Using ex-Gaussian analysis of reaction time in a psychomotor vigilance test to assess the effects

of napping following sleep restriction

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Abstract

The psychomotor vigilance test (PVT) is frequently used to measure the negative effects of sleep deprivation on vigilant attention. Although reaction times (RT's) follow an ex-Gaussian distribution and sleep deprivation generally affects the worst performance, many researchers still use meant RT to describe performance on PVT data. In addition, few studies have used the PVT to study the recuperative effects of napping on PVT performance. As a result, the purpose of our project is to determine whether a 20-minute nap taken in the afternoon differentially affects the shape of the RT distribution following partial sleep restriction. Analysis of ex-Gaussian parameters revealed that napping improved mu, suggesting a distributional shift of RT. As a result, 20-min naps taken in the afternoon improve decrements in vigilant attention caused by partial sleep restriction.

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1. Introduction

Although we may justify staying up late, we also underestimate the negative effects of sleep deprivation. For example, a lack of sleep has been shown to affect emotions (Killgore et al., 2006), levels of stress (Leproult et al., 1997), cognitive and motor performance (Scott, McNaughton, & Polman, 2006), and safety (Torsvall & Akerstedt, 1987). As a result, interest in eliminating the negative effects of sleep deprivation has increased (Tachibana, Ayas, & White, 2005). Napping, a simple and natural remedy to sleep deprivation, has been found to improve vigilant attention (Purnell et al., 2002; Sallinen et al., 1998) as measured by a psychomotor vigilance test (PVT). Despite this, it can also significantly decrease cognitive performance immediately after waking (Tassi & Muzet, 2000), as well as decrease the succeeding night's sleep (Dautovich, McCrae, & Rowe, 2008). Anecdotally, research suggests that short naps (< 30-min) and afternoon naps are preferable because they minimize a loss of nighttime sleep as well as the grogginess experienced when waking up (Hayashi, Motoyoshi, & Hori, 2005; Macchi et al., 2002; Buxton et al., 2000).

Therefore, this study will seek to demonstrate that there is a differential effect in PVT performance when partially sleep deprived between those who take a 20-minute nap in the afternoon compared to those who do not. Moreover, this may result in two sorts of effects: the nap might genuinely improve performance compared to a control condition (e.g., Mednick, Nakayama, & Stickgold, 2003), or it may simply counteract the negative effects of sleep deprivation (Mednick, et al., 2002; Sallinen et al., 1998) resulting in no change at all.

1.1 Sleep in Society

It has been found that the public perception of sleep's necessity is quite an underestimation (Dement and Vaughn, 2000). The time devoted to studying sleep disorders in medical school curriculums averages only 2.11 hours (Rosen et al., 1998), with no indication of this is increasing (Colten & Altevogt, 2006). Moreover, society would rather cope with sleep deprivation than get a good night's sleep (Mitler, et al., 1988), with as much as one in four respondents to a recent Gallup survey claiming that one couldn't be successful at a career and get a good night's sleep at the same time (National Sleep Foundation, 1995). Unfortunately, in its current state the American healthcare system is unable to meet the demands of sleep related health problems (Institute of Medicine, 2006). For example, over 20 million Americans are known to work shifts that require them to remain awake at night and sleep during the day, which can cause loss of sleep. Moreover, if loss of sleep occurs over long periods of time then this results in sleep debt which has been correlated with extensive negative health outcomes, such as diabetes and obesity, in addition to costing the healthcare system billions of dollars per year (Institute of Medicine, 2006).

Chronic partial sleep deprivation in young adults is quite common as studies have concluded that young adults with short Multi-Sleep Latency scores are more likely to suffer from chronic partial sleep deprivation rather than having normal trouble falling asleep (Carskadon & Dement, 1982). One National Sleep Foundation poll found that over half (58%) of teenage respondents slept less than 7 hours per night and almost a third have fallen asleep during school (NSF, 2014). Although parents who have rules enforcing bedtime behavior had children who slept on average 1.1 hours longer, enforcement is less likely to occur as children get older. As a result, teenagers without enforcement were twice as likely to fall asleep during school than

children with enforcement (NSF, 2014). In addition, parents also seem to underestimate their role as good models of sleep behavior. For example, parents who had electronic devices on in their bedrooms were also more likely to have teenagers who did as well, which was found to be significantly lower quality and quantity of sleep. This is a concern because over half (54%) of parents had at least two types of interactive electronics in their bedroom with 66% watching television or videos to help them fall asleep. Likewise, a National Sleep Foundation Poll in 2011 found that 1 in 10 Americans are likely to be woken by a cell phone a few times during the week and even more so for those under the age of 30. Overall, those who do use interactive devices within the hour of trying to fall asleep report significantly lower quality sleep (NSF, 2011). Regardless, it seems that this trend is getting worse as a National Sleep Foundation poll in 2005 found that 71% of adults are getting less than eight hours of sleep on weekdays compared to 68% of adults in 2002 (NSF, 2005). Likewise, it seems more are using caffeine to combat sleepiness as 74% of Americans in 2011 reported to consume at least one caffeinated beverage compared to 58% in 2008. Altogether, it was reported that at least 36% of the American population is suffering from sleep loss and this number is likely rising (Partinen, 1994; Bonnet & Arand, 1995). As a result, a practical and effective method for countering the harmful effects of sleep deprivation is needed that is alternative to caffeine, given the rising rates of sleep deprivation among young people.

1.2 Sleep and Accidents

Falling asleep at work is still a common occurrence and has caused some of the most severe anthropogenic disasters over the past century (Moss & Sills, 1981; Dinges et al., 1989). Dinges (1995) defined accidents as unintentional actions which are not random, uncontrollable

events. With this in mind, he found that up to 90% of serious accidents on the job occur due to human error across all industries. Although the percentage of which this is caused by fatigue is unknown, research shows that tiredness causes a real problem when operating transportation vehicles (Mitler et al. 1988).

The National Highway Traffic Safety Administration conservatively reports that sleepiness while driving is a causal factor in 83,000 crashes per year, resulting in 37,000 serious injuries and $1.5 billion in damages (Webb, 1995). Moreover, according to the National Sleep Foundation, this estimate is much higher in European countries, where sleepiness results in nearly 30% of car crashes. Despite preventing the effects of sleep related accidents with adequate sleep, the risk of accidents also depends on other factors, such as time spent driving and how challenging the drive is. For example, it was found that people are more likely to fall asleep while driving on long, boring, rural highways than in urban areas (National Sleep Foundation). Consequently, The National Transportation Safety Board found that 58% of truck driver crashes were caused by sleepiness, and that total time spent driving was a better predictor of accidents than driving experience when taking sleepiness into account (Lin et al., 1994). Moreover, one study found that of the 593 truck drivers surveyed, 47% had fallen asleep at the wheel at least once, with 25% having done so in the past year (McCartt et al., 2000). Additionally, truck drivers were more likely to work irregular shift hours, including night shifts, sleep less than 7 hours per night, and remain sedentary for long periods of time (Mitler et al., 1988; Marcus & Loughlin, 1996), with one study reporting that 62% of respondents drive on average 50 to 69 hours per week (Mitler et al., 1997). As mentioned previously by Harma et al. (1998), this results in increased amounts of insomnia, daytime sleepiness, and sleep deprivation, which may manifest while driving.

It follows that the more hours one works per week, the less time they would have for sleeping. As a result, physicians are also at significant risk of chronic partial sleep deprivation since it was found that residents in non-surgical rotations sleep on average 41.8 hours per week while surgical rotation residents sleep on average 38.3 hours per week (Fok et al., 2007) despite the recommended 56 hours of sleep per week (Carskadon & Dement, 2005). In addition, hospitals often encourage long working hours as restricting a physician's hours can be extremely expensive, with one study finding a required 71% increase in the number of physicians and 174% increase in the number of residents just to maintain the same workload (Payette, Chatterjee, and Weeks, 2009). Furthermore, some have postulated that working long hours on little sleep is seen as a source of pride among doctors (McCue, 1985, Brent, 1981). Regardless, sleep deprivation among physicians can seriously affect a physician's performance and patient's safety (Lewittes & Marshall, 1989; Landrigan et al., 2004). For example, it has already been found that interns who worked 76 hours per week compared to an average of 65 hours per week made 36% more medical errors (Landrigan et al., 2004). Likewise, residents who are fatigued due to lack of sleep reported significantly more surgical errors than those who were not (Kahol, et al., 2008) yet it is not uncommon for first year residents to work frequent 24 hour shifts (Steinbrook, 2002). As a result, more attention should be given to strategies that alleviate the negative effects of sleep deprivation especially in settings where there is a high amount of risk associated with performance errors.

