ABSTRACT

Title of Dissertation:	THE INFLUENCE OF CUMULATIVE SLEEP RESTRICTION ON HUMAN PERFORMANCE: EXAMINATION OF BRAIN DYNAMICS AND SUSTAINED ATTENTION
	Steven Kahl, Jr., Doctor of Philosophy, 2024
Dissertation directed by:	Dr. Bradley Hatfield, Kinesiology

Sustained attention (SA) impacts nearly every aspect of human performance. From the exactness of performing brain surgery to safely driving from one location to another, the ability to concentrate on a task for a period of time is important for success in work, school, relationships, and individual activities. As a key component of executive function (EF) and psychomotor performance, SA can be affected by many mental and physical processes. One process that can impact SA is restricted sleep, which is becoming more relevant in our ever-evolving technological society. Numerous studies have examined the impact of short bouts of restricted sleep on response time, a measure of SA, but few studies have examined the impact of the accumulating effect of sleep restriction (SR) on response time and brain dynamics as measured with electroencephalography (EEG). As part of a larger 40-day study, eight healthy participants (five female, average age 27.75) were observed for seven consecutive days and nights in a sleep lab, where they spent five hours in bed per night and engaged in numerous psychomotor vigilance tests (PVT), an indicator of SA, as part of their daytime activities. Through multiple

one-factor ANOVAs, response time significantly slowed, and brain dynamic changes occurred, measured by slow wave activity (SWA) maxima change in the Fz electrode, located in the midline frontal region, over the course of the entire week of continual SR compared to an extended sleep night. Employing mixed method effects revealed a statistically significant relationship between response time and SWA maxima differences. The data show that not only does response time increase the day after rising first and last SWA maxima levels converge (i.e., flattening of the line slope connecting these values) caused by short bouts of SR, but these phenomena continue this progression with prolonged SR. Over the course of the week-long SR, the final SWA maximum increased at a higher rate than the first SWA maximum, leading to the maxima difference shrinking as response time increases. These findings indicate that brain dynamics highlight less restorative sleep occurring alongside a lack of sustained attention when sleep is restricted on a consistent basis.

THE INFLUENCE OF CUMULATIVE SLEEP RESTRICTION ON HUMAN PERFORMANCE: EXAMINATION OF BRAIN DYNAMICS AND SUSTAINED ATTENTION

by

Steven Kahl, Jr.

Dissertation submitted to the Faculty of the Graduate School of the
University of Maryland, College Park, in partial fulfillment
of the requirements for the degree of
Doctor of Philosophy
2024

Advisory Committee:
Professor Bradley Hatfield, Chair
Associate Professor Rodolphe Gentili
Associate Professor Stephen McDaniel
Professor Min Qi Wang, Dean's Representative
Dr. Thomas Balkin
Dr. Allen Braun
Assistant Research Professor Hyuk Oh

Acknowledgements

This has been a long and incredibly rewarding journey. There is not enough space to adequately acknowledge and thank all the people who helped me reach this point in my academic career, but I will attempt to show my appreciation as best as I can.

I want to start by thanking my family for their never-ending love and support, especially my incredible wife Marlene. Without her many sacrifices, being able to ride my rollercoaster of emotions, providing a sounding board at many different times, and her constant love, support, and sanity, I would not have been able to reach this major milestone.

I want to acknowledge the risk my advisor, Dr. Bradley Hatfield, took by becoming my mentor and accepting me into his research and academic community. I was a middle-aged returning student with limited research experience and a spotty academic history, but he saw something in me that he was able to harness and help me become the academic and researcher I am now and will continue to become in the future. His decision to sponsor into the UMD doctoral program changed and improved my life in many ways and I cannot thank him enough for his patience, guidance, and mentorship he has provided. I hope I have left a positive impact in my role as student, instructor, and researcher.

My co-advisor, Dr. Hyuk Oh, has also provided innumerable levels of guidance throughout my doctoral program. His knowledge, experience, patience, and ability to push me further than I thought possible have been extremely valuable in helping me achieve professional levels I never dreamed of. Without him, my research capabilities and this dissertation would not be nearly as proficient as they are.

Helping me along the way have been numerous fellow researchers and doctoral colleagues. They include, but are not limited to, Dr. Kyle Jaquess, Dr. Bradley Ritland, Dr. Andrew Ginsberg, Dr. Calvin Lu, Dr. Shaun Edmonds, Kyle Pietro, and Jo Zimmerman. Each of you, and many others, have played a critical role in my ability to reach this milestone.

Table of Contents

Acknowledgements ii
Table of Contents iv
List of Tables vi
List of Figures vii
List of Illustrations viii
List of Abbreviations/Terms ix
Chapter 1: Introduction 1
1.1 Sleep Mechanics 2
1.2 Sustained Attention 8
1.3 Executive Function 10
1.4 Army Performance Triad 14
1.4.1 Physical Activity 14
1.4.2 Nutrition 17
1.4.3 Sleep 18
1.5 Slow Wave Activity as a Promising Predictor of Cognitive Performance 25
1.6 Research Gaps 31
1.7 Hypotheses 33
Chapter 2: Methods 35
2.1 Study Protocol 35
2.1.1 Sleep Schedules 35
2.1.2 Participants 38
2.1.3 First Screening Visit 38
2.2 Data Processing 40
2.3 Statistical Analysis 44
Chapter 3: Results 47
3.1 Response Time 47
3.2 SWA Measures 49
3.2.1 First SWA Maxima 49
3.2.2 Last SWA Maxima 50
3.2.3 SWA Maxima Difference 52
3.2.4 First Sleep Cycle Width 54
3.2.5 Last Sleep Cycle Width 55
3.2.6 Sleep Cycle Width Difference 57
3.2.7 First SWA Wave Energy Average 59
3.2.8 Last SWA Wave Energy Average 60
3.2.9 SWA Wave Energy Average Difference 62
3.2.10 Slope 64
3.2.11 Single Variable Summary 66
3.3 Univariate Modeling 66
3.3.1 Last SWA Maximum and RT 68
3.3.2 SWA Maximum Difference and RT 69
3.3.3 Line Slope and RT 71
3.4 Multivariate Modeling 72

Chapter 4: Discussion 73
Chapter 5: Conclusion 77
Appendices 78
Appendix 1 78
Appendix 2 79
Appendix 3 95
Appendix 4 104
Bibliography 106

List of Tables

Table 1. Descriptions of All 10 Reported Sleep Metrics
Table 2. Average Response Time
Table 3. Response Time Descriptive Data
Table 4. First SWA maxima (dB)
Table 5. SWA First Maxima Descriptive Data
Table 6. Last SWA maxima (dB)
Table 7. Last SWA Mamixa Descriptive Data
Table 8. SWA percentage difference
Table 9. SWA Maxima Difference Descriptive Data
Table 10. First Sleep Cycle Width (min)
Table 11. First Sleep Cycle Width Descriptive Data
Table 12. Last Sleep Cycle Width (min)
Table 13. Last Sleep Cycle Width Descriptive Data
Table 14. Sleep Cycle Width Difference
Table 15. Sleep Cycle Difference Descriptive Data
Table 16. First SWA Wave Energy (dB/min)
Table 17. First SWA Wave Energy Descriptive Data
Table 18. Last SWA Wave Energy (dB/min)
Table 19. Last SWA Wave Energy Descriptive Data
Table 20. SWA Wave Energy Difference
Table 21. SWA Wave Energy Difference Descriptive Data
Table 22. SWA Maxima Line Slope
Table 23. Line Slope Descriptive Data
Table 24 P-values of Single Variables Regressed with Sleep Night
Table 25. P-values of Single Variables regressed with Response Time

List of Figures

Figure 1. Buildup of Process S
Figure 2. A Model of Process S
Figure 3. Next Day Average Response Time Line Graph
Figure 4. Average First SWA Maxima Line Graph
Figure 5. Average Last SWA Maxima Line Graph
Figure 6. Average SWA Difference Line Graph
Figure 7. Average First and Last SWA Maxima Line Graph
Figure 8. Average First Sleep Cycle Width Line Graph
Figure 9. Average Last Sleep Cycle Width Line Graph
Figure 10. Average Sleep Cycle Difference Line Graph
Figure 11. Average First SWA Wave Energy Line Graph
Figure 12. Average Last SWA Wave Energy Line Graph
Figure 13. Average SWA Wave Energy Difference Line Graph
Figure 14. Average SWA Maxima Line Slope Line Graph
Figure 15. Last SWA Maximum Regression with Response Time Scatterplot
Figure 16. SWA Maximum Difference Regression with Response Time Scatterplot
Figure 17. SWA Maximum Difference Regression with Response Time Scatterplot

List of Illustrations

Illustration 1. Sample EEG signals

List of Abbreviations/Terms

ACC-Anterior cingulate cortex
ASR-Artifact subspace reconstruction
BAC-Blood alcohol content (level)
BEM-Boundary Element Model
BMI-Body Mass Index
BSL-Baseline
CDC-Centers for Disease Control and Prevention
DIPFIT-Dipole Fitting model
EEG-Electroencephalography
EF-Executive function
FIR-Finite Impulse Response
GUI-Graphical user interface
ICA-Independent component analysis
IFG-Inferior frontal gyrus
IRB-Institutional Review Board
MNI-Montreal Neurological Institute
MPFC-Medial prefrontal cortex
N1-Non-Rapid eye movement Stage 1
N2-Non-Rapid eye movement Stage 2
N3-Non-Rapid eye movement Stage 3
NREM-Non-Rapid eye movement
NSF-National Sleep Foundation
NTSB-National Transportation Safety Board
P3-Army performance triad
PSG-Polysomnography
PVT-Psychomotor vigilance task
REM-Rapid eye movement
ROTC-Reserve Officer Training Corp
RS-Recovery sleep: a period of time when the participant spends 10 hours in bed in the laboratory
SCN-Suprachiasmic nuclei
SE-Sleep extension
SR-Sleep restriction: a period of time when the participant is only allowed to spend five hours in bed per night in the laboratory
SWA-Slow wave activity
TSD-Total sleep deprivation: an extended period of time (usually beyond 24 hours) when the participant stays awake
UMD-University of Maryland
WCST-Wisconsin card sorting task
WRAIR-Walter Reed Army Institute of Research

Habitual sleep: a period of time when the participant sleeps in their natural setting (at-home bedtime, typical wake time, regular bed and environment)

Sleep condition: a period of time with the same sleep intervention (deprivation, restriction, recovery, habitual)
Sleep schedule: an entire set of 40 straight nights of sleep conducted during the study

Chapter 1: Introduction

The National Sleep Foundation (NSF) recommends adults receive 7-9 hours of sleep per night for a healthy lifestyle (Hirshkowitz et al., 2015). However, recent surveys revealed that insufficient sleep (6.8-hour average per night) has become widespread over the last few decades (Gottlieb et al., 2006). Chronic insufficient sleep has become a major concern, especially among military service members because they often engage in unpredictable duty schedules and nighttime operations, which has been associated with adverse health and safety consequences, as well as mission failure (Miller, Shattuck, & Matsangas, 2011). According to the National Training Center, hours of sleep are inversely related with increasing rank in the military (Belenky, 1997). Even more concerning, many sleep-deprived leaders cannot recognize their own levels of sleep debt or notice sleep debt in their subordinates (Miller et al., 2011). Similar to military officers, the general population also falls prey to not being aware of actual detriments from sleep loss. Previous experiments have shown that participants commonly underestimate their neurobehavioral shortcomings after sleep deprivation, using numerous different scales (Basner, Rao, Goel & Dinges, 2013). One of the most reliable methods for investigating many sleep parameters is using polysomnography (PSG) (Fabbri et al., 2021), which includes continual electroencephalographic (EEG) monitoring different sleep stages and heart rate (Rundo & Downey III, 2019). Measuring objective sleep measures through PSG and executive function task performance can help guide future research by comparing these objective measures over an extended period to highlight degrading task performance over an extended period of limited sleep.

This dissertation report provides a brief explanation of sleep: definitions, processes, and psychophysiological parameters. The next portion describes selected aspects of executive control, including sustained attention. To provide a context for the importance of sleep to military performance, the three major elements of the Army Performance Triad and its relation to this study are detailed. These sections lead to an important and relevant sleep metric that will be the crux of the ensuing results and discussion of the results, slow-wave activity and the associated methods and signal processing details. Finally, the reported results indicate that increased sleep need over a week of restricted sleep are related to decreased executive function performance. As such, the present results suggest that a continual lack of sleep can be objectively observed through both polysomnography and degraded performance, followed by discussion of the findings.

1.1 Sleep Mechanics

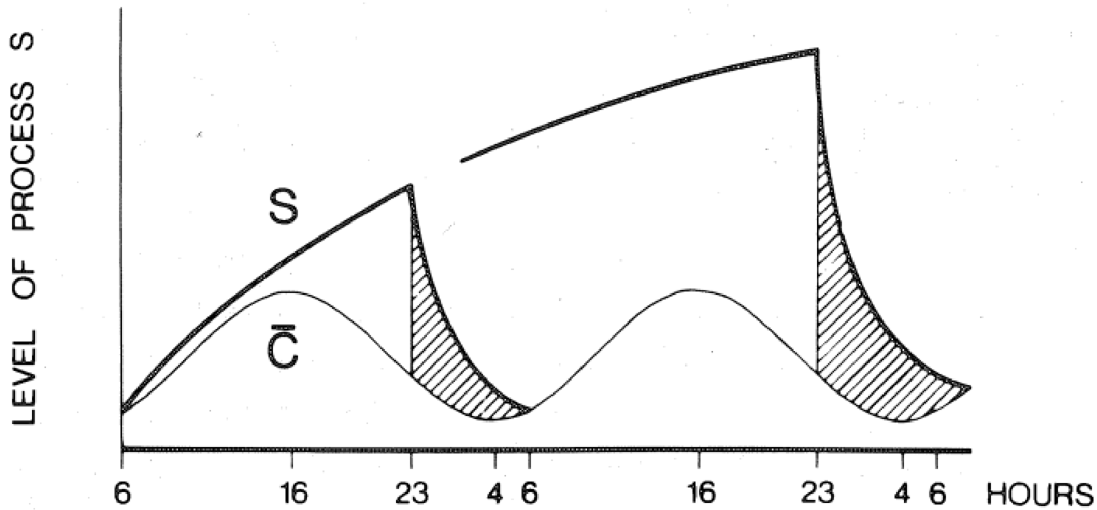
Sleep can be defined in multiple ways. Behaviorally, sleep is defined as consisting of six criteria, “(1) specific sleep site, (2) typical body posture, (3) physical quiescence, (4) elevated arousal threshold, (5) rapid state reversibility and (6) regulatory capacity” (Tobler, 1995, p. 35). For example, though humans can theoretically fall asleep anywhere, most planned sleep (1) occurs in designed areas (bed, cot, etc.), (2) in certain positions (lying down, fetal position, etc.), (3) during extended periods of inactivity, (4) that require a heightened level of outside stimuli for waking (a noise that might engage an awake person might not disturb a sleeping person), (5) to which the individual can quickly wake up (as opposed to a coma or other unconscious state) and (6) regulate some aspects, like continual restricted sleep.

