep deprived than during recovery ( $t(173)=-2.4, p<0.001$ ). Thus, during sleep deprivation performance was significantly improved on the component of performance thought to reflect executive functioning. See Figure 6.

Words that were errors (i.e., that started with the wrong letter) contribute to the number of switches. It is possible that the increase in the number of switches during sleep deprivation was due to an increased number of errors. We investigated whether sleep deprivation increased either perseverative or non-perseverative errors. There was no group by session interaction for perseverative errors ( $F(2, 173)=0.19, p=0.83$ ). Contrasts

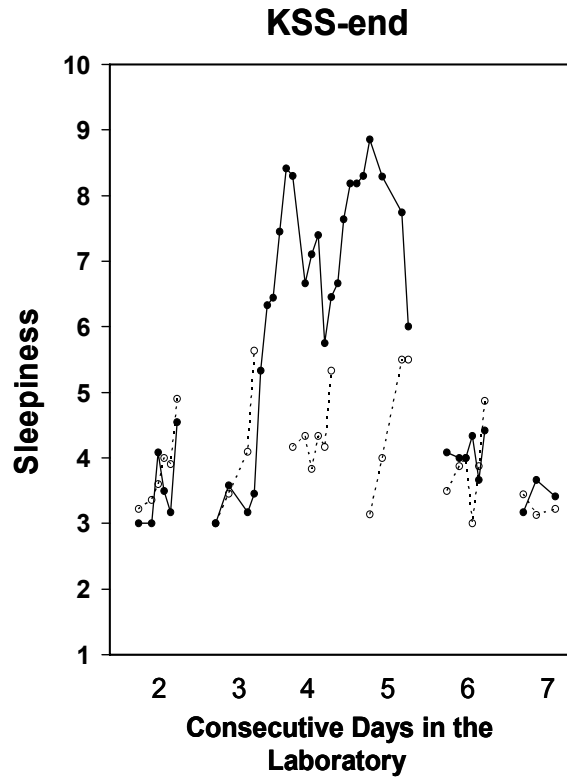
revealed that sleep deprived participants made no more perseverative errors while sleep deprived than during baseline ( $t(173)=-0.28, p=0.78$ ) or recovery ( $t(173)=-1.25, p=0.21$ ). Additionally, the two groups made an equivalent number of perseverative errors at session 2 ( $t(173)=1.29, p=0.20$ ). There was similarly no group by session interaction for nonperseverative errors ( $F(2, 173)=0.22, p=0.80$ ). Contrasts revealed that sleep deprived participants made no more nonperseverative errors while sleep deprived than during baseline ( $t(173)=0.08, p=0.93$ ) or recovery ( $t(173)=-0.19, p=0.85$ ). The two groups made an equivalent number of nonperseverative errors at session 2 ( $t(173)=0.17, p=0.86$ ). Thus, during sleep deprivation participants made no more errors than they did while rested. The improvement in switching during sleep deprivation (i.e., the increase in the number of switches) was not due to an increase in the number of errors but reflected a greater number of switches to task-appropriate words.

*Non-executive functions battery*

While there was only one administration of the executive functions battery at baseline, during sleep deprivation, and after recovery, there were multiple administrations of the non-executive functions task battery throughout these phases of the experiment. This battery was included to ensure that we replicated the basic effects of sleep deprivation. The data of the non-executive functions tasks were graphed across days to ensure the typical profile of performance impairment during sleep deprivation was obtained and to detect any outliers. For all outcome measures, no more than 5% of data were outliers. When outliers were found, analyses were run both with and without the outliers. As no substantive differences were found, only the analyses with the outliers removed are shown and reported. See Figures 7-9.