1.3 Sleep Physiology

A major milestone in sleep research has been the use of electroencephalography (EEG), a gross measure of electrical activity of the brain, and electrooculography(EOG), a measure of the electrical activity of the eye (Loomis et al., 1937). These diagnostic tools have enabled researchers to further develop an understanding of sleep (Aserinsky & Kleitman, 1953), as well as theories that can be correlated to other fields, such as neurology, physiology, endocrinology, and psychiatry (Shephard et al., 2005). As a result, there are a number of paradigms in sleep research which form its foundation.

One of the most fundamental advancements in sleep research was the staging of sleep, first introduced by Loomis et al. (1937). There are five stages of sleep divided into two parts: non-rapid eye movement (NREM) sleep, which consists of the first four stages, and rapid eye movement (REM) sleep, the fifth stage. Sleepers proceed consecutively through the four stages of NREM sleep, enter REM sleep, and then reverse back through the stages of NREM sleep to stage 1. This process occurs cyclically throughout the night, with each cycle lasting approximately 70-120 minutes. Additionally, it has been found that the average time spent in REM sleep and stage 2 NREM sleep increases with each cycle while the time spent in stage 3 and 4 decreases (Carskadon & Dement, 2005). The following stages of NREM sleep and REM sleep are described in further detail below:

Stage 1

The first stage of NREM sleep consists of alpha waves, similar to a relaxed awake state, and usually is less than 10 minutes. This period marks the transition of wakefulness to a drowsy

sleep state that is easily disrupted and the muscles are still active. The EEG pattern is a mixture of low voltage waves in a frequency range of 8 – 13 hertz.

Stage 2

Is defined by low-voltage theta waves (4-8 Hz) with the appearance of occasional high-voltage fluctuations known as k-complexes and sleep spindles**.** Likewise, this stage increases in length with each successive cycle, and the individual is more resistant to arousal than the first stage. Recently, new evidence has emerged that sleep spindle oscillations might be involved in long term cortical network reorganization when earning (Wilson, & McNaughton, 1994), however little is known about the function of k-complexes (Crowley et al., 2002).

Stage 3 & Stage 4

These two stages mark the end of NREM sleep, and are highlighted by a transition to delta waves (0.5-4Hz) in what is known as slow wave sleep (SWS). Stage three is an extremely short lasting period characterized by the appearance of 20% - 50% of delta waves, whereas stage four consists of more than 50% of delta waves. These stages are not as prevalent in the elderly as they are in others (Dijk et al., 2010), however a disruption causes a rebound of slow-wave sleep across all age groups, possibly because it is required for consolidation of declarative memory (Rauchs et al., 2005), and metabolic restoration in the brain (Benington &Heller, 1995).

In these stages the individual is the most resistant to arousal, while brain temperature, breathing rate, heart rate and blood pressure are at their lowest point during this stage. Events such as bedwetting, sleep walking, and night terrors also occur during this stage. Often, when

awakened the individual feels grogginess, also known as sleep inertia, which causes a decrease in performance and can last up to 30 minutes (Tassi & Muzet, 2000).

REM Sleep

Rapid eye movement sleep (REM) is characterized by low-voltage mixed-frequency brain waves consisting of theta waves, alpha waves, and sometimes beta waves, as well as rapid eye movements, rapid breathing, increased heart rate and blood pressure, and sometimes core body temperature. In light of this, REM sleep is referred to as paradoxical sleep as the EEG pattern, rapid eye movements, and physiological indicators reflect an awake state, however muscle activity is completely paralyzed and the person remains unresponsive to external stimuli. REM sleep deprivation studies have not clearly yielded the purpose of REM sleep as REM sleep deprivation has not caused any harm to healthy participants, although REM sleep rebound was observed (Vogel, 1975). Despite this, REM sleep is critical in childhood development as babies spend up to 50% of their sleep time in REM sleep which decreases to 60% by 6 months of age (National Sleep Foundation). In addition, REM sleep has shown in some studies to be necessary for memory consolidation (Rauchs et al., 2005), while others hypothesis that it serves to erase unwanted patterns of neural activity (Purves, et al., 2001).

Despite the ground-breaking work done on sleep stages, little was known on how the process of sleep and wakefulness was regulated until 1982 when Alexander Borbely discovered the two-processes model of sleep regulation (Borbely, 1982). This model postulates that sleep and wake states are regulated by two major processes: the sleep-wake homeostasis process (Process S) and circadian rhythms (Process C).

In order to describe Borbely's (1982) model, wakefulness must first be described. Wakefulness is characterized by beta waves (12-30 Hz) and gamma waves (25-100 Hz) on an EEG that are low-voltage and desynchronized. In order for wakefulness to occur, the forebrain must be activated by excitatory neurons. This is done by the ascending arousal system (AAS) which consists of two neuronal pathways in the brain stem (Saper et al., 2005). Therefore, for sleepiness to occur the AAS must be inhibited, and this occurs as a build-up of sleep-inducing substances, particularly adenosine, have been shown to inhibit parts of the AAS (McCarley, 2007). Furthermore, extracellular adenosine levels, a marker of spent metabolic energy (Masino & Boison, 2013), has also been found to inhibit the AAS (McCarley, 2007). In addition, a sleep-promoting area has also been identified in the hypothalamic preoptic area (Saper et al., 2005) which inhibits the AAS and can be inhibited by it in turn. As a result, these sleep-homeostatic factors function as a timer where the propensity to sleep is greater the longer the time spent awake.

The second part of the two process-model of sleep regulation are circadian rhythms. These refer to biological processes that cycle around a 24-hour clock in response to external cues, such as daylight and darkness (Goichot et al., 1998). All animals and plants have an internal circadian clock, however in humans it is found in the superchiasmatic nucleus of the hypothalamus (Dunlap, et al., 2004). In addition, input from nerve cells that detect brightness in the retina are transmitted to the superchiasmatic nucleus which resets the molecular clock genes. These clock genes express two proteins which enter the nucleus and activate transcription of an additional gene which exits the nucleus and suppresses the expression of the clock genes. The superchiasmatic nucleus then signals the rest of the body to bring it into synchrony with this cycle which circulates every 24 hours approximately (Jin et al., 1999). Likewise, the

superchiasmatic nucleus controls the secretion of the hormone melatonin in response to darkness in order to maintain this cycle and the activity of other hormones (Dunlap et al., 2004). Consequently, circadian rhythms rise and fall cyclically every 24 hours as a function of its molecular clock and external cues. Moreover, there is a dip in circadian rhythm which varies between individuals but predominately occurs between 1:00pm-4:00pm (Monk, et al., 1996) resulting in a natural inclination to sleep during this time (Broughton, 1998).