The Two Process for Sleep Regulation was first proposed by Borbely (1982). Borbely (1982) labels the two processes, Process C and Process S. These two processes combine to determine sleep propensity and duration (Borbely, 1982). Process C, also known as the circadian cycle, is a diurnal cycle that is roughly the mirror image of body temperature rhythms (Borbely, 1982). Numerous studies have shown that this process is not sleep or wake-dependent but does play a role in sleep regulation (Borbely, 1982). Figure 1 plots $\overline{C}$ which is the inverse of Process C and corresponds with the 24-hour body temperature rhythm. As can be seen in the figure**,** $\overline{C}$ reaches its apex around 1600 hours and its nadir around 0400 hours.

The second part of Borbely's (1982) Two Step Process for Sleep Regulation is Process S. Process S represents sleep need in an organism and is dependent on prior wakefulness and activity (Borbely, 1982). Process S is indexed by an accumulation of slow wave activity (SWA) in the brain during time spent awake and its dissipation during sleep (Borbely, 1982). As can be seen in the left side of the figure, Process S slowly builds while awake. Once someone naturally goes to sleep (i.e., at 2300 hours in the figure), Process S starts dissipating at a slightly faster rate. When Process S closely lines up with $\overline{C}$, the organism naturally wakes up and Process S accumulation begins again.

Figure 1

Buildup of Process S



Note. Diurnal cycle (C̄) during a typical waking period starting at 0600 hours (left). Continual buildup of Process S after one night of sleep deprivation with C̄ (right) (Borbely, 1982).

The level of Process S can change depending on how long an organism is awake and what occurs during those waking hours (Borbely, 1982). The more physically and mentally demanding the waking activities are, the higher the Process S levels will be at sleep onset (Borbely, 1982). The right side of Figure 1 highlights a different scenario. If you change the sleep-wake cycle by delaying sleep beyond natural tiredness, Process S continues to accumulate. The right side of Figure 1 provides an organism staying awake for an additional 24 hours beyond the standard 2300 hour sleep time. During this additional 24 hours of time awake, Process S continues to grow significantly, but at a slightly slower rate. Once the organism starts its sleep period, the elevated Process S level starts dissipating. This dissipation occurs faster than usual, showing that you do not need to double the time asleep when time awake is doubled. Similar to a normal sleep period, Process S approaches Process $\bar{C}$, but it takes more hours of sleep for the processes to meet. In theory, if sleep is delayed beyond natural tiredness and wake time does not adjust, the two processes continually have a greater distance to each other that should cause more sleep need that does not get satiated.

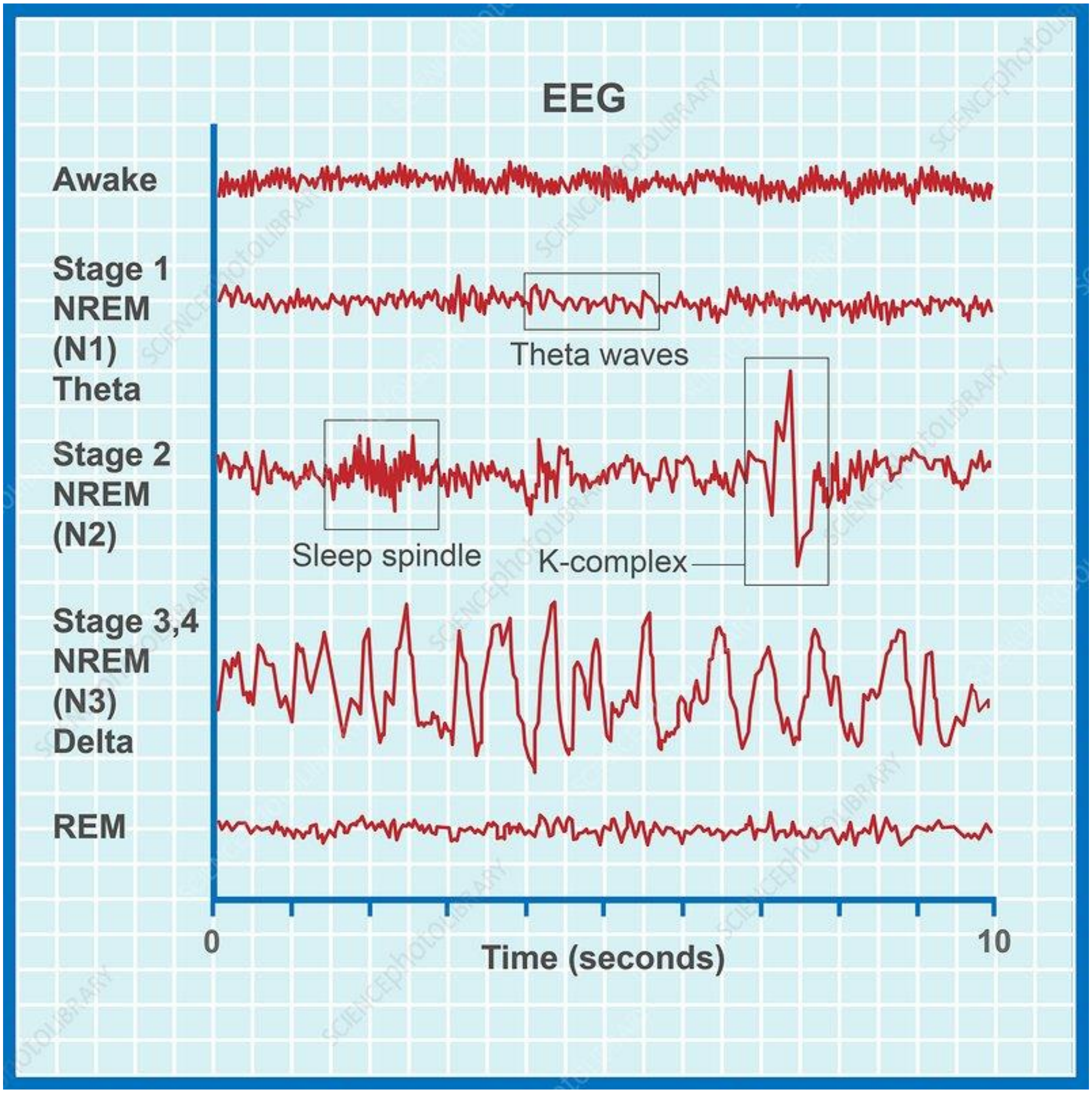
From a neurobiological perspective, Process C is initiated in the pacemaker of the hypothalamic suprachiasmatic nuclei (SCN), the master clock in the brain (Harvey, Murray, Chandler & Soehner, 2011). The SCN receives information from the photoreceptor cells of the retina (Harvey et al., 2011). Along with rods and cones, the retina contains photoresponsive ganglion cells that contain the pigment melanopsin (Harvey et al., 2011). Unlike rods and cones, these ganglion cells depolarize when activated by light (Purves et al., 2011). These ganglion cells follow a pathway through the retinohypothalamic tract terminating in the SCN (Harvey et al., 2011). After entering the retina, light is transformed into a neural signal and sent to the brain (Harvey et al., 2011). Light is also involved in the regulation of balanced secretion levels of cortisol, melatonin, and thyroid stimulating hormone. The pineal gland receives projections from the SCN, which helps it secrete melatonin, a crucial factor in sleep initiation (Harvey et al., 2011). Melatonin release is highest at night and suppression is highest during the day.

One of the ways sleep stages can be viewed is by measuring cortical signals using electroencephalography (EEG). These sleep stages consist of rapid eye movement (REM) and non-rapid eye movement (NREM). NREM sleep can be further classified into three stages, usually referred to as Stages 1, 2, and 3 (N1, N2, and N3) (Patel, Reddy & Araujo, 2021). Time spent in each of these cycles varies greatly, depending on aspects like recent sleep history, sleep hygiene (ambient temperature, background noise and light, caffeine intake, level of satiation), circadian rhythms, stress, and other factors. Sleep alternates throughout sleep stages, with N3 being the deepest of the NREM stages (Patel et al., 2021). In a healthy sleeper with a natural wake/sleep environment (no apnea, no alarm clock, proper sleep hygiene, etc.), four to five complete sleep cycles occur per night (Patel et al., 2021). Each cycle, lasting between 90-110 minutes, fluctuates from N1 through N3, eventually reaching REM sleep (Patel et al., 2021). As the night goes on, REM sleep periods lengthen and NREM sleep periods shorten (Patel et al., 2021).

REM and NREM sleep can be distinguished by different EEG profiles and body cues. Sleep begins with N1, which occurs when the EEG profile changes from waking alpha waves to mostly theta waves (Patel et al., 2021). N1 lasts for a few minutes and noticeable changes include going in and out of wakefulness and delayed or non-existent vocal communication. N2 is a deeper sleep state and covers nearly half of all non-REM sleep time. N2 occurs when the EEG profile starts showing regular sleep spindles and K-complexes (Patel et al., 2021). Sleep spindles are sharp, distinct wave bursts in the alpha range surrounded by minimal wave activity (Patel et al., 2021). K-complexes are definitive negative waves followed immediately by a positive aspect that is distinctively different than the surrounding waves (Patel et al., 2021). Physical changes in N2 include teeth grinding and reduced breathing and heart rate (Patel et al., 2021). N3 is the deepest sleep stage and is noticeable through slow delta waves, also known as slow wave activity (SWA) that have high amplitudes and little-to-no eye movement (Patel et al., 2021). As the deepest sleep state, arousal is much more challenging and waking during this stage tends to lead to compromised mental and physical performances for up to an hour (Patel et al., 2021). N3 is also the stage during which night terrors and sleepwalking are most likely to be observed, if present (Patel et al., 2021). REM sleep is the most restless stage and is generally when dreaming occurs (Patel et al., 2021). The REM EEG profile looks similar to the waking profile, but there is little-to-no muscle movement, except erratic breathing, occasional twitches and eye movements (Peever & Fuller, 2017). This similar profile includes lower range alpha signals and theta waves, without the occasional spindle or K-complex (Patel et al., 2021). Anatomically, the dorsolateral prefrontal cortex and areas in the brainstem like the raphe nuclei and the locus coeruleus are inactive during REM sleep (Purves et al., 2011). These same regions are active during waking hours, which partially explains how the brain is active during REM sleep without active movement (Purves et al., 2011). Illustration 1 provides a graphical representation of typical EEG waves during different sleep stages (https://www.sciencephoto.com/media/1158883/view/eeg-sleep-stages-illustration).

Illustration 1

Sample EEG Signals



<u>*1.2 Sustained Attention*</u>

The impact of sleep deprivation on the brain activity and the ensuing relationship to mental performance in the waking state is the focus of the present study. An important element of mental performance is that of sustained attention (SA), which is an executive function process involving task-relevant brain regions and inhibition of task-irrelevant brain regions when completing a given task (Stuss, Shallice, Alexander & Picton, 1995). Though sometimes used interchangeably with the term vigilance, SA is more likely a key component of vigilance (Sarter, Givens & Bruno, 2001; van Schie, Lammers, Fronczek, Middelkoop & van Dijk, 2021). Vigilance is defined as awareness of and preparation to respond to many potential outside stimuli and unpredictable changes (van Schie et al., 2021). As such, SA involves focusing on a given task while trying to minimize unimportant or non-essential stimuli (i.e., distractions) that reduce the quality of human performance (van Schie et al., 2021). In practical terms, SA could be exemplified as the neurocognitive process of a surgeon performing an operation and vigilance could be exemplified by driving in a residential neighborhood. The surgeon is sustaining their attention on successfully completing the procedure while focusing on realistically possible complications until that surgery is complete. The driver in a residential neighborhood is trying to keep their car in the proper lane at a safe speed while staying alert for other drivers who might be unaware of pedestrians or running animals, sounds of passengers or entertainment, and many other unknown possibilities. Testing of these processes is also conducted differently.

Many times, SA is measured through a response time test, like the psychomotor vigilance task (PVT), which helps to highlight the confusing use of the terms. The PVT involves a participant responding to a given stimulus as quickly as possible for a given period of time (usually five or 10 minutes). One of the challenges of the PVT is sustaining attention to the given stimulus throughout the testing period while also minimizing distractions (Stuss et al., 1995). Slower response time on the PVT could be an indication of weaker sustained attention. If the participant is distracted, response to a given stimulus can also be delayed, which would be measured by slower average response times during those distracted periods. Vigilance, itself, is more likely to be tested using such tasks as driving simulations within which the participant must pay attention to many different situational possibilities (Roca, Castro, Lopez-Ramon & Lupianez, 2011). SA is one aspect of a larger mental skillset known as executive functioning.

1.3 Executive Function

Along with sustained attention, sometimes referred to as cognitive inhibition, other processes of executive function (EF) include working memory and cognitive flexibility (Diamond, 2013). EF comprises a combination of different brain processes that are more advanced than simple responses and intuitive behavior (Diamond, 2013). Each of these EF categories involves top-down processes and is more complex than those of automatic responses to a stimulus or other such ‘mindless’ actions (Diamond, 2013). More advanced levels of EF, like planning and decision-making, derive from these three categories (Collins & Koechlin, 2012; Miyake et al., 2000). Consequently, EFs are critical for an individual’s adaptive social engagement, cognitive, emotional, and physical health (Diamond, 2013; van Schooten et al., 2019).

Adequate working memory involves the ability to maintain items in short-term memory while simultaneously performing more complicated mental processes and is crucial for reasoning, comprehension, and learning (Baddeley, 2010). Limited working memory usage can include delayed or incorrect information retrieval, forgetting prior stimuli, getting easily flustered and/or incorrect resolutions of simple problems. One example of using working memory is solving a multi-level arithmetic problem.

Working memory is commonly tested through the n-back series of tasks (Owen, McMillan, Laird & Bullmore, 2005), which include 1-back, 2-back, and 3-back tasks. The key portion of these tasks is the capacity to prioritize which stimulus is paramount while keeping previous images readily available. In a standard 2-back task, a button is pressed when the current stimulus matches the item presented two images prior. For example, in the sequence red, blue, green, blue, the button is pressed at the second blue because it matches the color that was given two presentations before. Successful performance requires keeping a running tally of information readily available for recall. Poor performances during n-back tasks include lapses when the button is not pressed in a reasonable time or pressed at the wrong time, as well as slower response times to the correct stimulus.

Neuroimaging techniques indicate the important role frontal and prefrontal regions play when performing n-back tasks, but the exact portion is not universally agreed upon (Owen et al., 2005). Patients with frontal lobe lesions tend to struggle while performing such n-back tasks, but they also struggle with tasks that do not involve working memory, like word fluency, indicating the struggle with defining the exact mechanisms for proper working memory (Mueller & Knight, 2006). Some areas that are consistently activated during n-back tasks include the bilateral rostral prefrontal cortex and the frontal operculum (Owen et al., 2005).