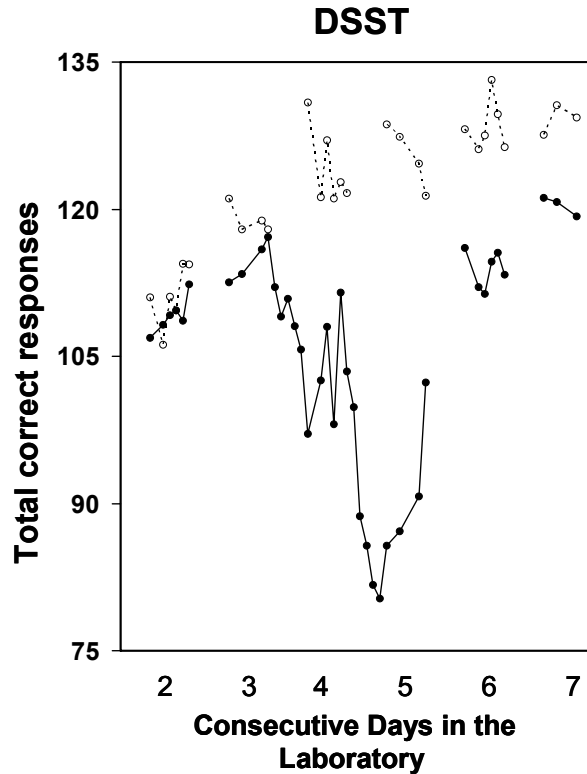


*Figure 7.* Lapses made on the PVT. Number of PVT lapses for both groups across days in the laboratory. For the sleep deprivation group, days 2 and 3 are baseline, days 4 and 5 are sleep deprivation, and days 6 and 7 are recovery.



*Figure 8.* Ratings on the KSS. Reported sleepiness on the KSS at the end of the test bout for both groups across days in the laboratory. For the sleep deprivation group, days 2 and 3 are baseline, days 4 and 5 are sleep deprivation, and days 6 and 7 are recovery.





*Figure 9.* Correct responses made on the DSST. Total number of correct responses made on the DSST for both groups across days in the laboratory. For the sleep deprivation group, days 2 and 3 are baseline, days 4 and 5 are sleep deprivation, and days 6 and 7 are recovery.

Further, mixed-effects ANOVAs with group and test bout and their interaction were used to statistically compare the sleep deprivation and control groups. Only test bouts that the two groups had in common were included (i.e., the ten test bouts that the experimental group took during sleep deprivation were excluded). The first two test bouts of day 1 were excluded, as they were practice bouts; the last test bout of the study was also excluded to control for any end-of-study effects.

For lapses on the psychomotor vigilance task, there was a significant effect of group ( $F(1, 513)=7.8, p=0.005$ ); the sleep deprived group made significantly more lapses. There was a significant effect of test bout ( $F(28, 513)=9.0, p<0.001$ ); more lapses were made

during the sleep deprivation portion of the experiment than were made during the baseline and the recovery portions of the experiment. There was a significant interaction between group and test bout ( $F(28, 513)=9.1, p<0.001$ ); sleep deprived participants made significantly more lapses during the sleep deprivation portion of the experiment. We thus replicated the basic effect of sleep deprivation for lapses on this gold-standard measure of alertness during conditions of sleep loss.

For correct responses on the Digit Symbol Substitution Test there was a main effect of group ( $F(1, 513)=4.0, p<0.05$ ); the sleep deprivation group made significantly fewer correct responses. There was a significant effect of test bout ( $F(28, 513)=9.8, p<0.001$ ); fewer correct responses were made during the sleep deprivation portion of the experiment than were made during the baseline and the recovery portions of the experiment. There was a significant interaction between group and test bout ( $F(28, 513)=8.1, p<0.001$ ); sleep deprived participants made significantly fewer correct responses during the sleep deprivation portion of the experiment. We thus replicated the basic effect of sleep deprivation on decreasing the number of correct responses on this task.

For sleepiness ratings on the Karolinska Sleepiness Scale there was a main effect of group ( $F(1, 476)=8.4, p=0.004$ ); the sleep deprivation group reported significantly greater sleepiness. There was a significant effect of test bout ( $F(28, 476)=10.7, p<0.001$ ); greater sleepiness was reported during the sleep deprivation portion of the experiment than during the baseline and the recovery portions of the experiment. There was a significant interaction between group and test bout ( $F(28, 476)=7.0, p<0.001$ ); sleep deprived participants reported significantly greater sleepiness during the sleep deprivation portion

of the experiment. We thus replicated the basic effect of sleep deprivation on increasing self-reported sleepiness.