1.4 Sleep deprivation studies

The negative outcomes of sleep deprivation on cognition are widely documented and include the following (Durmer & Dinges, 2005; Harrison & Horne, 2000; Olsen, Pallesen, & Eid, 2010):

- Delayed reaction times (RT)
- Decline in short-term memory and increased variability in psychomotor performance
- Impaired moral judgment
- Decline in decision making
- Decline of vigilant attention
- Learning difficulties
- Uncontrollable Microsleeps

Interestingly, even high amounts of sleep deprivation does not inhibit the ability to carry out tasks, rather it prevents performance at a consistent level over time because it disrupts vigilant attention, which is ability to sustain attention (Lim & Dinges, 2008; Goel et al., 2009). Thus, research aimed at alleviating the effects of sleep deprivation on performance has resulted

in a new theorem known as the state-instability hypothesis (Goel et al., 2009). This builds on the two-state process of sleep which dictates that the variability observed in performance is a result of an increased drive for sleep that disrupts vigilant attention resulting in an unstable state (Doran, Van Dogen, & Dinges, 2001; Lim & Dinges, 2008; Durmer & Dinges, 2005). Namely, the circadian rhythms and sleep-homeostatic factors are competing with each other which results in an unstable state of attention that becomes more variable as the time spent awake increases because the sleep-homeostatic drive also increases (Graw et al., 2004). Although the mechanism that disrupts vigilant attention is unclear, some have suggested that the effects are caused by microsleeps (Dorrian et al., 2005), which are short, momentary periods of sleep when the individual unknowingly becomes unconscious and unresponsive to stimuli (Horne et al., 2003). Indeed, EEG recordings show that waking alpha waves suddenly shift to theta waves during microsleeps corresponding to stage 2 of NREM sleep (Priest et al., 2001). Moreover, microsleeps increase in frequency and duration of as the time spent sleep deprived increases resulting in increased variability of cognitive performance (Lim & Dinges, 2008).

Studies of the effects of sleep deprivation on vigilant attention make use of the psychomotor vigilance test (PVT), the most common test used to measure vigilant attention and alertness (Dinges & Powell, 1985). It is similar to a simple response time test where a participant a key in response to a visual stimulus that appears randomly on a computer screen; however, it differs in that there is a randomized inter stimulus interval, typically 2-10 seconds, with the whole test taking 10 minutes. For this reason, the test can capture the effects of sleep deprivation because performance typically decreases over time for monotonous tasks when sleep deprived. This performance decrement is indicative of failing vigilant attention (Lim & Dinges, 2008). In addition, the PVT test has been found to be highly reliable when dealing with chronic partial

sleep deprivation, and acute total sleep deprivation (Basner & Dinges, 2011); although, it is unclear whether this holds for acute partial sleep deprivation. Overall, studies using PVT have consistently shown that sleep deprivation leads to a general slowing of all but the fastest RT's, with the slowest becoming increasingly variable, in addition to an increased number of lapses (Lim & Dinges, 2008; Doran et al., 2001).

PVT tests are flexible as there are different metrics which capture different aspects of sleep deprivation. For example, the mean response time, number of lapses and errors of commission, fastest 10% response time, median response time, mean reciprocal response time, and slowest 10% response time, are all valid, however they differ in sensitivity depending on the type of sleep deprivation (Basner & Dinges, 2011). Nevertheless, many researchers transform RT data and use mean RT for analysis (Marmolejo-Ramos et al., 2014), despite RT's not following a normal distribution as they are positively skewed (Hohle, 1965; Ratcliff & Murdock, 1976). As a result, analysis of reaction times often involves trimming extreme outliers (Marmolejo-Ramos & Matsunaga, 2009) despite this resulting in relevant information being excluded. Moreover, there is no standard method of trimming the data, and researchers do not agree on the criteria to do so (Heathcote, Popiel, & Mewhort, 1991). As a result, many have suggested that an ex-Gaussian distribution best describes PVT outcomes and should be used (Heathcote, Popiel, & Mewhort, 1991; Parris, Dienes, & Hodgson, 2013).

An ex-Gaussian distribution is a convolution of a Gaussian distribution and an exponential distribution, and is described by three parameters: mu, sigma, and tau (**Figure 1**). Mu and sigma, refer to the mean and standard deviation of the Gaussian portion respectively. Tau reflects the mean and standard deviation of the exponential portion (Heathcote, Popiel, & Mewhort, 1991). Although it has been argued that ex-Gaussian components mirror cognitive

processes (Hohle, 1965) others have demonstrated this is inconclusive (Matzke & Wagenmakers, 2009; Heathcote, Popiel, & Mewhort, 1991). Regardless, an ex-Gaussian distribution remains an effective method for describing RTs because most RT distributions have a positive skew (Luce, 1986). For example, Heathcote, Popiel, & Mewhort (1991) used an ex-Gaussian analysis to assess RT distributions in a Stroop task. The Stroop effect refers to performance disadvantage when naming the color of the font of a word that is incongruent with the word (e.g., the word "RED" written in blue font). Heathcote, Popiel & Mewhort, (1991) found that mu was faster for the congruent trial, relative to the neutral baseline (e.g., "XXX"), while tau was slower for both congruent and incongruent trials compared to the baseline. This work demonstrated the three parameters of an ex-Gaussian analysis offer a greater degree of sensitivity for analyzing RT data than a single parameter (i.e., the mean).
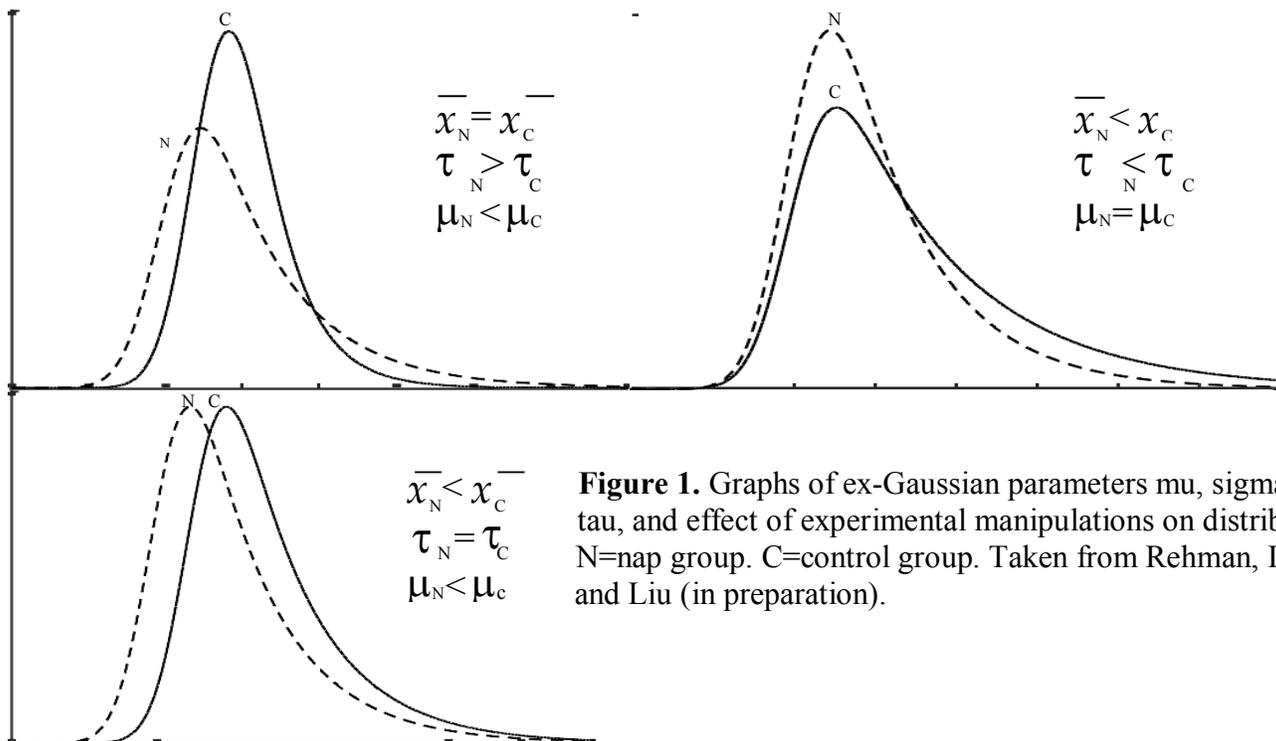

$$\overline{x}_N = \overline{x}_C$$
$$\tau_N > \tau_C$$
$$\mu_N < \mu_C$$

$$\overline{x}_N < \overline{x}_C$$
$$\tau_N \leq \tau_C$$
$$\mu_N = \mu_C$$

$$\overline{x}_N < \overline{x}_C$$
$$\tau_N = \tau_C$$
$$\mu_N < \mu_C$$

Figure 1. Graphs of ex-Gaussian parameters mu, sigma and tau, and effect of experimental manipulations on distribution. N=nap group. C=control group. Taken from Rehman, Ivanoff, and Liu (in preparation).