Cognitive flexibility is the capacity to change a course of action or thought process based on new information that may conflict with existing knowledge or rules and is manifest after first inhibition and a new course of action or mindset has started (Diamond, 2013). Proper cognitive adaptability involves being able to accept new information, critically analyze this new information, determine if this new information will help one make a new and improved course of action and implement any changes needed for a preferable result. Difficulties with cognitive flexibility include reaching cognitive overload during simple tasks, incorrectly analyzing conflicting information and/or needing to start over to change tasks or adjust a course of action.

The Wisconsin Card Sorting Test (WCST) is a popular task used to test cognitive flexibility (Miles et al., 2021). The WCST involves a deck of 60 cards with three types of stimuli on them: color (red, blue, yellow, or green), shapes (circles, triangles, stars, or crosses), and number (one-four) (Miles et al., 2021). The participant is given the deck of cards and asked to sort them based on one of the stimuli patterns, without prior knowledge of which pattern needs to be matched. The participant is informed if each match is correct or incorrect by the researcher until the pattern is deduced. Successful completion of this task requires cognitive flexibility to quickly switch pattern recognition after the previous pattern is complete. Scoring the WCST include measuring errors and latency (Eling, Derckx & Maes, 2008).

Brain regions commonly used during cognitive flexibility or task switching include the anterior cingulate cortex (ACC) and the inferior frontal gyrus (IFG) (Giller & Beste, 2019). Associations between ACC and IFG process changes and degraded cognitive flexibility performance have been tested with mental fatigue (Yu, Muckschel & Beste, 2021), aging (Giller & Beste, 2019), and Autistic populations (Sanders, Johnson, Garavan, Gill & Gallagher, 2008).

Although a number of EF performance tests can be utilized to examine the effects of sleep deprivation, the focus of the present study is on the concept of SA, which is relatively neglected in the scientific research in this area, even though it is critical to successfully completing everyday tasks (Esterman & Rothlein, 2019). A key aspect of successful SA is that of inhibition. Inhibition is the act of stopping impulses, intrusive thoughts, and unwanted emotions (Baumeister, 2014). It is estimated that nearly 90% of self-control can be attributed to stopping responses (Baumeister, 2014). To properly sustain attention on a given task, like responding only when a certain specific stimulus appears, inhibition of other thoughts, impulses, etc. need to be actively or passively achieved. Individuals with lower inhibition skills tend to have poor impulse control and tend to make poor decisions because they respond based on first response and not thinking through best practices (Diamond, 2013).

Inhibition leads to the concept of vigilance. Vigilance is the continual inhibition of a habitual response (Lara, Madrid & Correa, 2014). A simple example of vigilance is that of responding selectively when a specific stimulus is presented (Lara et al., 2014). A complex example of vigilance is that of a Soldier entering an unknown territory and in need of a high-speed response based on presentation of an ally or an enemy (i.e., friend or foe).

1.4 Army Performance Triad

One area of relevance that connects the present study to the importance of sleep is the recently created Army Performance Triad (P3) initiative from the Office of the Army Surgeon General. The three pillars of this triad to facilitate Soldier performance and leadership are physical activity, nutrition, and sleep (United States Army, n.d.). The purpose of the P3 is to optimize Soldiers' health through this holistic approach (United States Army, n.d.). Goals of the P3 include variable exercise intensities, incorporating proper exercise recovery days, eating at least eight servings of fruits and vegetables, drinking plenty of water, and attaining at least seven hours of sleep per 24-hour cycle (United States Army, 2021). Each pillar is important independently, but their connection to executive function and mental performance are part of the reason why they are included in optimal soldier health. The three elements are discussed below in the order of physical activity followed by nutrition leading to a final section on sleep, which is the focal point of the present study.

1.4.1 Physical Activity

Physical activity is known to be beneficial for many reasons. Though often studied as a way to prevent diseases like dementia, physical activity can also improve executive functioning in neurotypical individuals. An active group (based on the International Physical Activity Questionnaire) of young adults were much more efficient performing a rapidly changing task-switching paradigm compared to a sedentary control group (Kamijo & Takeda, 2010). This was examined through faster response times and fewer errors (Kamijo & Takeda, 2010). The authors propose that physically active young adults demonstrate more efficient executive function flexibility compared to their less active counterparts (Kamijo & Takeda, 2010). Efficient executive functioning is especially important for soldiers in constantly changing environments and continually maintaining their physical fitness may be better in these environments.

This relationship is also significant in older adults. Looking at a group of over 500 Boston-area community-dwelling adults averaging nearly 80 years of age, physical activity levels were correlated with different executive function task performances (Eggermont, Milberg, Lipsitz, Scherder & Leveille, 2009). The more physically active (using the Physically Active Scale for the Elderly) a participant was, the more likely they were to have higher executive function task performance improvements across all tasks (Eggermont et al., 2009). These significant results were still present after many other physical and mental measures were accounted for (Eggermont et al., 2009). As Soldiers age, both inside and outside the military community, proper physical activity can help maintain mental health.

Not only does current physical activity tend to indicate better executive function task performance, numerous studies noticed a link between physical activity interventions and improved executive function performance. Barenberg, Berse, and Dutke (2011) completed a meta-analysis of 23 studies focusing on short and long-term interventions that may have impacted executive function performance. In all nine long-term (10 or more weeks of different physical activities) interventions, executive function performance significantly improved throughout the physical activity intervention (Barenberg et al., 2011). This occurred in populations ranging from children to older adults, healthy to overweight and cognitively healthy to cognitively compromised (Barenberg et al., 2011). These improvements also occurred using different tasks and with different types and intensities of activity (Barenberg et al., 2011). Many of the studies employed random intervention assignments and some employed control groups.

One study that employed a control group and random assignment was performed by Davis et al., (2007) on overweight children. This group randomly placed children in either a 20 minute per day low-dose, a 40 minute per day high-dose, or non-exercise control group for 15 weeks and completed multiple child-appropriate cognitive tests before and after the potential interventions (Davis et al., 2007). The exercise groups were the same, except for duration (Davis et al., 2007). All children improved, but the high-dose group improved more than the low-dose group, which improved more than the non-exercise control group (Davis et al., 2007). These results indicate that exercising five times a week for four months significantly improves cognitive ability and doubling that time from 20 to 40 minutes can provide even more improvement (Davis et al., 2007).

The 14 short-term intervention studies mostly showed significant improvement, but not all of them (Barenberg et al., 2011). The authors hypothesize that the differing executive function tasks may have been a contributing factor leading to inconsistent results, but those tasks did show significantly significance effects when physical activity lasted longer than 10 weeks (Barenberg et al., 2011). Most of the 14 studies used either one or more exercise sessions and included a non-exercise control session (Barenberg et al., 2011).

One such study was performed by Pontifex, Hillman, Fernhall, Thompson, and Valentini (2009). This group counter-balanced 21 healthy adults through a three-day rotation of 30 minutes of resistance, cardio, or rest (Pontifex et al., 2009). All response times improved 30 minutes after each daily session, but cardio exercise improved more than resistance exercise, which improved more than rest (Pontifex et al., 2009). It appears that in the short-term, physical activity can benefit cognitive performance, but longer lasting exercise plans are much more likely to provide significant benefits.

Not only can starting a physical activity regimen at a young age improve executive function and cognition, starting a regimen at an advanced age can still improve cognition (Hillman, Erickson & Kramer, 2008). An example of this can be seen in a seminal study by Kramer and others (1999) comparing executive control process in a group improving aerobic fitness to a group stretching and muscle toning. The authors described the aerobic performance-impacted tests as task switching, response compatibility, and stopping (Kramer et al., 1999). After cardiorespiratory training in an adult population, multiple executive functions show significant improvements that were not found in non-cardiorespiratory training (Kramer et al., 1999). In this and many other populations, these changes can be noticed through different working EF performance measures and the anatomy of frontal brain regions, like the ACC and middle frontal gyrus (Hillman et al., 2008).

1.4.2 Nutrition

Along with physical activity, nutrition and different dietary profiles can also affect executive function performance. In a cross-sectional study of over 1,000 middle-aged Europeans, increased cholesterol and fatty acid intake were significantly associated with lower cognitive performance in numerous executive function tasks, independent of many other factors (Kalmijn et al., 2004). In comparison, increased fatty fish consumption (Omega-3) was significantly associated with decreased executive function impairment (Kalmijin et al., 2004). These associations were reached through self-reported food diaries and a lab-conducted battery of executive function tasks covering memory, inhibition, and cognitive flexibility (Kalmijin et al., 2004). Decreased performance on these types of tasks can be an indicator of the early stages of dementia (Fabrigoule et al., 1998).

Similar results can be found in short longitudinal studies. A study of 20 sedentary adult men with average BMIs were fed a mostly carbohydrate diet (61%) followed by a fat-heavy diet (74%) for multiple days each and performed numerous executive function tasks throughout (Edwards et al., 2011). Performance was worse in both simple response time and power of attention after the fat-heavy diet compared to the mostly carbohydrate diet (Edwards et al., 2011). A properly balanced diet should contain 55% carbohydrates (Lim, 2018), so not only can deviations affect physical health outcomes, but the literature suggests these deviations can also hinder cognitive performance.

1.4.3 Sleep

The pillar of this Triad that will be examined in this study is sleep. Sleep habits amongst military personnel tend to be problematic and inadequate sleep is a common occurrence in the Army (Lentino, Purvis, Murphy & Deuster, 2013). Some of the factors affecting sleep in the Army include shifting work hours, constant loud noises, and the uncertainty surrounding dangerous work environments (Lentino et al., 2013). Along with many biological processes, sleep loss can lead to impaired physical fitness and performance on many military-specific activities, such as marksmanship (Macera, Aralis, Rauh & MacGregor, 2013). It is also not uncommon for there to be a culture of denying the need for sleep or even being proud of performing after compromised sleep (Miller et al., 2011). This can also lead military officers to lie about their sleep debt or its effects (Miller et al., 2011). Objective measures, beyond extensive sleep lab studies can help determine when someone is suffering from compromised sleep and its effects.

In a commonly cited sleep and performance study (700 citations as of January 2020, according to Park, Kim, and Park, 2020), a typical night of sleep drastically improved performance on a motor skill learning task compared to a similar time window of waking (Walker, Brakefield, Morgan, Hobson & Stickgold, 2002). Different groups underwent training in the morning or evening, but all evidence pointed to significant retesting improvements when the ten-hour time gap included a typical night of sleep as opposed to retested later the same day (Walker, et al., 2002). A study of nearly 1,000 elite European athletes revealed that proper sleep is one of the most effective recovery strategies (along with fluid replacement), regardless of gender, sport, or participation level (Venter, 2014). As a recovery tool, sleep was one of the few methods that was universally successful and readily available (Venter, 2011). With so many demands of a typical Soldier (referred to be some as tactical athletes (Taylor, Hernández, Sessoms, Kawamura, & Fraser, 2020), proper sleep can help health recovery and maintenance with scant other resources.

Performance differences can be seen when both restricting and extending sleep. A recent study examined general physical fitness and different sleep measures in sedentary adults. The authors considered sedentary to be healthy adults with consistent BMIs between 18-35 and fewer than 20 minutes of vigorous activity occurring less than three days a week (Mochón-Benguigui, Carneiro-Barrera, Castillo & Amaro-Gahete, 2021). In general, the more time spent sleeping at night, the more active the participant is during the day, though they spent less time awake (Mochón-Benguigui et al., 2021). Participants also showed significantly higher VO_2max values and muscular strength with progressively more total sleep time and better sleep efficiency (Mochón-Benguigui et al., 2021). As this was not an exercise intervention study, participants were studied under their current, sedentary conditions and the authors included factors such as age, fat mass percentage and fat lean mass indices into their statistical analyses (Mochón-Benguigui, et al., 2021). This makes it more likely that increased sleep time and quality improve physical fitness, especially in what is now becoming a less active society.

Intentionally extending sleep can improve a combination of both physical and mental performance. For example, Ritland et al., (2019) tested 50 Reserve Officer Training Corp (ROTC) college students after they increased their time in bed by nearly 90 minutes for almost a week. By extending time in bed over multiple nights, performance improvements were significantly improved in response time, a trail making task (going from A to 1 to B to 2 on a piece of paper as quickly and accurately as possible) and standing broad jump distance compared to their habitual time in bed (Ritland et al., 2019). The control group, maintaining their habitual time in bed throughout the study, did not show significant improvements in performance, indicating that the extra time in bed may lead to improved performance in both cognitive and physical performance (Ritland et al., 2019).

These types of performance improvements have also been seen in more highly trained athletes, for whom performance improvements do not come as easily. Two seminal studies reported sport-specific improvements in varsity-level college athletes. Schwartz and Simon (2015) tested 12 Division III (non-scholarship) college tennis players after increasing their time in bed from seven to nine hours per night for a week. After this week of extended sleep, their serving accuracy percentage increased by over 15% (Schwartz & Simon, 2015). Mah, Mah, Kezirian & Dement (2011) tested 11 Division I (top college division) men's basketball players from Stanford University. After extending their time in bed to 10 hours per night for over a month, not only did response time and sprint time improve (similar to Ritland et al., 2019), but free-throw shooting and three-point shooting improved by about 9% each during practice sessions (Mah et al., 2011). These studies showed relevant sport-specific improvements shortly after sleep extension.

This effect can also occur when banking sleep by spending extra time in bed before participating in a sleep restriction intervention. Yamazaki, Antler, Lasek and Goel (2021) found significant response time performance decreases in a group of nearly 50 adults after spending four hours in bed per night. These four-hour in bed nights occurred just after they spent 10-12 hours in bed for two nights each (Yamazaki et al., 2021). The effects were even more pronounced in the group of nearly 50 adults after 36 hours of total sleep deprivation (similar to activity performed the night after staying up all night) (Yamazaki et al., 2021). Increased recovery nights of 12 hours in bed per night improved response time performance, but still did not bring performance to pre-intervention levels (Yamazaki et al., 2021). Not only can reduced sleep impair performance, even after banking sleep beforehand, but trying to catch up by increasing future sleep may not make up for those performance impairments.

Studies have revealed that sleep deprivation can significantly impact performance in other careers, including surgeons. Some studies have shown that restricted sleep tends to negatively impact cognitive performance more than motor performance (Whelehan, Alexander & Ridgway, 2020). In Whelehan's (2020) review, about 40% of over 30 available studies showed sleep deprivation negatively impacted performance in a simulated surgical setting and 30% showed sleep deprivation negatively impacted performance in actual surgical settings. This can manifest itself in increased medical errors and poorer decision making (Whelehan et al., 2020). These errors occurred in a population trained to perform in these adverse conditions. Sleep deprivation can also increase the risk of motor mistakes made during surgery, accounting for nearly 40% of the contributing factors reported (Ayas et al., 2006). These mistakes include making the right decision in a timely fashion but making an error in execution of the proper task.