## Discussion

In this carefully controlled laboratory experiment, dissociated components of executive functioning were found to be maintained at baseline levels after 51 hours of total sleep deprivation. These results challenge a prevailing view that executive functions are specifically vulnerable to impairment due to sleep deprivation (see Durmer & Dinges, 2005). Instead, the current results suggest that specific components of executive functioning can be resistant to the effects of sleep loss.

We took elaborate steps to avoid pitfalls commonly encountered in the study of cognitive functioning during sleep deprivation (see Van Dongen & Dinges, 2005a). The laboratory environment was strictly controlled in terms of variables known to affect sleep and performance, such as ambient temperature and light levels. Cognitive testing, sleep periods, meal times, and other study activities were scheduled at standardized times to control for circadian confounds.

Participants were administered a battery of tests known to be sensitive to sleep deprivation (i.e., the non-executive functions task battery) throughout baseline, sleep deprivation, and recovery periods to verify that well-known effects of acute total sleep deprivation were replicated in the current study. We were successful in inducing a considerable level of sleep pressure, as indicated by performance on our non-executive functions battery. That is, during sleep deprivation participants produced elevated sleepiness ratings on the Karolinska Sleepiness Scale and increased numbers of lapses on the Psychomotor Vigilance Test. The expected circadian and homeostatic effects were seen on these tests (e.g., Van Dongen & Dinges, 2005b). In addition, sleep deprived participants made significantly fewer correct responses on the Digit Symbol Substitution

Test while control participants, by contrast, displayed the normal pattern of learning across days in the laboratory (e.g., Van Dongen et al., 2003).

The study was focused on the effects of sleep deprivation on dissociated components of executive functioning. Because executive functions tests may become less executive in nature with repeated administration (e.g., Phillips, 1997), participants performed such tests only once during each of the baseline, sleep deprivation, and recovery periods. Three different versions of these tests were created and administered in randomized counterbalanced order to minimize practice effects. Additionally, the tests were administered in a randomized order within the overall battery to control for any carryover effects. A non-sleep-deprived control group was included to account for any effects of being in the laboratory and performing the cognitive tests repeatedly. Finally, a power calculation was done in advance of the study to ensure adequate sample size.

The executive functions task battery administered in our study included tests that allowed for the dissociation of executive and non-executive components of performance. This is important because executive functions, by definition, operate on more basic cognitive functions. Thus, performance on an executive functions task implicates other processes than just the target executive function(s) of interest. Much of the previous literature on executive functioning during sleep deprivation focused on global outcome scores, and yielded mixed and confusing results. We sought to clarify this issue by examining executive functioning in terms of dissociated cognitive processes.

The first executive functions test we used was a Sternberg working memory task modified after an adapted version developed by Whitney et al. (2004) that allows for the dissociation of two components of executive functioning (cf. Bunge et al., 2001).

Previous studies using more basic Sternberg-type working memory tasks have consistently reported a decrease in global speed and accuracy during sleep deprivation (e.g., Habeck et al., 2004; Mc-Ginty et al., 2004; Mu et al., 2005; Raidy & Sharff, 2005; Chee et al., 2006). The current results replicate this consistent finding. However, many of the previous studies concluded that since global performance of a working memory task was lower, therefore working memory was impaired by sleep deprivation. Yet, as we have argued, a low score on an executive functions test does not necessarily mean impairment of the target executive function, but could arise instead due to impairment of other processes involved in the task. Thus, although global speed and accuracy were significantly lower during sleep deprivation, it can not be assumed that working memory was necessarily impaired.