1.5 Napping

Many studies also seek to identify countermeasures for the negative effects of sleep deprivation, and napping has consistently shown to be an effective method (Brooks & Lacks, 2006). However, it has also been found that longer naps have immediate drawbacks. Upon waking from a long nap (>2 hours) there is a period of grogginess, known as sleep inertia, which negatively affects performance and can last up to 30 minutes (Tassi & Muzet, 2000). As a result, power naps (20 min) have been found to counter sleep inertia better compared with longer naps (Hayashi, Motoyoshi, & Hori, 2005; Takahashi & Arito, 1998; Tietzel & Lack, 2002).

In addition to the length of the nap, research also shows that the timing of the nap is important. The evening and morning have been called a "no-nap zone" by researchers as naps at this time tend to decrease nighttime sleep (Pigeon, Sateia, & Ferguson, 2003; Shen, Barbra, & Shapiro, 2006; Lavie, 1985; Strogatz, 1986). It has been found that exposure to darkness and sleep during these times has led to a phase shift of circadian rhythms that possibly disrupt nighttime sleep (Goichot et al., 1998; Van Cauter et al., 1998), however, this finding did not hold for naps taken in the afternoon (Buxton et al., 2000). The afternoon is an ideal "nap zone" as there is natural dip of circadian rhythms during this period which causes a decrease in cognitive functioning, mood, and other physiological processes (Giannotti et al., 2002). In fact, many cultures already do take naps in the afternoon (Dinges & Broughton, 1989) because it's the period during the day which is most likely to cause increased levels of sleepiness (Broughton, 1998). Although, the mechanisms as to why afternoon naps lead to better sleep efficiency and shorter sleep latency (Lavie & Weler, 1989) is unknown, it is evident that the timing of naps has important implications for nighttime sleep.

Milner and Cote (2009) identified several key napping studies that show the benefits of napping outlined in **Table 1**, however none of them have used an ex-Gaussian distribution to describe or analyze the results. Furthermore, none of them have sought to determine the effects of power naps taken in the afternoon. As a result, the objective of this study is to determine whether the 20-min power nap taken in the afternoon improves RT from a PVT when partially sleep deprived.

Table 1. Summary of studies looking at effects of nap length on different measures for sleep deprived and non-sleep deprived subjects.

Author(s)	Methods	Results
Smith et al., (2007)	9 participants in a within-subject design comparing 30-min & no-nap conditions during shift work	Naps significantly improved PVT response times & subjective sleepiness
Purnell et al., (2002)	24 participants in a within-subject design comparing 20 min nap & no nap conditions during shift work	Naps improved reaction time & vigilance but not subjective sleepiness
Takahashi & Arito (2000)	12 participants in within-subjects design comparing 15 min & no nap conditions on 4h sleep	Naps improved accuracy on a task measured by P300 event-related potential
Sallinen et al., (1998)	14 participants in within subject's design comparing 30 & 50 min naps to no nap during night shift	Naps reduced lapses on reaction time test

1.6 Current Study

The purpose of the current study is to determine whether a short 20-min nap affects the RT distribution of the PVT. Utilizing a power nap reflects research which anecdotally suggests this is effective for countering sleep inertia while the time of the nap taken in the afternoon reflects research which suggests this is beneficial for minimizing nighttime sleep disruption.

2. Methodology

Participants

Twelve healthy men and women between the ages of 18 and 35 with normal sleeping patterns were recruited from Saint Mary's University campus, and randomly assigned to either the nap or no-nap condition without their knowledge. Seven participants were in the no-nap condition and five in the nap condition. Participants had normal vision or corrected to normal vision by contact lenses, and were free of permanent metal, as established by an interview. All participants gave informed consent and REB approval was obtained prior to conducting the study. Participants were also compensated with a stipend of $125.

Design

   Night 1

Participants arrived at the Chronobiology Lab located in the Abbie Lane Building of Nova Scotia Health Authority around 11:00pm. After a brief introduction to the bedroom and amenities they were instructed to sleep and the lights were shut off between 11:15pm and 12:00pm. The lights were then turned back on at 7am the following morning so that each participant had eight hours of sleep time in darkness. Upon awakening they were allowed to leave the lab for the remainder of the day.

   Night 2

The participants arrived at the Chronobiology Lab at 11:00pm and upon arrival, completed the first PVT (PVT1) at 11:10 pm. The participants then returned to their rooms and were told not to sleep until 4:00 am and their lights were kept on during this time. Participants could access Wi-

Fi, movies, board games, or converse with the research assistant to stay awake. At 4:00am the lights were shut off and participants had 3 hours of sleep time until 7:00 am when the lights were switched on. There was one instance a when a PVT was mistakenly administered at 3:30am to a participant however this test result was not included in the analysis. After waking they were instructed and monitored to remain awake for the remainder of the day. Breakfast was provided at 8:00am in the cafeteria. At 10:00am the participants completed their second PVT (PVT2). Lunch was then provided at 12:00pm. Participants in the nap condition were notified at 2:30pm they were in the nap condition and were allowed a 20-minute nap in their rooms at 2:40pm with the lights shut off. If they were not in the nap condition, they remained awake during this time. The participants then completed their third PVT (PVT3) at 3:30 pm, marking the completion of their participation. They were provided taxi slips in order to get home safely. A summary of the design is shown in **Figure 2.**
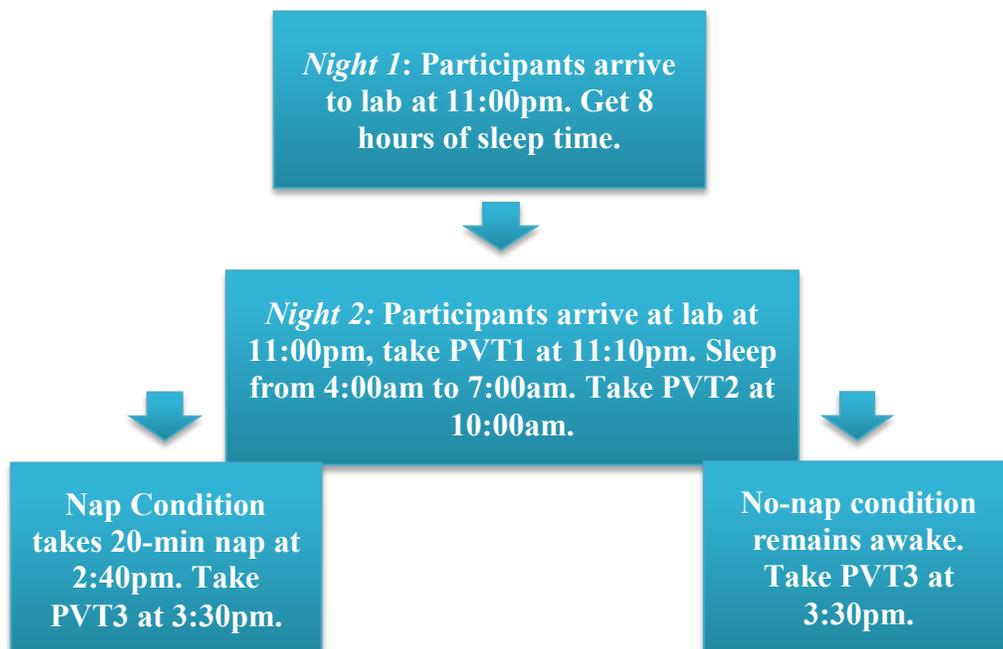

Figure 2. Experimental design of study. PVT (1-3) refer to first, second and third PVT sessions respectively.
PVT

The psychomotor vigilant test used was computer-based and created using a custom-made script on matlab using the Psychophysics Toolbox extensions (Brainard, 1997; Pelli, 1997). The PVT took approximately 10-mintues to complete. Counter stimuli were presented with a random inter-stimulus interval of 2-10 seconds. Participants began the test by pressing "G", and were instructed to press "B" as soon as possible whenever the red rectangle which appeared on the screen turned yellow. RT's that occurred before the counter target were considered false starts, and RT's slower than 500ms were considered lapses. After 30 seconds of inactivity, the target timed out and the volunteer was presented with feedback (i.e., the message "OVERUN" was displayed). The outcome metrics obtained were mean response time (meanRT), inverse mean response time (invRT), lapses, and the ex-Gaussian parameters mu, sigma, and tau.