These sleep-related issues can also manifest themselves in transportation. For example, accidents caused by fatigued airplane pilots were 4-8% more likely to occur based on official statistics from the National Transportation Safety Board (NTSB) (Caldwell, 2005). The Air Force Safety Center attributes 7.8% of Class A mishaps, at least partially, to fatigue (Caldwell, 2005). At night, that percentage increases nearly four-fold to 25% (Caldwell, 2005). A Class A mishap is defined as an error that results in a loss of life or over $2 million in damage (Poisson & Miller, 2014). In the Navy, that 7.8% increases to 12.2% (Caldwell, 2005). These numbers cover data from the 1970s to the 1990s.

Bringing these sleep-related issues to a civilian population provides that about 20% of all traffic accidents have been attributed to drivers falling asleep at the wheel (Horne & Reyner, 1995). In a vehicle merging task, total braking time was hindered by 25% after one night of four-hour sleep and 28% after two nights of four-hour sleep (Mahajan & Velaga, 2020). According to the Centers for Disease Control and Prevention (CDC) (2011), multiple states report that more than 6% of respondents report falling asleep while driving at least once during the previous month. Multiple studies have correlated performing after 24 hours of wakefulness to having a Blood Alcohol Level (BAC) of 0.10, which is at or above the legal limit for driving in nearly every country (Dawson & Reid, 1997; Lamond & Dawson, 1999; Williamson, Feyer, Mattick, Friswell & Finlay-Brown, 2001). Just 17 hours of wakefulness (driving at midnight after waking up at 0700 hours) has been equated to a BAC of 0.05 (Williamson & Feyer, 2000). A person with a 0.05 BAC, equivalent to four drinks in two hours for a 170-pound man or three drinks for a 137-pound female, has been shown to exhibit significant impairments in many studies (Fell & Voas, 2014). This type of performance comparison can be done with different sleep structures. Using multiple neurobehavioral measures, performance after 14 nights of sleep restriction (four hours per night) mirrored that of three nights of complete sleep deprivation

(Van Dongen, Maislin, Mullington & Dinges, 2003). According to the CDC (2011), about 5% of respondents report getting four hours or less of sleep on a consistent basis. Based on these results, it is possible that about 1 out of every 20 people are regularly functioning as if they are legally drunk.

In a study of 69 adults, participants restricting their sleep to either three, five or seven hours per night over seven nights saw decreased performance compared to the nine-hour per night group (Belenky et al., 2003). This performance decrease (both longer response times and increased lapses) was inversely related to the number of hours of sleep per night (Belenky et al., 2003). After three nights of eight hours of recovery sleep per night, performance improved, but not to the nine-hour per night group (Belenky et al., 2003). The more sleep was restricted, the worse the performance and in some cases, performance continued to decline the longer the restricted sleep intervention occurred.

Sleep restriction can impact many different aspects of everyday life, including mental and physical health. Haack and Mullington (2005) examined a group of nearly 50 people, about half slept for four hours a night for nearly two weeks, while the others continued their habitual sleep of eight hours per night. Along with the expected increase in tiredness and fatigue in the sleep deprived group, optimism drastically decreased and there were significant increases in anger and body discomfort in the deprived group compared to the control group (Haack & Mullington, 2005). To show that compromised sleep was potentially a factor in these results, all reported changes returned to baseline levels after the deprived sleep group engaged in a recovery night of eight hours of sleep (Haack & Mullington, 2005). In addition to body discomfort, the sleep restricted group indicated significantly higher levels of nausea and backaches (Haack & Mullington, 2005). Headaches, joint and muscle pain also increased in the sleep deprived group, but not quite to the level of statistical significance (Haack & Mullington, 2005).

1.5 Slow Wave Activity as a Promising Predictor of Cognitive Performance

A promising biomarker that captures a critical element of sleep in the restorative stage 3 of non-REM sleep is that of slow wave activity (SWA), which represents cortical neuron activity characterized by slow oscillations in membrane potentials (Huber, Felice Ghilardi, Massimini & Tononi, 2004). These slow oscillations generally manifest in EEG waves measuring from roughly 0.5 to 4 Hz. Synaptic strengthening during wakefulness can create a local SWA increase when measured during sleep (Maatta et al., 2010). By depressing synaptic activity through different manipulations, specific brain regions have shown a local SWA decrease (Maatta et al., 2010).

Since SWA increases with a higher sleep need and decreases with lower sleep needs, it is generally seen as an appropriate marker for sleep homeostasis (Greene, Bjorness & Suzuki, 2017). The higher the amplitude of SWA in a sleeping person, the more sleep that person needs.

SWA tends to follow consistent behavior during both sleep and wake time. As shown in Process S, SWA slowly builds during wakefulness and the intensity of this buildup is based on prior waking activity (Borbely, 1982). The more physically and mentally challenging waking hours are, the more sleep needed that night to wake up feeling rested. After an initial increase in measured SWA after falling asleep, SWA slowly dissipates during the second half of non-REM sleep (Maatta et al., 2010). When SWA activity levels approach zero, non-REM sleep transitions into REM sleep (Maatta et al., 2010). During REM sleep, SWA starts increasing again (Maatta et al., 2010). When these new SWA values approach sleep Process S levels, REM sleep transitions back to non-REM stages (Borbely, 1982). These new SWA levels that trigger the next set of REM sleep stages are not as high as the first waking SWA levels (Borbely, 1982). Without an outside stimulus waking up an individual (alarm clock, loud noise, etc.), this cycle of increasing and decreasing SWA levels repeats until Process S approaches zero (Borbely, 1982). In theory, an individual is properly rested when Process S nears zero and that individual will naturally wake up rested and ready for the upcoming day (Borbely, 1982). Such a state would be expected to facilitate proper executive psychomotor function.

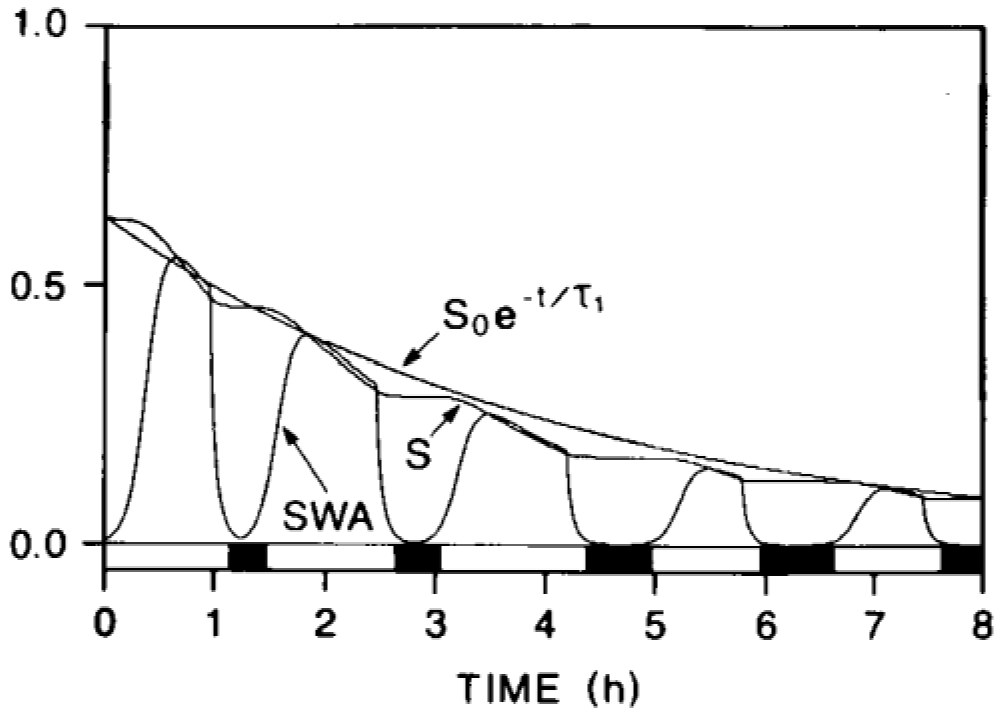
The work of Maatta et al. (2010) reveal the impact that outside variables can exert on localized SWA. Maatta et al. (2010) had participants perform a visuomotor task at different times of the day to assess their effect on that night's SWA levels. Regardless of time of day, performing this visuomotor task showed higher first SWA levels compared to the night prior when no task was performed. By performing the task in the evening compared to the morning, SWA in the right parietal region (the region activated the most during the task itself) was even higher at sleep onset (Maatta et al., 2010). Though it is impossible to match physical and mental effort from day to day, giving participants a challenging task increased that night's SWA levels in the impacted region.

Localized SWA can be seen without manipulating the prior day's workload. Cajohen, Foy and Dijk (1999) measured SWA through numerous EEG electrodes during a baseline night of sleep and a night of recovery sleep after 40 hours of sleep deprivation. Both nights were roughly eight-hour sleep episodes (Cajohen et al., 1999). The authors measured SWA through the four midline sites (Fz, Cz, Pz, Oz) and compared them to the averaged linked mastoids (A1, A2; one on each side of the head) (Cajohen et al., 1999). In both cases, SWA values were highest in the frontal region and slowly decreased dorsally through the brain and these ratios continued throughout the entire sleep period (Cajohen et al., 1999). There was a significant SWA increase after sleep deprivation compared to baseline in all but the occipital region in the first quarter of the sleep episodes (Cajohen et al., 1999). Both the frontal and central regions revealed significant increases in the first half of the eight-hour episodes (Cajohen et al., 1999). Cajohen et al. (1999) showed that throughout the night, SWA is higher in the frontal region and sleep deprivation might have more of an effect on processes involving the frontal lobe. This increased SWA activity after sleep deprivation is most pronounced after sleep onset and is still present after five or six hours of sleep, though not to a significant level during one sleep episode.

Achermann and Borbely (1990) updated Borbely's 1982 model that helps illustrate the relationship between SWA and Process S (along with an exponentially declining curve) during a typical night of sleep (see Figure 2). This figure represents a standard eight-hour period of sleep that cycles through REM and non-REM sleep. On the x-axis, REM sleep is represented by black bars and the three non-REM sleep stages are represented by the longer white bars. In the previous model (Borbely, 1982), SWA was quantified as the mean value per cycle. In this updated model, SWA is the integrated value of SWA as it oscillates up and down as Process S declines. As the duration of a sleep period increases, the need for sleep decreases. This can be seen with the declining Process S curve.

Figure 2

A Model of Process S



Note. Slow wave activity (SWA) levels during a typical eight-hour sleep period (Achermann & Borbely, 1990).

This updated model provides SWA as rising and falling throughout the night, as opposed to the roughly linear dissipation shown in the 1982 model (Achermann & Borbely, 1990). The rise and fall of SWA occurs during non-REM sleep, but each subsequent maxima does not reach previous levels. As a sleep period continues, the organism is not as tired at sleep onset, so SWA levels do not need to climb as high.

Since that organism is not completely rested after one non-REM sleep cycle, SWA rises sharply until reaching the current urge to sleep (Process S). It seems that an organism deems itself rested and ready for the next waking period when Process S is close to the brain's dissipated SWA levels. The empirical data that helps inform this updated model indicates SWA maxima slowly decreases over successive non-REM sleep episodes and minimal SWA changes during REM sleep (Achermann & Borbely, 1990). Importantly, additional empirical data show higher first SWA levels after prior sleep deprivation (Achermann & Borbely, 1990).

Not only can one see changes in sleep manifest itself in EEG profiles, but one can also see sleep changes manifested in the brain through EF processes. These EF processes can work together in the frontal cortex. For example, one of the benefits of proper sleep is being able to properly differentiate between conflicting possibilities (Wilckens, Erickson & Wheeler, 2012). This proper differentiation of multiple options allows for a higher likelihood of making the optimal choice, while inhibiting improper choices. In many cases, this cognitive adaptability can be enhanced with better working memory skills. The more possibilities that you can consider, the more likely you are to find and choose the ideal solution. Another important aspect of memory retrieval is proper timing (Wilckens et al., 2012). As important as it is to choose the ideal option, being able to make that decision in a timely manner is also critically important.

Other brain regions are important in EF processes. Many working memory items come from long-term memory, which are stored in the two hippocampi, which connect to the ACC (Stickgold, 2005). One of the known benefits of sleep is long-term memory consolidation, which mostly occurs in the hippocampi (Stickgold, 2005). During proper sleep, items remembered throughout the day connect to other memories and important items that allow for neurocognitive pruning that create space for future storage (Stickgold, 2005).

Inhibition is generally processed in the amygdala (Anderson & Platten, 2011). Proper sleep allows for proper connectivity between the amygdala and the medial prefrontal cortex (MPFC) (Anderson & Platten, 2011). This proper connectivity helps prevent improper emotional responses that can derive from incoming stimuli (Anderson & Platten, 2011; Drummond, Paulus & Tapert, 2006).

EEG SWA power has been correlated with some EF task performances. For example, Diep and colleagues (2020) used an automated acoustic stimulation device to increase SWA (0.5-4 Hz) in middle-aged men during habitual sleep. This full-night increase over the full 10-20 standard EEG montage was correlated with individual increases in task performance in verbal fluency and working memory (Diep et al., 2020). The tasks that showed significant improvements in performance with a SWA increase were task switching between word prompts and word categories and the 2-letter n-back tests (Diep et al., 2020). These improvements occurred without a significant increase in non-REM time, when most SWA occurs (Diep et al., 2020). With performance improving after SWA power increases without a noticeable increase in time spent during those stages, it makes it more likely that changes in lower range power individually match certain EF performances.

A large study of over 200 adults also revealed some commonalities between increased SWA and improved task performance the next day (della Monica, Johnsen, Atzori, Groeger & Dijk, 2018), but the results are mixed. After controlling for sex and age, increased SWA was significantly correlated with better performances on working memory tasks like verbal 1 and 2-back n-back (della Monica et al., 2018). As the authors state, there were a large number of comparisons and correlations (della Monica et al., 2018), so they performed numerous false discovery rate analyses to avoid having too many false positives, while still discovering proper significances. The strong significance between SWA and task performance did not survive this further analysis at the $p<0.10$ level (della Monica et al., 2018). This could indicate that the first significance is indeed a false positive or the drastic age differences (20-84) between participants (young adults and the elderly have very different sleep needs and profiles) or one of many other potential confounding variables explains the significance disconnect.