Our modified Sternberg task allowed for the dissociation of two components of executive functioning: working memory scanning efficiency and the ability to overcome proactive interference. That is, beyond a global slowing in RT, this test allowed for the examination of whether sleep loss differentially affects dissociated component processes of executive functioning. For example, RTs from two different memory set sizes should share all cognitive components except the time taken to scan the additional items in working memory for the bigger set size. The slope of the line of the RTs through the two different set sizes, then, should dissociate the component process of working memory scanning efficiency while the intercept should capture other elements of task performance. Similarly, RTs can be compared between non-recent and recent negative probes, which share all component processes except the additional time taken to overcome the proactive interference associated with recent probes. By subtracting the RT

between these two types of probes, then, the ability to overcome proactive interference can be dissociated.

For the modified Sternberg, although significant performance impairments were seen during sleep deprivation for both speed (as reflected in the intercept of the line of RT by set size) and accuracy, sleep deprivation had no effect on either dissociated component of executive functioning. That is, the slope (of the line of RT by set size) was equivalent between rested and sleep-deprived conditions, and thus working memory scanning efficiency was preserved. Similarly, the difference in RT between probes manipulated to have high proactive interference and standard probes (i.e., recent and non-recent) was also equivalent between rested and sleep deprived conditions, and the ability to overcome proactive interference was preserved. Thus, executive components of cognition were preserved even as global performance of the executive task was impaired. This finding underscores the importance of dissociating components of performance to properly understand the source of decrements during sleep deprivation.

The second executive functions test was a probed recall task (Bunting, 2006) that allowed for the dissociation of the ability to overcome proactive interference. Probed recall tasks direct participants to recall certain portions of the memory list such as the first, middle, or last sections, or, the associate that has been paired with a given stimulus. Previous studies of probed recall tasks during sleep deprivation have used a delay between study and recall (Cajochen, Khalsa, Wyatt, Czeisler, & Dijk, 1999; Drake et al., 2001), and are thus not directly comparable to the present study where recall was probed immediately after presentation of the stimuli. The current study found no evidence for

decreases in global performance (number of words remembered) on this task during sleep deprivation.

Again, our probed recall task allowed for the dissociation of the ability to overcome proactive interference. In half of the lists, the item type switches for the last four items; this manipulation releases proactive interference for these items. By comparing the number of items recalled from the last portion of the list when the item type remains the same as to when it has been switched for these items, the effect of proactive interference can be dissociated. As in the modified Sternberg task, this component process was preserved during sleep deprivation. One caveat is that as some of our participants were at ceiling for this task it is possible that we were unable to see an effect. However, the mean was in the opposite direction during sleep deprivation, suggesting that ceiling effects did not confound the results.

The third executive functions task was a letter verbal fluency task that allowed for the dissociation of the executive function of shifting between linguistic subcategories. In this task, participants are asked to generate as many words as possible in one minute that begin with a given letter. For letter verbal fluency tasks, previous studies on the effects of sleep deprivation on global performance reported mixed findings. Horne (1988) administered two letters and reported impairment. In that study, however, verbal fluency was administered directly after a one hour executive functions test which may have engendered carryover or time-on-task effects. In the present study, the order of tasks within the overall test battery was randomized for each participant in order to control for such effects. In a later study by Harrison and Horne (1997), the letter verbal fluency task was administered by itself; impairment was not found for the first two of three initial start



letters but only for the third. It should be noted this study assessed a variable similar to our phonemic cluster size and similarly reported no significant differences between sleep deprived and control sessions. As the sample size in that study was only 7, practice effects were not taken into account, and subjects served as their own controls in a manner that cannot be completely counterbalanced with an odd number, it is difficult to gauge the reliability of these findings. In the current study we had 12 participants undergo sleep deprivation and 11 control participants were also included; additionally, practice effects were accounted for in data analyses. Binks et al. (1999) found no impairment on the letter verbal fluency task during sleep deprivation. Testing in this study, however, as the authors point out, was administered during the wake maintenance zone on the evening following a single night of sleep deprivation; during this time performance impairments due to sleep deprivation may be masked due to circadian alerting factors. In the current study, participants were tested near the circadian nadir during extended (two-night) sleep deprivation.