Statistical Analyses

A Kolmogorov-Smirnov test was conducted to determine whether the ex-Gaussian parameters were significantly different from an ex-Gaussian distribution based on 10,000 samples of random numbers using the parameters from the fit. A Wilcoxon (independent) rank-sum test was performed between the outcome measures from the nap and no-nap conditions for at PVT2 and PVT3. A Wilcoxon (dependent) signed-rank test was done between PVT2 and PVT3 within nap and no-nap conditions. Using JASP (v0.8.1), a calculation of Cohen's d was done for the outcome metrics in each of the tests conducted. Additionally, using G*Power (Faul, Erdfelder, Buchner, & Lang, 2007), a power analysis for determining the sample size required to find a significant effect for the outcome metrics was performed for each outcome measure.

3. Results

The raw RT's for each session and participant were used to calculate the ex-Gaussian parameters using the *egfit* function in Matlab (Mathworks, Natick, MA) provided by Lacouture and Cousineau (2008). These functions use the *fminsearch* implementation of the simplex search method to find an optimized fit of the ex-Gaussian to the data. Before the data was fit, RT's were trimmed by specifying a RT's longer than 1.5s as lapses. The fits were assessed by using a Kolmogorov-Smirnov test of the actual data distribution and a randomly generated distribution (from 10,000 samples) using the parameters from the best fit. The Bonferroni corrected p-value for the K-S test was 0.0014. Based on this, the results of the K-S test (**Table 2**) showed that all ex-Gaussian parameters provided a reasonable fit to the empirical data.

Table 2. P-values of K-S test for each PVT session.

Subject	Nap Condition	Session 1	Session 2	Session 3
1	No	0.0753	0.0423	0.9185
2	No	0.1739	0.4340	0.1988
3	No	0.1876	0.3467	0.4322
4	Nap	0.0576	0.0453	0.0408
5	No	0.2312	0.0967	0.0462
6	Nap	0.0691	0.5560	0.15456
7	Nap	0.1496	0.5011	0.4838
8	Nap	0.0461	0.7152	0.6331
9	No	0.3445	0.4627	0.6478
10	No	0.6432	0.7588	0.8066
11	Nap	0.9201	0.7822	0.2998
12	No	0.3118	0.2146	0.3071

Results of the Wilcoxon test between nap conditions for PVT2 (**Table 3**) showed that none of the outcomes significantly differed between conditions. However, at PVT3 the ex-Gaussian parameter mu and invRT were significantly different between nap conditions (**Table**

4). There was no significant difference between any of the PVT outcome measures at PVT2 and

PVT3 for the nap group (**Table 5**) or no-nap group (**Table 6**).

Table 3. Results of the Wilcoxon test between nap condition and no-nap condition for outcome metrics tested at PVT2.

Outcome	p-value
mu	0.2020
sigma	0.4318
tau	0.5303
lapses	0.2475
meanRT	0.2677
invRT	0.2020

Table 4. Results of the Wilcoxon test between nap condition and no-nap condition for outcome metrics tested at PVT3.

Outcome	p-value	Cohen's d	Predicted (N)
mu	0.0480	-1.218	11
sigma	0.4318	-0.657	33
tau	0.2020	-0.836	21
lapses	0.0657	-1.285	11
meanRT	0.0732	-1.016	15
invRT	0.0480	0.966	16

Note: Positive Cohen's d reflects a decreasing outcome with increasing effect size. Negative Cohen's d reflects an increasing outcome with increasing effect size.

Table 5. Results of the Wilcoxon test between PVT2 and PVT3 for outcome metrics tested in the nap condition.

Outcome	p-value	Cohen's d	Predicted (N)
mu	0.0625	1.687	7
sigma	0.6250	0.190	362
tau	0.8125	-0.016	50763
lapses	1	0.135	715
meanRT	0.1250	1.125	13
invRT	0.1250	-1.401	9

Note: Positive Cohen's d reflects a decreasing outcome with increasing effect size. Negative Cohen's d reflects an increasing outcome with increasing effect size.

Table 6. Results of the Wilcoxon test between PVT2 and PVT3 for outcome metrics tested in the no-nap condition.

Outcome	p-value	Cohen's d	Predicted (N)
mu	0.1562	0.620	36
sigma	0.6875	0.321	129
tau	0.2188	-0.775	24
lapses	0.1250	-0.815	22
meanRT	1	0.069	2732
invRT	0.6875	-0.168	463

Note: Positive Cohen's d reflects a decreasing outcome with increasing effect size. Negative Cohen's d reflects an increasing outcome with increasing effect size.

4. General Discussion

As predicted, all participants RT's from each PVT session followed an ex-Gaussian distribution. Although this result agrees with previous findings (Parris, Dienes, & Hodgson, 2013; Balota & Yap, 2011) it has been found that in 2010, among 285 articles which used RT measures for sleep deprivation 95% mainly utilized mean RT (Balota & Yap, 2011). This presents a problem because the data is not normally distributed thus trimming the data for extreme outliers and using the mean to describe the distribution is dangerous as it doesn't provide an accurate picture of the results (Heathcote, Popiel, & Mewhort, 1991).

At Session 2 it was found that there was no significant difference for any of the PVT outcome metrics between nap conditions. This result supports the value of Session 2 as a baseline and it was expected given that all the participants had undergone the same procedure at this time. At Session 3, the outcomes mu and inverse of the mean RT were better for the nap group than the no-nap group. There was no effect of group on the other metrics. However, it seems that those outcomes non-significantly affected by the nap were under-powered. Indeed, a

power analysis (G*Power; Faul et al., 2009) suggested that increasing the sample by just nine participants would likely have led to a significant advantage for the nap group with the tau, lapses, and meanRT outcomes 95% of the time. This result suggests that napping improves a wide variety of outcomes, however, it seems it is more likely to improve overall performance (mu) rather than the counteract the negative effects of sleep deprivation. This is important because we can conclude napping may lead to harm reduction. For example, it's been noted that the majority of trucking accidents in Washington that involved transporting hazardous materials was a result of inattention (Richards, 1986) likely due to sleep debt (Mitler et al., 1988). Moreover, trucking in general makes vigilant attention more susceptible to the effects of sleep deprivation because of its long repetitive nature (McCartt et al., 2000). As a result, harm associated with sleep deprivation can be reduced by taking 20-min naps in the afternoon which might improve the top-down neurobiological mechanisms involved in sustaining attention (Goel et al., 2009).

The conclusion that napping actually improves wakefulness mechanisms is also supported by the results from the test between PVT2 and PVT3 for the nap condition and no-nap condition. It was found that there was no significant difference for any of the outcome metrics, however, high effect sizes for mu, meanRT and invRT, and results from the power analyses suggest that these outcomes were likely underpowered. In addition, the effect size for mu, meanRT, and invRT decreased, while it increased for tau, and lapses from PVT2 to PVT3 for the no-nap condition. Furthermore, the power analyses showed that the sample size leading to a significant advantage actually increased for mu, meanRT, and invRT compared to the nap condition, but for tau and lapses it decreased compared to the nap condition. According to the state-instability hypothesis, variability increases as time spent sleep-deprived increases, due to

the increased frequency and duration of lapses of vigilant attention, hypothesized to be caused

microsleeps (Doran, Van Dogen, & Dinges, 2001). Tau captures the increased variability well

since lapses of vigilant attention would increase the slowest RT (Doran, Van Dogen, & Dinges,

2001; Lim & Dinges, 2008), however, it is unclear whether naps would improve the variability

thereby shortening tau. This study provides weak evidence for the state-instability hypothesis as

the results suggest that sleep deprivation increases variability of RT over time, as seen in tau.