1.6 Research Gaps

Performance changes through response time, mostly through tests like the PVT, are considered by many the gold standard for its sensitivity in capturing objective changes due to impacted sleep (Chaisilprungraung et al., 2022). More specifically, it is well established that response time continues to significantly increase the more sleep is restricted to the individual. Measuring SWA through EEG (i.e., 1-4 Hz) has been the most consistent marker for determining an individual's sleep need and sleep debt (Borbely, 1982). Higher SWA values are associated with longer time being awake, leading to increased sleep debt (Wilckens, Ferrarelli, Walker, & Buysse, 2018). As shown previously in Figure 1, SWA oscillates during sleep, progressively lowering after each non-REM/REM sleep cycle (Borbely, 1982).

Continually restricting sleep increases first SWA values, and in turn, raises an individual's sleep debt and tiredness (Borbely, 1982; Wilckens et al., 2018). An ideal sleep period ends with natural waking due to minimal SWA levels as a result of the sleep process (Borbely, 1982). Numerous studies have linked higher first natural SWA levels with degrading next-day performance (Diep, et al., 2020; Maatta et al., 2010; Van Dongen et al., 2003; Wilckens et al., 2018) but few, if any, have tried to link final natural SWA levels, as measured by EEG, with next-day performance.

Other considerations when considering previous sleep restriction and performance studies include intervention length (usually lasting one to four nights), analysis of partial sleep period (often the first sleep cycle), and short PVT tracking periods of the following day (mostly confirmed to morning times). This study is one of the first investigations to study polysomnography over the course of seven nights, compare multiple sleep metrics through multiple sleep cycles each night, and track PVT every few hours throughout the entire waking period.

One example of how this study advances knowledge from previous research is by comparison to the work of della Monica and colleagues (2018) that appeared in Frontiers of Psychiatry. That study employed one-night polysomnography to study individuals aged 20-84 years old performing tasks up to five times the next day. The authors investigated 51 performance measures (including sleep metrics, like SWA and executive function performance, like PVT) leading to an array of different results, which were partially discussed earlier.

The present study narrows the population to 18-35 years old, utilizes polysomnography for an entire week's worth of restricted sleep, analyzes seven days of next-day performance accomplished up to 10 times per day and focuses on the unique relationship between specific measures (SWA during sleep and PVT during the following day) and their potentially unique relationship.

Based on results from previous studies and to better understand the cumulative impact of continuous restricted sleep, the following hypotheses were tested:

1.7 Hypotheses

The hypotheses will focus on the impact of continually manipulated sleep restriction on various EEG measured sleep metrics and more importantly to this study, the relationship between SWA and EF performance.

The first series of hypotheses involve the impact of continual sleep restriction on different individual sleep metrics, without regard to the relationship with executive function.

1. The first SWA maximum will increase significantly across the seven nights of restricted sleep.

This prediction will occur since each participant is awoken after five hours in bed and not able to try to sleep again for 19 hours. Sleep debt will continue to climb as the participant is deprived of proper time in bed.

2. The final SWA maximum will also increase significantly across the seven nights of restricted sleep.

If five hours of sleep per night is properly restrictive, sleep is not having its proper restorative impact. Final SWA values should increase when combined with higher first SWA values and the body's inability to adequately recover from continually short sleep periods.

Hypothesis 3 combines the previous two hypotheses. If final SWA values increase throughout the week and the body is not properly quenching its sleep debt, these final values should increase at a higher rate than first SWA values.

3. The slope of the line connecting the first and last maximum will significantly get closer to zero (i.e., less negative in value) over the course of seven nights of restricted sleep.

The final SWA maximum should always be smaller than the first SWA maximum, so the slope of the line connecting these values will be negative. If the final SWA maximum values increase more drastically over time, this line slope will become more negative as it approaches zero. Graphically, this phenomenon will appear as a flattening line over time.

The central set of hypotheses predict EF behavior as a function of different sleep metrics. If response time increases during the restricted sleep period, sleep metrics that are impacted by this sleep restriction should also change along with increased response times.

4. Response time will increase as a function of increased first SWA maxima, regardless of sleep night.
5. Response time will increase as a function of increased final SWA maxima, regardless of sleep night.
6. Response time will increase as the slope of the line connecting the first and last SWA maxima approximates zero, regardless of sleep night.

Chapter 2: Methods

2.1 Study Protocol

This study was performed at the Walter Reed Army Institute of Research (WRAIR) Sleep Research Center at the Center for Military Psychiatry and Neurosciences Research in Silver Spring, Maryland. The study protocol was approved by WRAIR's Institutional Review Board (IRB) and the data transfer was approved by the IRBs of WRAIR and the University of Maryland (UMD).

2.1.1 Sleep Schedules

Enrolled participants were randomly assigned to one of two sleep schedules, each lasting 40 days. Both schedules consisted of the same seven sleep conditions arranged in a different order. The baseline for both schedules consisted of a 14-day at-home habitual sleep condition, followed by a 7-day sleep extension condition inside the Sleep Research Center. In Schedule 1, participants underwent a 62-hour sleep deprivation condition, while in Schedule 2, they experienced a 7-day sleep restriction condition in the laboratory. After either deprivation or restriction, each group had a night of recovery sleep (10 hours in bed) before proceeding with another 7-day sleep extension condition in the laboratory. Following this second week of extended sleep, Schedule 1 entered a 7-night sleep restriction condition, while Schedule 2 faced 62 hours of total sleep deprivation. Both groups finished with a night of recovery sleep. Below is a timeline of the two different schedules with differences bolded and in italics:

Schedule 1: Baseline (BSL) → Sleep Extension (SE) → ***Total Sleep Deprivation (TSD)*** → Recovery Sleep → Sleep Extension (SE) → ***Sleep Restriction (SR)*** → Recovery Sleep

Schedule 2: Baseline (BSL) → Sleep Extension (SE) → ***Sleep Restriction (SR)*** → Recovery Sleep → Sleep Extension (SE) → ***Total Sleep Deprivation (TSD)*** → Recovery Sleep

The calendars below are samples for both sleep schedules with nights of interest in bold. These sample schedules are based on a potential participant starting on a Sunday.

Schedule 1	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Day 1-7	Home	Home	Home	Home	Home	Home	Home
Day 8-14	Home	Home	Home	Home	Home	Home	Home
Day 15-21	SE	SE	SE	SE	SE	SE	**SE**
Day 22-28	TSD	TSD	TSD	Recovery	SE	SE	SE
Day 29-35	SE	SE	SE	SE	**SR**	SR	SR
Day 36-40	**SR**	SR	SR	**SR**	Recovery		

Schedule 2	Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
Day 1-7	Home	Home	Home	Home	Home	Home	Home
Day 8-14	Home	Home	Home	Home	Home	Home	Home
Day 15-21	SE	SE	SE	SE	SE	SE	**SE**
Day 22-28	**SR**	SR	SR	**SR**	SR	SR	**SR**
Day 29-35	Recovery	SE	SE	SE	SE	SE	SE
Day 36-40	SE	TSD	TSD	TSD	Recovery		

Baseline (BSL)-

To establish each participant’s typical nightly sleep pattern, their regular sleep/wake cycles were tracked for 14 days using both the Readiband (Fatigue Science, Inc.) and the Motionlogger (Ambulatory Monitoring, Inc.) wrist actigraphs and sleep diaries. The average sleep duration over these 14 days was considered as the participant’s baseline sleep duration. This ensured adherence to a standard sleep schedule, which falls between 6 and 8 hours of sleep per night. If a participant typically slept less than 6 hours per night, a week with 5 hours per night in bed would not significantly restrict sleep. Similarly, if a participant typically slept more than 8 hours per night, a week with 10 hours per night in bed was not regarded as extended sleep.

Sleep Extension (SE)-

Following a 14-day baseline condition, a 7-night sleep extension condition was planned. Participants, after engaging in their usual daily activities outside the lab, returned to the lab around 1830 hours. A 10-hour bed rest period was allotted for active sleep attempts. Bedtime was established at 2100 hours, and wake-up time was consistently at 0700 hours throughout the week.

Total Sleep Deprivation (TSD)-

The total sleep deprivation condition lasted for 62 hours, starting at 0700 on the seventh night of sleep extension in the sleep lab. This 62-hour condition ended at 2100 two days later. For example, if the sleep deprivation condition began at 0700 on a Friday, it would conclude at 2100 on the subsequent Sunday. During this time, participants were involved in various tasks.

Sleep Restriction (SR)-

Contrary to the sleep extension condition, participants resided in the sleep laboratory for the entire 7-day sleep restriction condition. The rationale for this continuous stay in the lab was to prevent participant's daytime napping while they were outside the lab. During this week, participants remained active throughout the day. Each night, participants' time in bed was limited to 5 hours, from 0200 hours to 0700 hours.

Recovery Sleep (RS)-

Following both sleep restriction and total sleep deprivation conditions, participants experienced a 10-hour night of recovery sleep. This served as a sleep washout period to help 'reset' their circadian rhythms. The recovery sleep was scheduled from 2100 to 0700 hours.

2.1.2 Participants

The WRAIR sleep laboratory has the capacity to accommodate four participants simultaneously. Over the course of three years, interrupted by the beginning of the COVID-19 pandemic, data was collected in four separate sessions. From the pool of 16 participants, eight successfully completed the 40-day study protocol. Successful completion is defined as the fulfillment of all seven sleep conditions and the execution of 80% or more of the scheduled PVT tasks during each condition. The criteria for inclusion and exclusion are detailed in Appendix 1.

Among the final eight participants, five were female. Four identified as Asian American, while two identified as African American and two as Caucasian. The participants had an average age of 27.75 years with a standard deviation of 5.9 years. Their average BMI was 25.5 with a standard deviation of 3.3. Over a habitual two-week period, their average sleep duration was 435.8 minutes (approximately 7.3 hours) with a standard deviation of 43.4 minutes per night.

2.1.3 First Screening Visit

Upon obtaining informed consent, the prospective participant undertook a multiple-choice comprehension assessment. Following the successful completion of this assessment, the participant was subjected to a standard physical examination administered by a certified medical professional. The participant was then screened through a series of surveys and an interview. The interview aimed to ascertain the participant's eligibility based on a set of predetermined inclusion and exclusion criteria, overall health status, caffeine consumption habits, and sleep patterns (see Appendix 1).

Once the participant successfully passed all the screening procedures, they were equipped with an actigraph wearable device. Comprehensive instructions pertaining to its operation were provided to the participant. In addition, the participant was provided a dedicated smartphone intended exclusively for executing tasks related to the study, like PVT tests and caffeine consumption. The participant was instructed to adhere to their habitual sleep patterns over the subsequent two weeks, and guidance was provided on maintaining a sleep diary and tracking other habits relevant to the study.

Sleep EEG data were collected from each participant over four distinct nights, utilizing 18 measurement sites along with two references A1 and A2 as per the International 10-10 system.

Actigraphy-

Participants wore the actigraph device throughout the 40-day study period. During the first two weeks, the device played a crucial role in determining each participant's habitual sleep patterns under normal home conditions. The device was also worn in the sleep laboratory, supplemented by a second actigraph device. This redundancy was important to address any potential technical issues with PSG and to validate PSG measurements.

Psychomotor Vigilance Task (PVT)-

The PVT requires participants to press a button as quickly as possible when a stimulus appears on the screen. This analysis utilized one version of the PVT, lasting five minutes. The average response time and the number of lapses were derived from the PVT, with a lapse defined as a failure to respond to the stimulus within 0.5 seconds. It was anticipated that the average response time would increase during the sleep restriction condition compared to the baseline. Conversely, this metric should decrease during the sleep extension condition. The PVT was administered approximately every two hours.

2.2 Data Processing

Sleep EEG data were analyzed for four separate nights per participant. The measured sites are Fp1, Fp2, Fpz, F3, F4, F7, F8, Fz, C3, C4, Cz, P3, P4, Pz, T7, T8, P7, P8, and two reference sites, A1 and A2. Site names are based on the 10-10 international electrode system (Electrode Position Nomenclature Committee, 1994). The four nights are an extended night of 10 hours of sleep (EXT) and nights one, four, and seven of restricted sleep lasting five hours (SR1, SR4, and SR7). To keep comparisons similar, only the first five hours of the extended night were used. This is similar to a format used by Skorucak, Arbon, Dijk and Achermann (2018). Part of the sleep analysis includes variable changes over the course of a given period and changes occurring during hours six through ten of extended sleep are not possible during the five hours of restricted sleep per night.

The EEG data collected during sleep were processed using the EEGLAB toolbox in MATLAB (Mathworks, Inc., Natick, MA, version R2021b). EEG and reference channels were identified and labeled using the Montreal Neurological Institute (MNI) coordinates for the Boundary Element Model (BEM) Dipole Fitting (DIPFIT) model (Dayarian & Khadem, 2024).

The high-pass filter was above 0.3 Hz using the windowed sinc Finite Impulse Response (FIR) filter (Iber, Ancoli-Isreal, Chesson & Quan, 2007). Bad channels were automatically identified and removed with the Clean Rawdata plugin and ASR (artifact subspace reconstruction) process (Mullen et al., 2005). This removed any channels that were flat for more than five seconds, a maximum acceptable high-frequency noise standard deviation of four and a minimum acceptable correlation with nearby channels of 0.8 (Iber et al., 2007). These removed channels were interpolated to preserve the integrity of the EEG data structure. This interpolating process resulted in a 24-channel montage with cleaned data. For all left hemisphere electrodes, the value of A2 was subtracted, and for all right hemisphere electrodes, the value of A1 was subtracted. For all midline electrodes, the average values of the two reference sites, A1 and A2, were subtracted.

Next, a low-pass filter was applied using EEGLAB with a cutoff frequency of 35 Hz (Iber et al., 2007). The entire dataset was then adjusted according to two event markers, which indicate the beginning (lights off) and the end (lights on) of the EEG sleep recording (Iber at al., 2007). Subsequently, the decimation process was utilized to reduce the sampling rate of the signal from 256 Hz to 128 Hz to facilitate a better separation of noise components through the Extended InfoMax Independent Component Analysis (ICA) with the corrected rank. The unmixing matrix obtained from the decimated EEG data was applied to the dataset that was not downsampled.

Artifacts were identified using ICLabel, which was configured to operate within a spectral range of 0.3-35 Hz. When the probability of an independent component exceeding a threshold, set at 90% in ICLabel, indicated a high likelihood of being classified as muscle, eye movement, or line noise, these identified artifacts were subsequently removed (Pion-Tonachini, Kreutz-Delgado, & Makeig, 2019).

For Fz, the spectrogram was computed from 0.3 to 13 Hz with a spectral resolution of 0.01 Hz using a 30-second Hamming window with a 15-second overlap. The power spectrum of SWA was calculated from 0.5 and 4 Hz over the entire five-hour time interval.