The present study dissociated two components of performance on the letter verbal fluency task: mean phonemic cluster size, which is believed to represent automatic processing; and number of switches between phonemic clusters, believed to represent executive processing (Troyer et al., 1997). The numbers of perseverative and non-perseverative errors were also examined. The number of switches, representing executive processing, was significantly increased during sleep deprivation while neither cluster size nor number or type of errors were significantly affected. It is not likely that a component of executive functioning was enhanced during sleep deprivation. It is more likely that switching on this task was easier due to the degradation of some other cognitive process.

For example, if less proactive interference was generated by previously generated items and clusters, it might be easier to switch between clusters. Indeed, as Abwender et al. (2001) point out the interpretation of cluster size and switching is still unresolved.

In our data, the dissociated executive functions component (switching between phonemic subcategories) was highly correlated (0.71) with global performance (total number of words generated). This correlation arose because average cluster sizes were less than one (0.53) and heavily skewed towards zero; thus more than half of all generated words constituted a switch between phonemic clusters. In the article where clustering and switching were initially proposed (Troyer et al., 1997), average phonemic cluster size was similarly low (0.36). In short, we believe that improvement of the variable of switching between clusters may simply reflect the global improvement in performance during sleep deprivation on the letter verbal fluency task.

This improvement on the letter verbal fluency task was an unexpected finding, although a similar phenomenon has been observed in studies of acetylcholine blocking. When acetylcholine is blocked in rested individuals using scopolamine, performance on the letter verbal fluency test is improved even as performance of other types of fluency tests is impaired (see Pompéia, Rusted, & Curran, 2002). One of the neurochemical effects of sleep deprivation is a reduction in levels of acetylcholine in the brain (Boonstra, Stins, Daffertshofer, & Beek, 2007). Therefore, it could be that in sleep deprivation the same mechanism is at work. In any case, current findings on this task provide no evidence that components of executive functioning are impaired during sleep deprivation.

The current study is among the first to focus on the effects of sleep deprivation on dissociated components of executive functioning. For both the modified Sternberg and

the probed recall task the dissociated executive functions component of the ability to overcome proactive interference responded the same way during sleep deprivation—performance was maintained. Examining global performance on these two tasks leads to confusion about whether or not executive functioning is impaired during sleep deprivation. Isolating the executive component of interest, by contrast, leads to the clear conclusion that the ability to overcome proactive interference—one key element of executive functioning—is not affected by sleep loss. Of course, as with any null results, there is always the possibility that with more power a significant result would have been obtained. Yet, power analyses were done in advance of the study to determine the sample size. Additionally, mixed-effects models were used which likely enhanced statistical power even further. Thus, any results would be very slight in any case and likely not clinically relevant.

For the modified Sternberg, we additionally separated the executive functions component of working memory scanning efficiency (i.e., slope) from other, largely non-executive components of task performance (i.e., intercept). We saw that although global performance of this task was impaired, this was due to changes in the intercept, reflecting largely non-executive components of cognition, while the slope, reflecting a relatively pure measure of executive functioning, was not affected. We had enough power to see a significant change in the intercept, which makes it more likely that we had adequate power to detect a change in the slope. That is, the significant change in the intercept strengthens our argument that the null result for the slope was not due to a lack of power, but arose because sleep deprivation does not affect this component of executive functioning.

Although other studies have occasionally reported on the performance of executive functions components during sleep deprivation (Jennings et al., 2003; Habeck et al., 2004; Heuer et al., 2005; Turner et al., 2007), to our knowledge only one of these examined an executive functions component also considered in our study. Habeck et al. (2004) used a Sternberg task and reported that while the RT intercept was significantly higher after 48 hours of wakefulness, the slope was unchanged, and concluded that working memory scanning efficiency was unaffected by sleep deprivation. This was a controlled laboratory study using a large number of subjects and a control group. This lends further credibility to the present findings that sleep deprivation does not affect this component of executive functioning.

Taken together, the present results challenge existing theories of how sleep loss affects cognitive functioning (Doran et al., 2001; Harrison et al., 2000; Pilcher et al., 2007). The theory that sleep deprivation specifically impairs executive functioning due to disruption of prefrontal cortical metabolism (Harrison et al., 2000) was *not* supported by the current results. The dissociated components of executive functioning examined in the current study have been localized to the prefrontal cortex (e.g., Rypma, Berger & D'Esposito, 2002; Jonides & Nee, 2006). Yet, these executive functioning components were selectively preserved (not impaired) by sleep deprivation.