However, our results also showed that napping has an effect on the ex-Gaussian component mu,

resulting in a universal improvement of RT in addition to the slow RT. That napping improved

mu but not tau, points to a role of vigilant attention. Had napping improved state-instability, we

would have expected to see a selective effect on tau, and not mu. Our findings are consistent

with studies demonstrating that naps improve levels of wakefulness (Macchi et al., 2002; Song et

al., 2002).

Our finding of an effect of napping on mu also supports the idea that short naps may

reduce the harm associated with sleep deprivation. The greater need for motivated behavior to

counteract the increased variability of cognitive performance has been shown to lead to errors of

commission (Goel et al., 2009). We did not observe any effect of napping on false starts,

suggesting our finding is not due to motivational changes.

In conclusion, we observed that napping has a selective effect on PVT performance: it

improved the ex-Gaussian parameter mu, but did not strongly affect tau, sigma, lapses or false

starts. The pattern of results suggests that a short nap, following a night of restricted sleep,

improved vigilant attention. One major limitation to this study was the sample size, and thus this

issue should be addressed in future research. Future research could also look at mapping

cognitive mechanisms onto ex-Gaussian parameters in order to better understand why mu and

tau is differentially affected. Although some have said that research does not suggest any underlying cognitive mechanisms (Matzke & Wagenmakers, 2009), they did not consider models using RT's from a PVT. In addition, it may be informative to test further from the time the nap is taken, in an attempt to determine how long the improvements from a nap last. We currently do not know whether performance improvements from a nap are long-lasting. In addition, more research is needed to determine whether microsleeps are the cause of the increased variability of RT seen in sleep deprivation studies (Williams & Lubin, 1959; Torsvall & Akerstedt, 1987). Overall, 20-min naps in the afternoon improved overall vigilant attention following sleep restriction which is beneficial because there is a reduction of the harm associated with sleep deprivation.

Works Cited

Akerstedt, T. (1998). Shift work and disturbed sleep/wakefulness. *Sleep Medicine Reviews, 2*, 117-128.

Alhola, P., & Polo-Kantola, P. (2007). Sleep deprivation: Impact on cognitive performance. *Neuropsychiatric Disease and Treatment*, 3(5), 553-567.

Aserinsky, E., & Klietman, N. (1953). Regularly occurring periods of eye motility, and concomitant phenomena, during sleep. *Science,* 118(3062), 273-274

Basner, M., & Dinges, D.F. (2011). Maximizing sensitivity of the psychomotor vigilance test (PVT) to sleep loss. *Sleep,* 34(5), 581-91.

Benington, J.H., & Heller, H.C. (1995). Restoration of brain energy metabolism as the function of sleep. *Progressive Neurobiology,* 45, 347–60

Buxton, O.M., L'Hermite-Baleriaux, M., Turek, F.W., & van Cauter, E. (2000). Daytime naps in darkness phase shift the human circadian rhythms of melatonin and thyrotropin secretion. *American Journal of Physiology – Regulatory, Integrative and Comparative Physiology*, 278(2), R373-82.

Balota, D.A., & Spieler, D.H. (1999). Word frequency, repetition, and lexicality effects in word recognition tasks: Beyond measures of central tendency. *Journal of Experimental Psychology: General,* 128, 32-55

Balota, D.A. & Yap, M.J. (2011). Moving beyond the mean in studies of mental chronometry: The power of response time distributional analyses. *Current Directions in Psychological Science*, 20(3), 160-166.

Balota, D.A., Yap, M.J., Cortese, M.J., & Watson, J.M. (2008). Beyond mean response latency: Response time distributional analyses of semantic priming. *Journal of Memory & Language*, 59, 495-523.

Bonnet, M.H., & Arand, D.L. (1995). We are chronically sleep deprived. *Sleep,* 18(10), 908-911.

Borbely, A.A. (1982). A two-process model of sleep regulation. *Human Neurobiology*, 1(3), 195-204

Brainard, D.H. (1997). *The Psychophysics Toolbox,* Spatial Vision, 10:443-446.

Brent, D.A. (1981). The residency as a developmental process. *Journal of Medical Education*, 56, 417-422.

Brooks, A., & Lack, L. (2006). A brief afternoon nap following nocturnal sleep restriction: Which nap duration is most recuperative? *Sleep,* 29(6), 831-840.

Broughton, R.J. (1998). SCN controlled circadian arousal and the afternoon "nap zone". *Sleep Research Online,* 1, 166-78

Carskadon, M.A., & Dement, W.C. (1982). Current perspectives on daytime sleepiness: The issues. *Sleep,* 5(2),S56-66

Carskadon, M.A., & Dement, W.C. (2005). *Normal human sleep: An overview*. Philadelphia: Elsevier Saunders; 13–23.

Colten, H.R., & Altevogt, B.M. (2006). *Sleep disorders and sleep deprivation: An unmet public health problem.* (Eds). Institute of Medicine (US) Committee on Sleep Medicine and Research; Washington (DC): National Academies Press (US)

Crowley, K., Trinder, J., Kim, Y., Carrington, M., & Colrain, I.M., (2002). The effects of normal aging on sleep spindle and K-complex production, *Clinical Neurophysiology*, 113(10), 1615-1622

Dautovich, D.N., McCrae, S.C., & Rowe, M. (2008). Subjective and objective napping and sleep in older adults: Are evening naps "bad" for nighttime sleep? *Journal of the American Geriatrics Society,* 56(9), 1681-1686.

Dement, W.C., & Vaughan, C.C. (2000). *The promise of sleep: A pioneer in sleep medicine explores the vital connection between health, happiness, and a good night's sleep.* Published by Macmillian: Milton Keyes, United Kingdom.

Dijk, D.J., Groeger, J.A., Stanley, N., & Deacon, S. (2010). Age-related reduction in daytime sleep propensity and nocturnal slow wave sleep. *Sleep,* 33(2), 211-223.

Dinges, D.F. (1995). An overview of sleepiness and accidents. *Journal of Sleep Research, 4 (2),* 4-14

Dinges, D.F., Graeber, R.C., Carskadon, M.A., Czeisler, C.A., & Dement W.C. (1989). Attending to inattention. *Science,* 245(4916), 342.

Dinges, D.F., & Broughton, R.J. (1989). *Napping: A ubiquitous enigma.* In D.F. Dinges and R.J. Broughton (Eds) Sleep and Alertness: Chronobiological, Behavioral, and Medial Aspects of Napping. Raven Press, New York, NY, 1-7

Dinges, D.F. & Powell, J.W. (1985). Microcomputer analyses of performance on a portable, simple visual RT task during sustained operations. *Behavior Research Methods*, 17, 652-655.

Doran, S.M., Van Dongen, H.P., & Dinges, D.F. (2001). Sustained attention performance during sleep deprivation: evidence of state instability. *Archives Italiennes de Biologie,* 139, 253–67.

Dorrian, J., Rogers, N.L., & Dinges, D.F. (2005). *Psychomotor vigilance performance: Neurocognitive assay sensitive to sleep loss.* New York: Marcel Dekker; pp. 39–70.

Dunlap, J.C., Loros, J.J. & DeCoursey, P. (2004). Chronobiology: Biological Timekeeping. (Sunderland, M.A: Sinauer Associates).

Durmer J.S., & Dinges D.F. (2005). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology*. 25(1), 117–129

Faul F., Erdfelder E., Buchner A., & Lang A.G. (2009). Statistical power using G*power 3.1: Tests for Correlation and regression analyses. *Behavior Research Methods,* 41, 1149-1160.

Fok, M.C., Townson, A. Hughes, B. & Miller, W.C. (2007). Work hours, sleep deprivation and fatigue: A British Colombia snapshot. *British Colombia Medical Journal,* 49(7), 387-392.