Thresholds were established at a level representing 10% of the height of the prominence for the first and last full sleep cycle. These thresholds, intersecting the bottom of each side of the curve, allowed for the identification of the start and end points of each wave. If the thresholds did not intersect either side of the curve, they were adjusted to determine reasonable start and end points of the wave. Various sleep-related measures were derived for the first and last full sleep cycles. These include maximum (in dB), sleep cycle width (in minutes), prominence (in dB), and the average power (in dB/min). An additional 10 measures were calculated to further understand the SWA in the first and last full sleep cycles. These measures include the maximum SWA (in dB; (1) MAX1 for the first cycle, (2) MAXL for the last cycle), the time width of the sleep cycles (in s; (3) WIDTH1 for the first cycle, (4) WIDTHL for the last cycle), the energy of the SWA waves (in dB; (5) ENERGY1for the first cycle, (6) ENERGYL for the last cycle), the percentage difference between the first and last cycles in terms of maxima ((7) ΔMAX), width ((8) ΔWIDTH), energy ((9)

ΔENERGY), and the slope of the line connecting the first and last SWA maxima ((10) SLOPE). Variable descriptions are in Table 1. It should be noted that only full sleep cycles were considered, as partial cycles were unpredictable.

The formula for the slope of the line connecting the first and last maxima was ((last maximum-first maximum)/(minute where last maximum occurred-minute where first maximum occurred)). The negative slopes indicated a decrease in the SWA maxima over the course of the sleep cycles. This could potentially suggest changes in sleep depth or quality over time.

Table 1

Descriptions of All 10 Reported Sleep Metrics.

Variable #	Abbreviation	Full name	Description
1	MAX1	First cycle SWA maximum	Highest SWA value during the first full non-REM sleep cycle
2	MAXL	Last cycle SWA maximum	Highest SWA value during the last full non-REM sleep cycle
3	WIDTH1	First cycle width	Time period of the first full sleep cycle, consists of non-REM and REM sleep
4	WIDTHL	Last cycle width	Time period of the last full sleep cycle, consists of non-REM and REM sleep
5	ENERGY1	First cycle energy	Average area of the curve of the first full sleep cycle
6	ENERGYL	Last cycle energy	Average area of the curve of the last full sleep cycle
7	ΔMAX	SWA maxima difference	Difference between the first and last SWA maxima
8	ΔWIDTH	Width difference	Difference between the first and last cycle width
9	ΔENERGY	Energy difference	Difference between the first and last cycle energy
10	SLOPE	SWA line slope	Slope of the line connecting the first and last SWA maxima

2.3 Statistical Analysis

Response time was derived from PVT tasks using PC-PVT 2.0 from the Biotechnology High Performance Computing Software Applications Institute (BHSAI, Fort Detrick, MD). Response time data were processed in Microsoft Excel (Microsoft 365 MSO, Version 2406) with each individual response attempt a separate row. Each of the eight participant's data were processed in a separate Excel spreadsheet. For this paper, response time was analyzed for the entire day after one extended sleep night and nights one, four, and seven of restricted sleep. These four days of individual trials for all eight participants were accumulated into one Excel spreadsheet and imported to R Studio (version 4.2.2). Participants were coded as '1' through '8' with visits coded as '1' through '4' for extended night, sleep restriction night 1, 4, and 7.

There were three levels of statistical comparisons. The first level considers the impact of sleep intervention itself on the eleven measures of interest (response time plus the 10 sleep metrics). Using a standard linear model in R Studio, the relationship between each measure and sleep night was compared. The second level used univariate analysis to regress response time with each of the ten different sleep variables to see if any single variable correlates with response time. This second level was accomplished using a fit linear mixed-effects model with standard notation using the 'lmer' function and participant number as the random effect in R Studio. The third level included multivariate analyses by utilizing the same 'lmer' function and combining different sleep measure combinations. This third level considered all possible combinations of two groups of sleep variables with response time.

Each individual variable was analyzed for significance ($p<0.05$), as single sleep nights and across all sleep interventions. All sleep measures were assessed during the first night of extended sleep and three nights of restricted sleep captured on the first, fourth, and seventh nights. With sleep restricted to five hours in bed per night, only the first five hours of extended sleep were analyzed. This was done so the comparisons have an equal time frame, which is important for sleep research (Skorucak et al., 2018). For example, SWA values continue to diminish the longer sleep occurs. SWA values over a ten-hour sleep period will be lower than a five-hour sleep period, even if no other factors are considered due to the longer time period. PVT tasks, measuring response time, were performed during the day, so comparisons were done for the entire day after sleep interventions. Statistical models examined the full model (if restricted night as a whole was significantly different than extension) and if each restricted night by itself was significantly different than extension. All regressions were performed as two-tailed tests, except for slope. In a healthy individual analyzed with properly working machinery, last SWA maxima will be lower than first SWA maxima. This relationship allows for slope regressions to be performed as one-tail tests. Converting a two-tailed test to one-tailed includes halving the two-tailed test results.

Response time (in milliseconds) was measured through PVT performance. Each in-lab PVT session consisted of nearly 100 responses. There were nine separate sessions performed the next day after each night of restricted sleep. Each session occurred roughly two hours after the previous one. After taking out false starts, each participant responded to nearly 800 stimuli each day. There were eight PVT sessions after the extended night sleep period.

During sleep, SWA increases during the first part of non-REM sleep, decreases during the second half of non-REM sleep and approximates zero during REM sleep. Once another non-REM sleep cycle begins, SWA begins to rise. This can be seen in the figures in Appendix 2. There are multiple variables that can be analyzed during a given night of sleep. For this study, each cycle's SWA maximum, time duration (width) of each cycle, SWA average energy for each cycle and the slope of the line connecting the SWA maxima were calculated for all eight participants. This study examined the first SWA cycle maximum, the last SWA cycle maximum, and the differences between the first and last full sleep cycle for maxima, width, and energy. The slope of the line connecting the first and last maxima were also analyzed.

Chapter 3: Results

3.1 Response Time

Table 2 provides each participant's average response time for the entire day after one night of extended sleep and the first, fourth, seventh nights of restricted sleep. After each of the three restricted sleep nights, response time was slower by at least 3% compared to the extended night. Figure 3 is a line graph of the grand average response times (from Table 2) showing the increase in time after the extended sleep night. Table 3 provides the mean, standard deviation, kurtosis, and skewness for each next-day response time average.

Table 2

Average Response Time (ms).

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	234.13	237.16	234.75	237.26
2	229.78	246.06	285.73	276.44
3	312.98	288.41	353.11	263.64
4	222.36	252.47	240.70	240.56
5	217.43	228.01	232.98	238.49
6	207.20	212.32	218.97	220.03
7	214.59	230.12	198.71	216.96
8	187.45	187.61	187.20	184.66
AVG	228.24	235.27	244.02	234.76

Figure 3

Next Day Average Response Time Line Graph

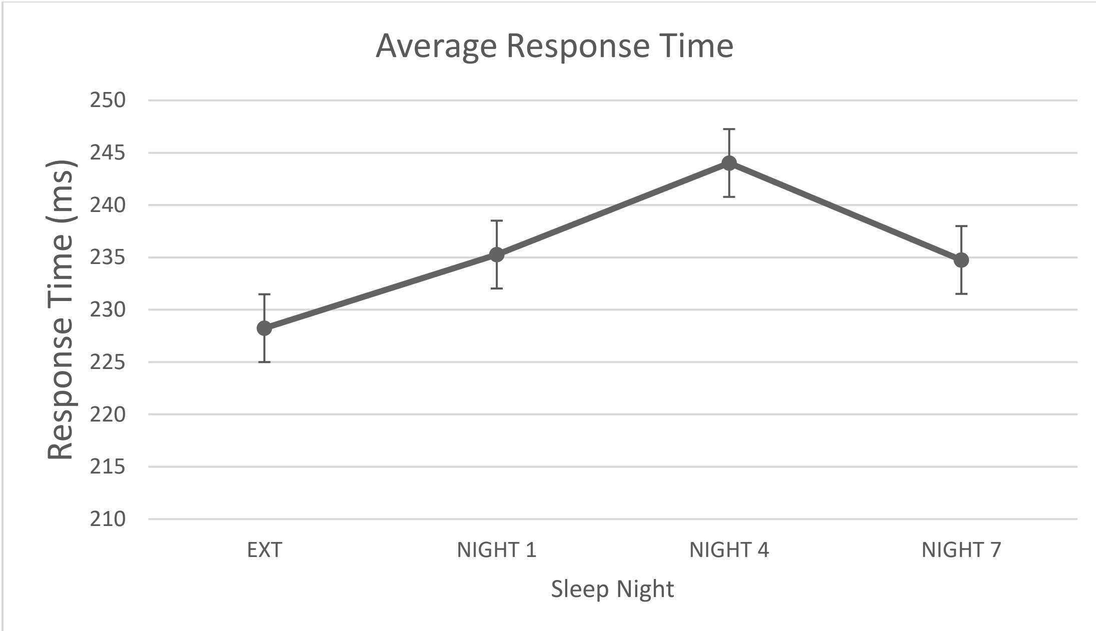


Table 3

Response Time Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	228.24	37.15	4.92	1.96
Night 1	8	235.27	29.56	1.17	0.26
Night 4	8	244.02	53.14	1.97	1.37
Night 7	8	234.76	28.46	0.41	-0.31

Note. Mean and SD in ms.

Examination of the averages of each participant showed no significance below 0.05 (full model, $p=0.88$, all single nights, $p>0.42$). After examining every PVT trial for all eight participants for all four days of interest, PVT performance the day after all three restricted nights achieved significance in comparison to the performance after the extended night (full model and each night, $p<0.001$). With only eight data entries per night based on performance average, there may not have been enough power to find significance. Using all pertinent performance trials instead of collapsing them into block averages provides major significance.

<u>3.2 SWA Measures</u>

3.2.1 First SWA Maxima

As stated earlier, SWA maxima is one measure of sleep need. Table 4 provides the highest SWA level (in decibels) during the first non-REM sleep cycle for each participant during the extended sleep night and nights one, four and seven of sleep restriction. This first full SWA maxima can be an indication of sleep need. There is no statistical significance (all $p>0.39$) with any of the restricted night first SWA levels compared to the extension night. Figure 4 is a line graph of grand averages of the first SWA maxima highlighting its increase for the first and fourth night of restricted sleep. Table 5 provides the mean, standard deviation, kurtosis, and skewness for each nightly first SWA maxima.

Table 4

First SWA Maxima (dB)

ID	EXT	NIGHT1	NIGHT4	NIGHT7
1	27.05	24.61	26.61	23.27
2	29.95	27.93	28.44	27.51
3	23.57	24.16	24.37	22.82
4	23.85	44.94	23.15	24.58
5	22.66	22.33	22.38	21.90
6	19.94	18.82	20.84	20.13
7	22.96	21.12	35.03	19.93
8	21.82	21.44	28.53	21.05
AVG	23.98	25.67	26.17	22.65

Figure 4

Average First SWA Maxima Line Graph

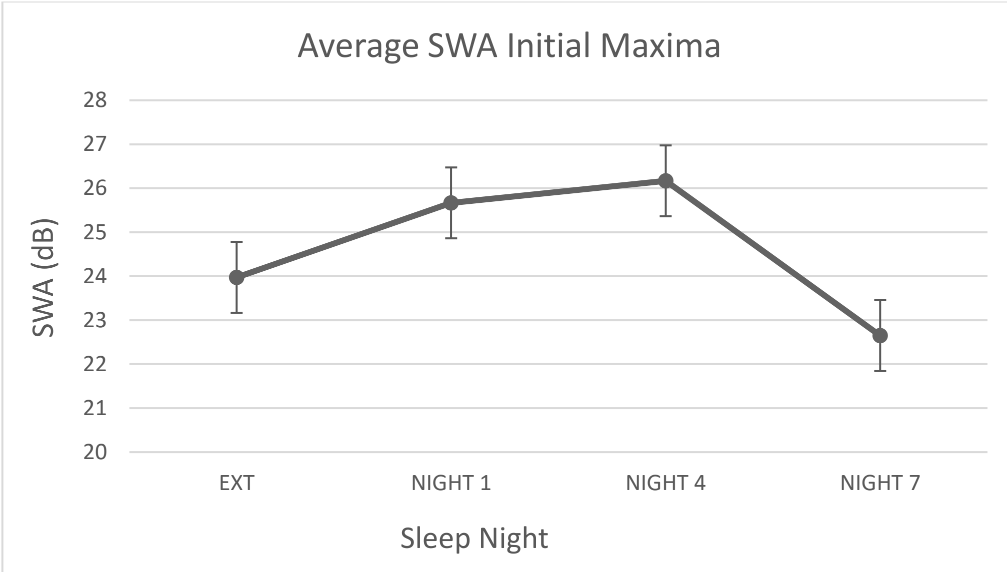


Table 5

SWA First Maxima Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	23.98	3.14	0.94	1.00
Night 1	8	25.67	8.25	5.53	2.25
Night 4	8	26.17	4.54	0.99	0.98
Night 7	8	22.65	2.52	0.78	0.96

Note. Mean and SD in dB.

3.2.2 Last SWA Maxima

The first SWA maxima and its fluctuations provide information related to the impact of continual sleep restriction on sleep need. Examination of the last SWA maxima and its fluctuations provides information related to the impact of continual sleep restriction on the entire sleep period. Table 6 provides each participant's last SWA maxima for all four nights of interest. The average of all eight participants for each night stays relatively consistent. There is no statistical significance (all $p>0.67$) with any of the restricted night's last SWA levels compared to the extension night. Figure 5 provides a line graph of grand averages of the last SWA maxima highlighting its increase for the first and fourth night of restricted sleep. Table 7 provides the mean, standard deviation, kurtosis, and skewness for each nightly final SWA maxima.

Table 6

Last SWA Maxima (dB)

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	24.59	20.16	21.42	21.25
2	27.63	22.80	24.35	26.17
3	23.47	18.36	23.87	18.81
4	17.53	40.92	22.56	23.28
5	21.18	21.32	21.82	20.20
6	16.72	17.37	16.35	16.72
7	22.06	20.62	16.85	17.78
8	19.51	18.23	20.78	20.36
AVG	21.59	22.47	21.00	20.57

Figure 5

Average Last SWA Maxima Line Graph

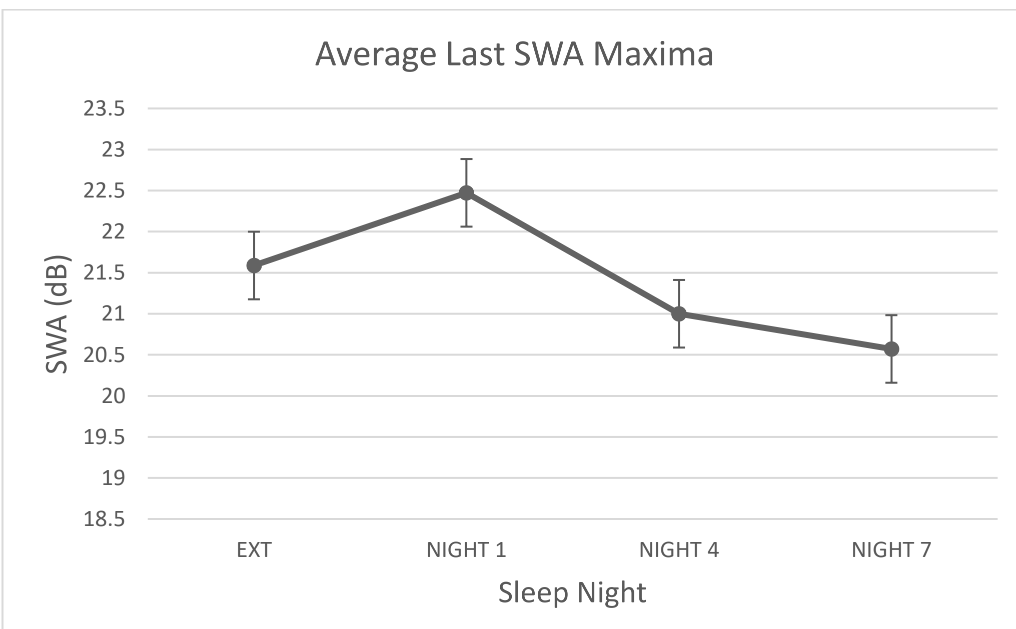


Table 7

Last SWA Mamixa Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	21.59	3.66	-0.56	0.25
Night 1	8	22.47	7.67	6.73	2.53
Night 4	8	21.00	2.97	-0.64	-0.77
Night 7	8	20.57	3.05	0.37	0.75

Note. Mean and SD in dB.