At first glance, maintained components of executive functioning during sleep deprivation appear inconsistent with a positron emission tomography (PET) report of *decreased* prefrontal cortical metabolism (Thomas et al., 2000). A closer look at some recent functional magnetic resonance imaging (fMRI) papers provides a possible reconciliation. PET provides an absolute measurement of cortical metabolism, fMRI

provides a relative measurement. Drummond, Brown, Salamat, & Gillin (2004), for example, using fMRI examined the response of the sleep-deprived brain to parametrically manipulated task demands on a grammatical reasoning task. As tasks become more difficult, an increasing burden is placed on executive functioning components to coordinate task demands. Under rested conditions, increased difficulty on this task resulted in a linear increase of brain metabolism in a handful of brain regions. During sleep deprivation, an even steeper linear *increase* to increasingly difficult task demands was reported in some of these same regions, including some prefrontal areas. Additionally, new areas that were not associated with performance of the task under rested conditions showed increased activation as tasks became more difficult during sleep deprivation; and this increased activation was correlated with maintained performance. This study suggests, then, that although the prefrontal cortex may show *decreased* absolute activation during sleep deprivation, this area may be able to selectively *increase* relative activation during sleep loss as tasks become more difficult, and require more executive functioning.

One of the prefrontal areas that showed an increased compensatory response in the above study during sleep deprivation was the left inferior frontal gyrus, a portion of the prefrontal cortex shown to be critical to our dissociated executive functions components during rested conditions (Mummary et al., 1996; Rypma, Berger & D'Esposito, 2002; Jonides & Nee, 2006). This area further showed an *increased* compensatory response during sleep deprivation in two later studies that used a working memory and a verbal list learning task, respectively (Chee & Choo, 2004; Drummond, Meloy, Yanagi, Orff, & Brown, 2005). By contrast to the previous studies, Choo et al.

(2005) reported that this region showed a *decreased* hemodynamic response to difficult task demands on an n-back test during sleep deprivation, yet in that study performance was not maintained (i.e., it was impaired). Taken together, these findings suggest that at least for some complex tasks, cortical regions, including the area reported to be most important for the current components of executive functioning, display a disproportionate *increase* in relative activation as tasks become more challenging during sleep deprivation; this increased relative activation is associated with maintained performance (i.e., appears to be a compensatory response). These neuroimaging findings, in concert with the current behavioral findings, undermine the view that sleep deprivation selectively impairs executive functioning through decreased metabolism in the prefrontal cortex. It appears instead that executive functioning may be especially resilient to sleep deprivation.

The second theory that we consider is state-instability hypothesis (Doran et al., 2001), which asserts that performance impairment during sleep deprivation is driven by increasing moment-to-moment intrusion of sleep-initiating mechanisms which destabilize attention and thereby cognitive performance. While clear counterexamples were provided by the current results to the view that sleep deprivation selectively impairs executive functioning, wake-state instability theory was partially supported. According to this theory, rapid performance *variability* is the key to detecting performance impairments during sleep loss, and thus tests which collect relatively large samples of performance in a relatively brief amount of time are most suitable. Indeed, performance was not impaired during sleep deprivation for either of the two executive functions tasks that had relatively low response density, probed recall and letter verbal fluency. Performance was impaired

during sleep deprivation for the test that had the highest response density, the modified Sternberg task.

According to wake-state instability theory, decrements appear in performance during sleep deprivation due to increasing variability in the capacity to sustain attention. It is possible that attention deficits were responsible for the significant decrement in performance found on the modified Sternberg. Attention deficits would likely translate into difficulty in encoding the probe on this task, which is reflected in the intercept. The intercept was indeed the only component process of the modified Sternberg that was significantly higher during sleep deprivation, although it cannot be ruled out whether this was due to deficits in encoding the probe, deciding yes or no, or executing the motor response. It is noteworthy that the executive function component of the task was preserved even as the intercept was impaired. Executive components of performance, then, should be examined separately to fully understand the impact of sleep deprivation on cognition.