Giannotti, F., Cortesi, F., Sebastiani, T., & Ottaviano, S. (2002). Circadian preference, sleep and daytime behavior in adolescence. *Journal of Sleep Research*, 11(3), 191-199.

Goel, N., Rao, H., Durmer, J.S., & Dinges, D.F. (2009). Neurocognitive consequences of sleep deprivation. *Seminars in Neurology,* 29(4), 320-339

Goichot B., Weibel L., Chapotot F., Gronfier C., Piquard F., & Brandenberger G. (1998). Effect of the shift of the sleep-wake cycle on three robust endocrine markers of the circadian clock. *American Journal of Physiology - Endocrinology and Metabolism*. 275, E243–E248.

Graw, P., Krauchi, K., Knoblauch, V., Wirz-Justice, A., & Cajochen, C. (2004). Circadian and wake-dependent modulation of fastest and slowest reaction times during psychomotor vigilance task. *Physiology & Behavior*. 80, 695-701.

Harma M., Tenkanen, L., Sjoblom, T., Alikoski, T., & Heinsalmi, P. (1998). Combined effects of shift work and life-style on the prevalence of insomnia, sleep deprivation and daytime sleepiness. *Scandinavian Journal of Work, Environment, and Health, 24(4),* 300-307

Harrison, Y., & Horne, J.A. (1996). Occurrence of microsleeps during daytime sleep onset in normal subjects. *Electroencephalography and Clinical Neurophysiology,* 98, 411–4.

Harrison, Y., & Horne, J.A. (2000). The impact of sleep deprivation on decision making: A review *Journal of Experimental Psychology: Applied*, 6(3), 236-249.

Hayashi, M., Motoyoshi, N., & Hori, T., (2005). Recuperative power of a short daytime nap with or without stage 2 sleep. *Sleep*, 28(7), 829-836.

Heathcote, A., Popiel, J.S., & Mewhort, D.J.K. (1991). Analysis of response time distributions: An example using the stroop task. *Quantitative Methods in Psychology*, 109, 340-347.

Hohle, R.H. (1965). Inferred components of reaction times as functions of foreperoid duration. *Journal of Experimental Psychology,* 69(4), 382-386.

Horne, J.A., Reyner, L.A, & Barrett, P.R. (2003). Driving impairment due to sleepiness is exacerbated by low alcohol intake. *Occupational and Environmental Medicine,* 60, 689–692.

Institute of Medicine. (2006). S*leep Disorders and Sleep Deprivation: An Unmet Public Health Problem.* Washington, DC: The National Academies Press. Doi: 10.17226/11617.

JASP Team (2016). JASP (Version 0.8.0.0) [Computer software]

Jin, X., Shearmen, L.P., Weaver, D.R., et al., (1999). A molecular mechanism regulating rhythmic output from the superchiasmatic circadian clock. *Cell,* 96(1), 57-68.

Kahol, K., Leyba, M.J., Deka, M., Deka, V., Mayes, S., Smith, M., Ferrara, J.J., & Panchanathan, S. (2008). Effect of fatigue on psychomotor and cognitive skills. *American Journal of Surgery,* 195(2), 195-204.

Karalunas, L.S., Guerts, H., Konrad, K., & Nigg, T.J. (2014). Annual research review: Reaction time variability in ADHD and autism spectrum disorders: Measurement and mechanisms of a proposed trans diagnostic phenotype. *Journal of Child Psychology and Psychiatry*, 55(6), DOI: 10.1111/jcpp.12217

Killgore, W.D., Balkin, T.J., & Wesensten, N.J. (2006). Impaired decision making following 49 h of sleep deprivation. *Journal of Sleep Research,* 15(7), 7–13

Lacouture, Y., & Cousineau, D. (2008). How to use MATLAB to fit the ex-Gaussian and other probability functions to a distribution of response times. *Tutorials in Quantitative Methods for Psychology*, 4, 35-45.

Landrigan, C.P., Rothschild, J.M., Cronin, J.W., et al. (2004). Effect of reducing interns' work hours on serious medical errors in intensive care units. *New England Journal of Medicine*, 351, 1838-1848.

Lavie, P. (1985). *Ultradian rhythms: Gates of sleep and wakefulness.* In: Schulz H, Lavie P, eds. Ultradian rhythms in physiology and behavior. Berlin Heidelberg New York, Tokyo: Springer, 148-64.

Lavie, P., & Weler, B. (1989). The timing of naps: Relationship with post-nap sleepiness. *Electrocencephalography and Clinical Neurophysiology*, 72, 218-224.

Leproult, R., Copinschi, G., Buxton, O., & Van Cauter, E., (1997). Sleep loss results in an elevation of cortisol levels the next evening. *Sleep,* 20(10), 865-70.

Lewittes, L.R. & Marshall, V.W. (1989). Fatigue and concerns about quality of care among Ontario interns and residents. *Canadian Medical Association Journal*, 140, 21-24

Lim, J., & Dinges, D.F. (2008). Sleep deprivation and vigilant attention. *Annals of the New York Academy of Sciences,* 1129, 305-22

Lin, T.D., Jovanis, P.P., & Yang, C.Z. (1994). *Time of Day Models of Motor Carrier Accident Risk,* Transportation Research Record 1457, National Academy Press, Washington, DC.

Loomis, A.L., Harvey, E.N., & Hobart, G.A. (1937). Cerebral states during sleep as studied by human brain potentials. *Journal of Experimental Psychology*, 21, 127–144

Luce, R.D. (1986). *Response times: Their role in inferring elementary mental organization*. New York: Oxford University Press.

Macchi, M.M., Boulos, Z., Ranney, T., Simmons, L. & Campbell, S.S. (2002). Effects of an afternoon nap on nighttime alertness and performance in long-haul drivers. *Accident Analysis and Prevention,* 34, 825–834.

Marcus, C.L., & Loughlin, G.M. (1996). Effect of sleep deprivation on driving safety in housestaff. *Sleep,* 19(10), 763-766.

Marmolejo-Ramos, F. et al. (2014). The activation of representative emotional verbal contexts interacts with vertical spatial axis. *Cognitive Processing,* 15(3), 253-267.

Marmolejo-Ramos, F., & Matsunaga, M. (2009). Getting the most form your curves: Exploring and reporting data using informative graphical techniques. *Tutorials in Quantitative Methods for Psychology,* 5(2), 40-50

Masino, S., & Boison, D. Eds. (2013). *Adenosine: A link between metabolism and brain activity*. Springer: Verlag, New York

Matzke, D., & Wagenmaker, E.J. (2009). Psychological interpretation of the ex-Gaussian and shifted Wald parameters: A diffusion model analysis. *Psychonomic Bulletin & Review*, 16(5), 798-817.

McCarley, R.W. (2007). Neurobiology of REM and NREM sleep. *Sleep Medicine,* 8(4), 302-30.

McCartt, A.T., Rohrbaugh, J.W., Hammer, M.C., & Fuller, S.Z. (2000). Factors associated with falling asleep at the wheel among long-distance truck drivers. *Accident Analysis and Prevention,* 32(4): 493-504.

McCue, J.D. (1985). The distress of internship. *New England Journal of Medicine*, 342, 445-452.

Mednick, S.C., et al., (2002). The restorative effect of naps on perceptual deterioration. *Nature Neuroscience,* 5, 677-681. Doi: 10.1038/nn864

Mednick, S., Nakayama, K., & Stickgold, R. (2003). Sleep-dependent learning: A nap is as good as night. *Nature Neuroscience,* 6, 697-698.

Monk, T.H., Buysse, D.J., Reynolds, C.F., et al. (1996). Circadian determinants of the post –lunch dip in performance. *Chronobiology International*, 13, 135-45.

Milner, C.E., & Cote, K.A. (2009). Benefits of napping in healthy adults: Impact of nap length, time of day, age, and experience with napping. *Journal of Sleep Research*, 18(2), 272-281.

Mitler, M.M., Carskadon, M.A., Czeisler, C.A. et al., (1988). Catastrophes, sleep and public policy: Consensus report. *Sleep,* 11(1), 100-109.