3.2.3 SWA Maxima Difference

In a typical sleep pattern, SWA maxima progressively diminishes as sleep continues. Table 8 provides the percentage difference between the first and last SWA maxima covering the first five hours of an extended sleep night and the full five hours of the first, fourth, and seventh nights of restricted sleep. Percentage differences are not significant (all $p>0.16$), but this is not surprising with only eight data points per night. Figure 6 is a line graph showing the grand average percentage difference between the first and last SWA maxima. The graph provides the percentage difference increasing from the extended night of sleep to the first and fourth nights of restricted sleep. Figure 7 includes both the first and last SWA maxima graphed together to show the specific average changes over the four nights. Table 9 provides the mean, standard deviation, kurtosis, and skewness for each nightly SWA maxima difference.

Table 8

SWA Percentage Difference

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	9.07%	18.07%	19.52%	8.68%
2	7.72%	18.37%	14.35%	4.84%
3	0.43%	23.98%	2.01%	17.58%
4	26.52%	8.94%	2.56%	5.30%
5	6.53%	4.54%	2.51%	7.77%
6	16.15%	7.66%	21.56%	16.97%
7	3.95%	2.40%	51.90%	10.78%
8	10.59%	14.97%	27.17%	3.29%

AVG	10.12%	12.37%	17.70%	9.40%

Figure 6

Average SWA Difference Line Graph

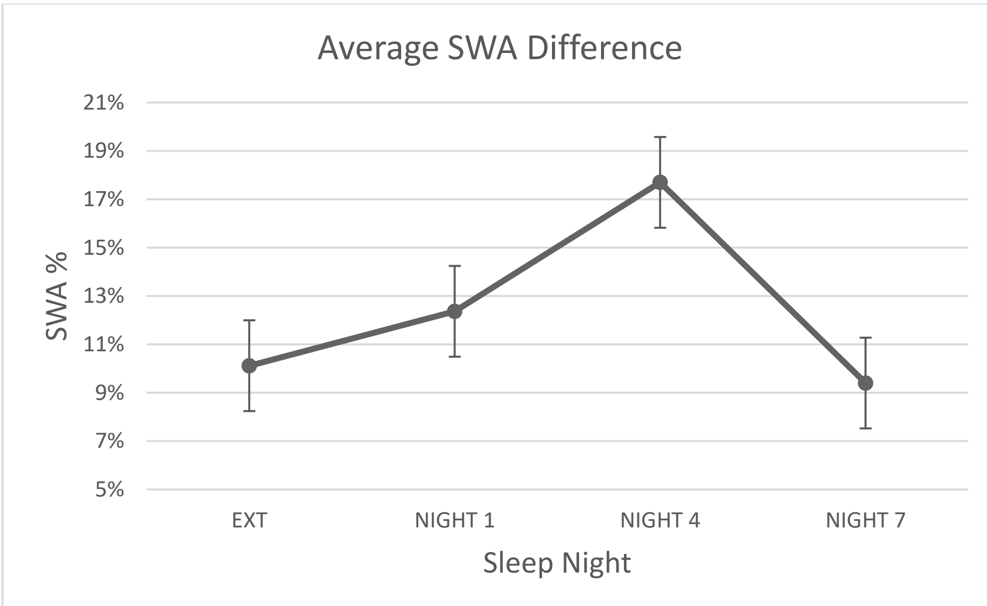


Figure 7

Average First and Last SWA Maxima Line Graph

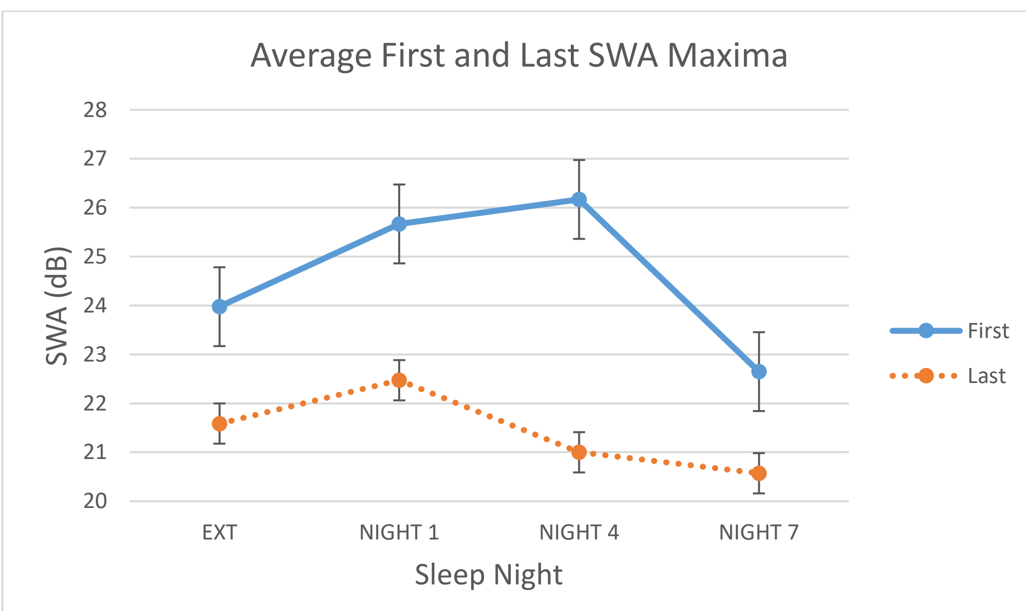


Table 9

SWA Maxima Difference Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	0.10	0.08	1.86	1.24
Night 1	8	0.12	0.08	-1.32	0.16
Night 4	8	0.18	0.17	1.62	1.19
Night 7	8	0.09	0.05	-0.92	0.71

Note. Mean and SD in %.

3.2.4 First Sleep Cycle Width

The width (in minutes) of each sleep cycle indicates the time spent in non-REM sleep. This is also the time between each REM sleep cycle. There is no statistical significance (all $p>0.54$) with any of the restricted night first sleep cycle widths compared to the extension night. Table 10 provides the time (in minutes) of each participant's first non-REM sleep cycle width for all four nights and each night's average. Figure 8 is a line graph of each night's first non-REM sleep cycle width. Table 11 provides the mean, standard deviation, kurtosis, and skewness of the first non-REM sleep cycles.

Table 10

First Sleep Cycle Width (min)

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	48.78	62.29	60.96	73.95
2	60.78	53.03	58.59	53.25
3	59.66	73.70	74.33	64.11
4	42.95	18.74	55.99	51.28
5	64.41	54.10	63.10	60.94
6	49.40	34.31	46.98	41.68
7	76.09	45.28	31.23	50.27
8	25.70	46.96	11.99	66.07
AVG	53.47	48.55	50.40	57.70

Figure 8

Average First Sleep Cycle Width Line Graph

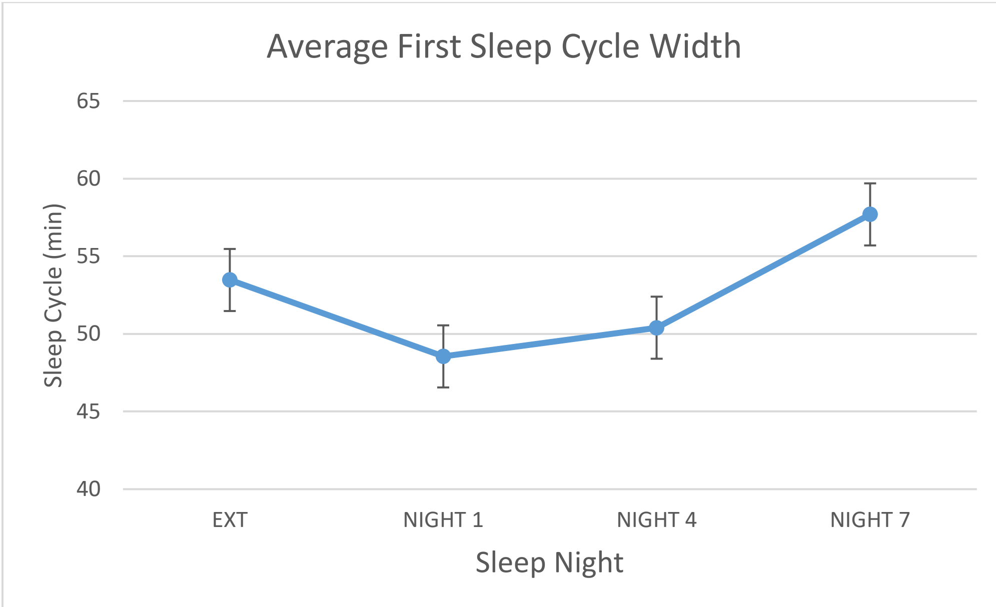


Table 11

First Sleep Cycle Width Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	53.47	15.32	0.63	-0.49
Night 1	8	48.55	16.81	0.55	-0.43
Night 4	8	50.40	19.99	0.86	-1.10
Night 7	8	57.70	10.41	-0.60	-0.31

Note. Mean and SD in minutes.

3.2.5 Last Sleep Cycle Width

Another point of interest is the width of the last non-REM sleep cycle before waking. The last full sleep cycle is also measured in minutes. Partial final cycles are not addressed here because sleep cycles are not symmetrical parabolas and cannot be estimated properly. The last sleep cycle could be a participant's second, third, or fourth sleep cycle of a given night. Using only the second sleep cycle might not capture the full effect of continuous restricted sleep, but not everyone's sleep profile contained more than two sleep cycles every night for comparison. There is no statistical significance (all $p>0.22$) with any of the restricted night final sleep cycle widths compared to the extension night. Table 12 provides the time of each participant's last full non-REM sleep cycle width for all four nights and each night's average. Figure 9 is a line graph of each night's last full non-REM sleep cycle width. Table 13 provides the mean, standard deviation, kurtosis, and skewness of the last full non-REM sleep cycles.

Table 12

Last Sleep Cycle Width (min)

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	44.47	22.24	18.26	42.37
2	83.58	50.55	49.23	84.76
3	72.71	43.19	62.93	24.52
4	41.75	18.59	109.74	64.69
5	48.34	31.96	44.02	31.98
6	46.84	39.90	30.28	47.66
7	16.86	30.95	25.36	41.11
8	10.51	16.68	34.20	63.61
AVG	45.63	31.76	46.75	50.09

Figure 9

Average Last Sleep Cycle Width Line Graph

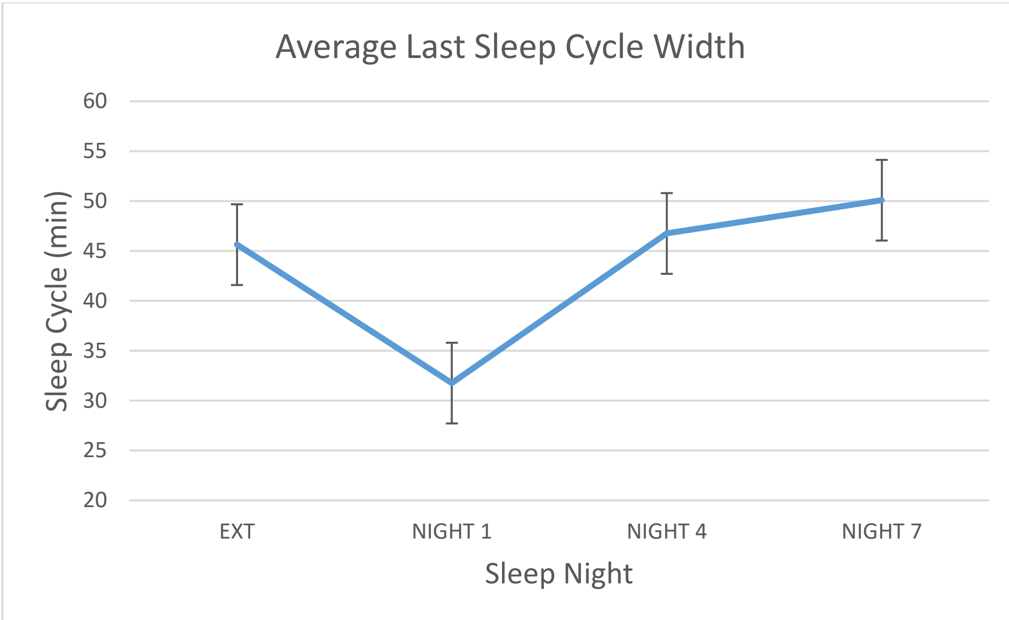


Table 13

Last Sleep Cycle Width Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	45.63	24.67	-0.44	0.09
Night 1	8	31.76	12.20	-1.25	0.22
Night 4	8	46.75	29.13	3.09	1.65
Night 7	8	50.09	19.73	-0.22	-0.31

Note. Mean and SD in minutes.

3.2.6 Sleep Cycle Width Difference

The sleep cycle width difference is the percentage difference between the width of the first full non-REM sleep cycle and the last full non-REM cycle. Table 14 provides the time difference percentage between the first and last full sleep cycle. Positive numbers indicate the last sleep cycle was shorter than the first cycle. There is no statistical significance (all $p>0.32$) with any of the restricted sleep cycle width differences compared to the extension night. This is not surprising because many people do not seamlessly switch through the different sleep stages. Width differences indicated no significance (all p>0.32). Figure 10 is a line graph of the grand average percentage difference between the first and last full non-REM sleep cycle. Table 15 provides the mean, standard deviation, kurtosis, and skewness for the average difference between the first and last full non-REM sleep cycle.