The final and most recent model considered here, the controlled attention model (Pilcher et al., 2007), was not supported by the current findings. According to this model, sleep deprivation impairs the ability to sustain attention in the face of mental and environmental distractors, particularly when performing tasks that are monotonous. Recent evidence suggests that the ability to overcome mental distractors is independent from the ability to overcome environmental distractors (Friedman & Miyake, 2004). A recent study reported that an *environmental* distraction (a television program playing in the background) interfered with performance significantly more during sleep deprivation than during rested conditions (Anderson & Horne, 2006). The current study included two

tests that each create *mental* distraction, in the form of proactive interference. One of these, the probed recall task, is challenging while the other, the modified Sternberg task, is simple and repetitive. For both the challenging and the simple task, proactive interference was no more distracting under conditions of sleep loss than during rested conditions—if anything, it was slightly less distracting. Thus, the controlled attention model can be refined. Specifically, the sleep deprived individual appears immune to mental distractors, or proactive interference, even for less intrinsically engaging tasks.

In conclusion, the current study dissociated executive functions tasks into executive and non-executive components to understand the true source of any performance changes on executive functions tasks during sleep deprivation. We found no evidence of either executive or non-executive performance impairment for two brief executive functions tasks, probed recall and letter verbal fluency. For a longer executive functions task, the modified Sternberg, we found impairment during sleep deprivation only for non-executive components of performance while two key components of executive functioning were maintained. Future studies might examine such additional components of executive functioning as those involved in regulating emotion. The components of executive functioning that were examined, however—working memory scanning, the ability to overcome proactive interference, and shifting between linguistic subcategories—were resistant to 51 hours of total sleep deprivation.



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## Appendix A

As an additional check of key modified Sternberg results, we analyzed the data a second way. For the first method of analysis all data points were entered into the mixed-effects ANOVA and the statistical test of interest was a three-way interaction. For the second method of analysis, reported below, we calculated key outcome variables in advance to enter into the mixed-effects ANOVA. Variables were calculated as an average per subject per session. By averaging we reduced the number of data points but also reduced the amount of noise in the data. For this second method of analysis, the statistical test of interest was a two-way interaction. Key outcome variables were the slope, the intercept, and the cost of overcoming proactive interference.

For slopes, there was no interaction of group by session ( $F(2, 40)=0.0, p=1.0$ ). Planned contrasts confirmed that there was no significant difference in slopes in the experimental group from session 1 to session 2 ( $t(40)=1.4, p=0.24$ ), and that there was no significant difference in slopes between the sleep deprivation and control subjects at session 2 ( $t(40)=0.04, p=0.84$ ). Thus, the slope of RT through set size, reflecting working memory scanning efficiency, was maintained at baseline levels during sleep deprivation. See Figure 10.