Mitler, M.M., Miller, C.J., Lipsitz, J.J., Walsh, J.K., & Wylie, D.C. (1997). The sleep of long-haul truck drivers. *New England Journal of Medicine,* 337(11), 755-761

Moss T.H., & Sills D.L. (1981). T*he Three Mile Island Nuclear Accident: Lessons and Implications.* New York: New York Academy of Sciences

National Sleep Foundation (2005). *Adult sleep habits and styles*. [Online pooll]. Retrieved from https://sleepfoundation.org/sleep-polls-data/sleep-in-america-poll/2005-adult-sleep-habits-and-styles

National Sleep Foundation, (2011). *Technology and Sleep.* [Online poll]. Retrieved from https://sleepfoundation.org/sleep-polls-data/sleep-in-america-poll/2011-technology-and-sleep

National Sleep Foundation (2008). *Sleep in America*. [Online Poll]. Retrieved from https://sleepfoundation.org/sites/default/files/2008%20POLL%20SOF.PDF

National Sleep foundation (2014). *Sleep in the Modern Family*. [Online Poll]. Retrieved from https://sleepfoundation.org/sleep-polls-data/sleep-in-america-poll/2014-sleep-and-family

Olsen, O.K., Pallesen, S., & Eid, J. (2010). The Impact of Partial Sleep Deprivation on Moral Reasoning in Military Officers. *Sleep*, *33*(8), 1086–1090.

Parris, A.B., Dienes, Z., & Hodgosn, L.T. (2013). Application of the ex-Gaussian function to the effect of the word blindness suggestion on stroop task performance suggests no word blindness. *Frontiers in Psychology,* 4, (647), 1-8.

Partinen, M. (1994). Sleep disorders and stress. *Journal of Psychosomatic Research*, 38, 89-91

Payette, M., Chatterjee, A., & Weeks, W.B. (2009). Cost and workforce implications of subjecting all physicians to aviation industry work-hour restrictions. *American Journal of Surgery*, 197, 820–5.

Pelli, D.G. (1997). *The Video Toolbox software for visual psychophysics*: Transforming numbers into movies, Spatial Vision, 10:437-442.

Pigeon, W.R., Sateia, M.J., & Ferguson, R.J. (2003) Distinguishing between excessive daytime sleepiness and fatigue: toward improved detection and treatment. *Journal of Psychosomatic Research*, 54, 61–69. doi: 10.1016/S0022-3999(02)00542-1. pmid:12505556

Priest, B, Brichard, C, Aubert, G, et al. (2001). Microsleep during a simplified maintenance of wakefulness test. A validation study of the OSLER test. *American Journal of Respiratory Critical Care Medicine*, 163, 1619–25.

Purnell, M.T., Feyer, A.M., & Herbison, G.P. (2002). The impact of a nap opportunity during the night shift on performance and alertness of 12-hr shift workers. *Journal of Sleep Research*, 11, 219-227.

Purves, D., Augustine, G.J., Fitzpatrick, D., et al., editors. Neuroscience. 2nd edition. Sunderland (MA): Sinauer Associates; (2001). *The Possible Functions of REM Sleep and Dreaming*. Available from: https://www.ncbi.nlm.nih.gov/books/NBK11121/

Ratcliff, R., & Murdock, Bennet, B., (1964). Retrieval processes in recognition memory. *Psychological Review,* 83(3), 190-214.

Rauchs, G., Desgranges, B., Foret, J., & Eustache, F. (2005). The relationships between memory and sleep stages. *Journal of Sleep Research*, 14(2), 123-40.

Richards, A. (1986). Analysis of accidents involving hazardous materials in Washington State. *Science*, 234, 125, (Research Project). Data discussed by Abelson, P.H.

Rosen, R., Mahowald, M., Chesson, A., Doghramji, K., Goldberg, R., Moline, M., Millman, R., Zammit, G., Mark, B., & Dement, W. (1998). The taskforce 2000 survey on medical education in sleep and sleep disorders. *Sleep*, 21(3), 235–238.

Saper, C.B., Lu, J., Chou, T.C., & Gooley, J. The hypothalamic integrator for circadian rhythms. *Trends in Neuroscience*, 28, 152-57

Sallinen, M., Harma, M., Akerstedt, T., Rosa, R., & Lillqvist, O. (1998). Promoting alertness with a short nap during a night shift. *Journal of Sleep Research, 7*, 240-247.

Scott, J.P., McNaughton, L.R., & Polman, R.C. (2006). Effects of sleep deprivation and exercise on cognitive, motor performance and mood. *Physiology & Behavior, 87*(2), 396-408.

Shen, J., Barbera, J., & Shapiro, C.M. (2006) Distinguishing sleepiness and fatigue: focus on definition and measurement. *Sleep Medicine Reviews, 10*, 63–76. doi: 10.1016/j.smrv.2005.05.004. pmid:16376590

Shephard, J.J., Buysse, D.J., & Chesson, J.A.L., et al. (2005). History of the development of sleep medicine in the United States. *Journal of Clinical Sleep Medicine*, 1(1), 61–82.

Smith, S.S., Kilby, S., Jorgensen, G., & Douglas, A.J. (2007). Napping and nightshift work: Effects of a short nap on psychomotor vigilance and subjective sleepiness in health workers. *Sleep and Biological Rhythms, 5*(2), 117-125.

Song, G., Huang, F., Miao, D., Li, Q., Liu, X., Wang, W. & Chen, Z. (2002). Effects of naps during sleep deprivation on symbol recognition and P300. *Chinese Mental Health Journal*, 16, 515–517.

Steinbrook, R. (2002). The debate over residents' work hours. *New England Journal of Medicine.* 347(16), 1296–1302.

Stickgold, R., & Walker, M.P. (2013). Sleep-dependent memory triage: evolving generalization through selective processing, *Nature Neuroscience,* 16 (2), 139–145

Strogatz, S.H. (1986). *The mathematical structure of the human sleep-wake cycle.* Lecture notes in mathematics, No. 69. Berlin: Springer,

Tachibana, N., Ayas T.A., & White D.P. (2005). A quantitative assessment of sleep laboratory activity in the United States. *Journal of Clinical Sleep Medicine*, 1(1), 23–26

Takahashi, M. & Arito, H. (2000). Maintenance of alertness and performance by a brief nap after lunch under prior sleep deficit. *Sleep*, 23(6), 813-9.

Tassi, P., & Muzet, A. (2000). Sleep inertia. *Sleep Medicine Reviews*, 4(4), 341-353.

Tietzel, A. J. & Lack, L. C. (2002). The recuperative value of brief and ultra-brief naps on alertness and cognitive performance. *Journal of Sleep Research,* 11, 213–218

Torsvall, L., & Akerstedt, T. (1987). Sleepiness on the job: continuously measured EEG changes in train drivers. *Electrocencephalography and Clinical Neurophysiology*, 66(6), 502–511.

United States Nuclear Regulatory Commission (USNRC): *Report on the Accident at the Chernobyl Nuclear Power Station*. Washington, DC: Government Printing Office; 1987. NU-REG 1250.

Van Cauter, E., Moreno-Reyes, R., Akseki, E., L'Hermite-Balériaux, M., Hirschfeld, U., Leproult, R., & Copinschi, G. (1998) Rapid phase advance of the 24-h melatonin profile in response to afternoon dark exposure. *American Journal of Physiology Endocrinology Metabolism*, 275, E48–E54.

Vogel, G.W. (1975). A review of REM sleep deprivation. *Archives of General Psychiatry,* 32(6), 749-61.dv

Walker, P.M. (2009). Sleep in cognition and emotion. *Annals of the New York Academy of Sciences,* 1156, 168-197.

Webb, W.B. (1995). The cost of sleep-related accidents: a reanalysis. *Sleep,* 18(4), 276-80

Williams H.L. & Lubin A. (1959). Impaired performance with acute sleep loss. *Psychological Monographs*, 73, 1–26.

Wilson, M. A., & McNaughton, B. L. (1994). Reactivation of hippocampal ensemble memories during sleep. *Science*, *265*, 676–679.