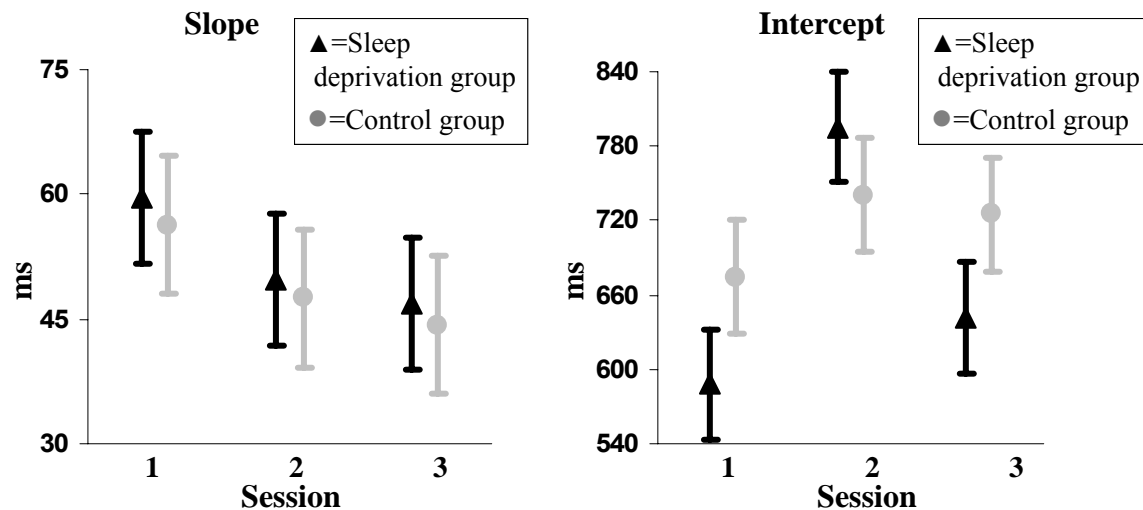
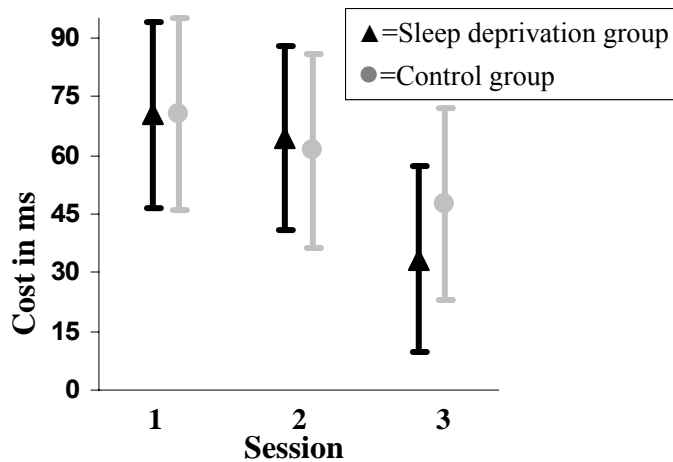


Figure 10. Second method of analysis for the slope and intercept on the modified Sternberg task. On the left, slopes are shown for the sleep deprivation and control groups at each session. On the right, the corresponding intercepts are shown. Symbols indicate the mean  $\pm$  standard error.

Next we turn to the intercept. There was an interaction of group by session ( $F(2, 40)=10.6, p<0.001$ ). A planned contrast revealed that the intercept was significantly greater in the sleep deprivation group at session 2 than at session 1 ( $t(40)=72.0, p<0.001$ ). At session 2 the intercept was equivalent for the sleep deprivation and the control groups ( $t(40)=0.7, p=0.40$ ). This equivalence should be seen in light of the fact that the control group started out with a significantly greater intercept. Thus, an additional contrast was performed that revealed that the two groups were significantly different from each other at session 2 relative to baseline ( $t(40)=16.2, p<0.001$ ). Thus, the intercept, reflecting largely non-executive components of performance, was impaired during sleep deprivation. See Figure 10.

For the cost of overcoming proactive interference, there was no interaction of group by session ( $F(2, 40)=0.08, p=0.92$ ). Planned contrasts confirmed that there was no significant difference in the cost of overcoming proactive interference in the experimental

group from session 1 to session 2 ( $t(40)=0.03, p=0.85$ ), and that there was no significant difference in this cost between the sleep deprivation and control groups at session 2 ( $t(40)=0.01, p=0.92$ ). Thus, the ability to overcome proactive interference was maintained at baseline levels during sleep deprivation.



**Figure 11. Second method of analysis for the cost of overcoming proactive interference on the modified Sternberg task. The difference in RT between non-recent and recent probes is shown for the sleep deprivation and control groups. Symbols and error bars indicate mean  $\pm$  standard error.**

In short, the results of this second method of analysis mirror the results of the first method. Using both methods, we found that sleep deprivation significantly lengthened the intercept, reflecting an impairment of non-executive components of performance. Both methods also converged in that sleep deprivation did not significantly affect either working memory scanning efficiency or the cost of overcoming proactive interference, reflecting two key executive components of performance. See Figure 11.