Table 14

Sleep Cycle Width Difference

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	8.85%	64.29%	70.05%	42.70%
2	-37.51%	4.69%	15.98%	-59.15%
3	-21.87%	41.40%	15.35%	61.75%
4	2.80%	0.82%	-96.00%	-26.14%
5	24.96%	40.92%	30.23%	47.53%
6	5.20%	-16.27%	35.54%	-14.33%
7	77.84%	31.64%	18.80%	18.23%
8	59.10%	64.48%	-185.27%	3.72%
AVG	14.92%	29.00%	-11.91%	9.29%

Figure 10

Average Sleep Cycle Difference Line Graph

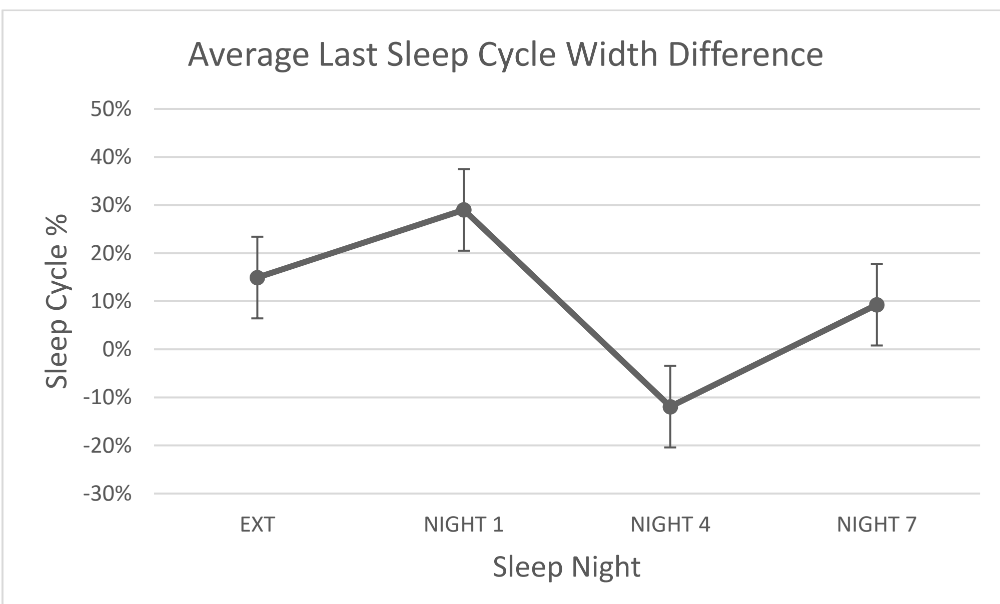


Table 15

Sleep Cycle Difference Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	0.15	0.39	-0.40	0.46
Night 1	8	0.29	0.30	-1.30	-0.27
Night 4	8	-0.12	0.85	1.79	-1.56
Night 7	8	0.09	0.41	-0.80	-0.38

Note. Mean and SD in %.

3.2.7 First SWA Wave Energy Average

SWA wave energy (in dB) is the energy in each distinct wave and is impacted by the maximum of the curve, the prominence rate, and the width of the curve. The average SWA wave energy of the curve is derived from dividing the SWA wave energy into the width of that curve (in minutes). This SWA wave energy average (in dB/min) informs more than a gross energy value. There is no statistical significance (all $p>0.11$) with any of the restricted night first SWA wave energy averages compared to the extension night. Table 16 provides the SWA wave energy average of each participant's first non-REM sleep cycle for all four nights and each night's average. Figure 11 is a line graph of each night's first SWA wave energy average. Table 17 provides the mean, standard deviation, kurtosis, and skewness of the first SWA wave energy average.

Table 16

First SWA Wave Energy (dB/min)

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	10.20	10.74	8.60	11.93
2	14.36	13.91	14.24	12.32
3	12.64	8.89	8.16	7.02
4	10.03	8.27	9.05	10.26
5	9.16	9.13	9.06	8.08
6	6.55	5.30	7.48	5.37

7	5.14	5.30	17.31	5.46
8	4.92	2.24	38.35	8.24
AVG	9.13	7.97	14.03	8.59

Figure 11

Average First SWA Wave Energy Line Graph

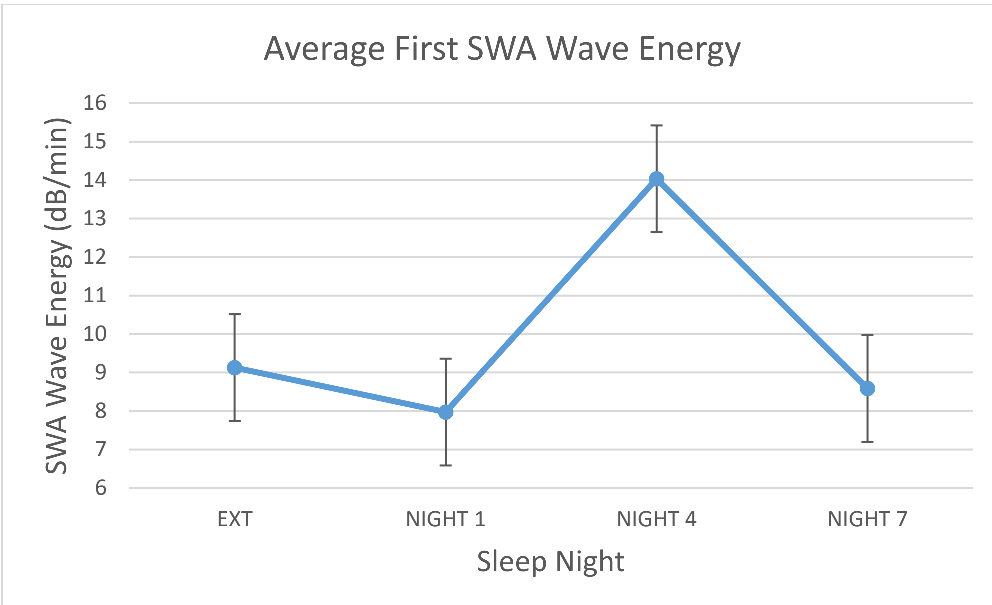


Table 17

First SWA Wave Energy Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	9.13	3.42	-1.11	0.16
Night 1	8	7.97	3.63	0.001	0.03
Night 4	8	14.03	10.41	5.46	2.28
Night 7	8	8.59	2.70	-1.42	-0.31

Note. Mean and SD in dB/min.

3.2.8 Last SWA Wave Energy Average

As with the sleep cycle width variable, SWA wave energy average was calculated for the last full sleep cycle. This last cycle variable comes from the same curve as the ones in the last width cycle. There is no statistical significance (all $p>0.51$) with any of the restricted night final SWA wave energy averages compared to the extension night. Table 18 provides the SWA wave energy average of each participant's last non-REM sleep cycle for all four nights and each night's average. Figure 12 is a line graph of each night's last SWA wave energy average. Table 19 provides the mean, standard deviation, kurtosis, and skewness of the last SWA wave energy average.

Table 18

Last SWA Wave Energy (dB/min)

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	9.45	8.77	12.03	8.15
2	11.25	9.27	8.46	8.83
3	7.12	4.72	7.23	3.66
4	3.56	2.02	6.20	8.64
5	7.97	15.24	13.09	7.87
6	3.55	4.94	2.41	3.86
7	3.15	12.68	2.01	5.06
8	4.13	2.47	4.29	6.83
AVG	6.27	7.51	6.97	6.61

Figure 12

Average Last SWA Wave Energy Line Graph

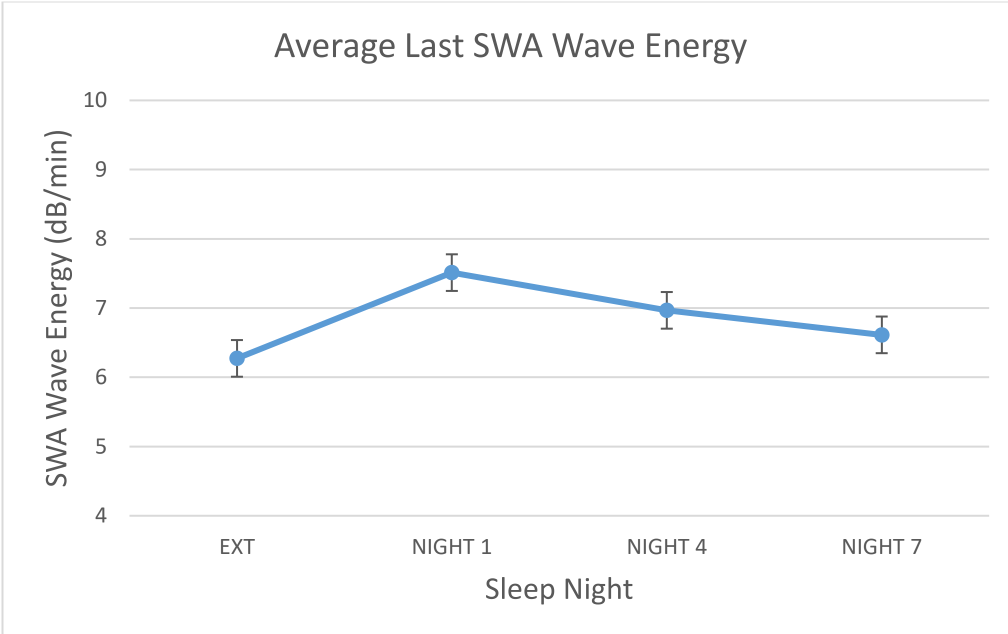


Table 19

Last SWA Wave Energy Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	6.27	3.10	-1.40	0.51
Night 1	8	7.51	4.80	-1.05	0.47
Night 4	8	6.97	4.11	-1.13	0.35
Night 7	8	6.61	2.13	-1.76	-0.31

Note. Mean and SD in dB/min.

3.2.9 SWA Wave Energy Average Difference

SWA wave energy average difference is the percentage difference between the SWA wave energy average of the first and last full non-REM sleep cycle. Table 20 indicates this percentage difference. A positive percentage means the first curve had a higher SWA energy average than the last curve. For all three restricted nights, the difference between the two curves was smaller than the extended night, though not significant (all $p>0.12$). With numerous factors affecting SWA energy, the lack of significance is not surprising. Figure 13 is a line graph of the nightly SWA wave energy average percentage difference between the first and last full non-REM sleep cycle. Table 21 provides the mean, standard deviation, kurtosis, and skewness for the SWA wave energy average difference between the first and last full non-REM sleep cycle.

Table 20

SWA Wave Energy Difference

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7
1	7.44%	18.39%	-39.93%	31.70%
2	21.68%	33.37%	40.57%	28.36%
3	43.66%	46.92%	11.41%	47.85%
4	64.46%	75.57%	31.49%	15.75%
5	13.04%	-66.90%	-44.59%	2.59%
6	45.75%	6.84%	67.72%	28.09%
7	38.71%	-139.04%	88.37%	7.34%
8	16.03%	-9.97%	88.82%	17.11%
AVG	31.35%	-4.35%	30.48%	22.35%

Figure 13

Average SWA Wave Energy Difference Line Graph

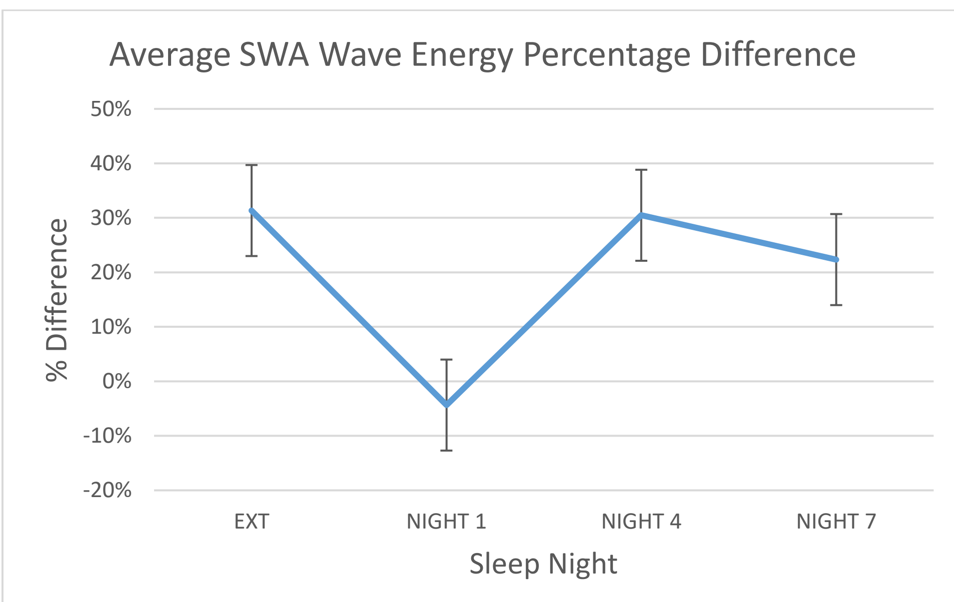


Table 21

SWA Wave Energy Difference Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	0.31	0.20	-0.94	0.42
Night 1	8	-0.04	0.69	1.12	-1.15
Night 4	8	0.30	0.52	-1.20	-0.44
Night 7	8	0.22	0.15	-0.09	-0.31

Note. Mean and SD in %.

3.2.10 Slope

The slope of the line indicates the rate of change between the first and last SWA maxima across the time series. The slope was calculated by taking the difference between the last and first maxima and dividing by their difference in time during the night ((last maximum-first maximum)/(minute where last maximum occurred-minute where first maximum occurred)). All slopes being negative indicate that all last maxima were lower than their corresponding first maxima. The slopes are relatively low because the denominator is in minutes, where the difference is roughly 150-250 minutes per night, and the numerator is SWA power, where the difference is in single or double digits. Line slope was not significant (all $p>0.18$). Table 22 provides the slope of each line connecting the first and last SWA maximum for each participant and nightly average. Figure 14 is a line graph showing the nightly average of all line slopes for each night. Table 23 provides the mean, standard deviation, kurtosis, and skewness for the average SWA wave energy difference between the first and last full non-REM sleep cycle.

Table 22

SWA Maxima Line Slope

ID	EXT	NIGHT 1	NIGHT 4	NIGHT 7

1	-0.0137	-0.0225	-0.0290	-0.0102
2	-0.0094	-0.0331	-0.0363	-0.0065
3	-0.0005	-0.0321	-0.0065	-0.0238
4	-0.0806	-0.0247	-0.0035	-0.0059
5	-0.0106	-0.0109	-0.0054	-0.0160
6	-0.0321	-0.0064	-0.0276	-0.0174
7	-0.0394	-0.0022	-0.0736	-0.0113
8	-0.0127	-0.0151	-0.0425	-0.0056
AVG	-0.0249	-0.0184	-0.0280	-0.0121

Figure 14

Average SWA Maxima Line Slope Line Graph

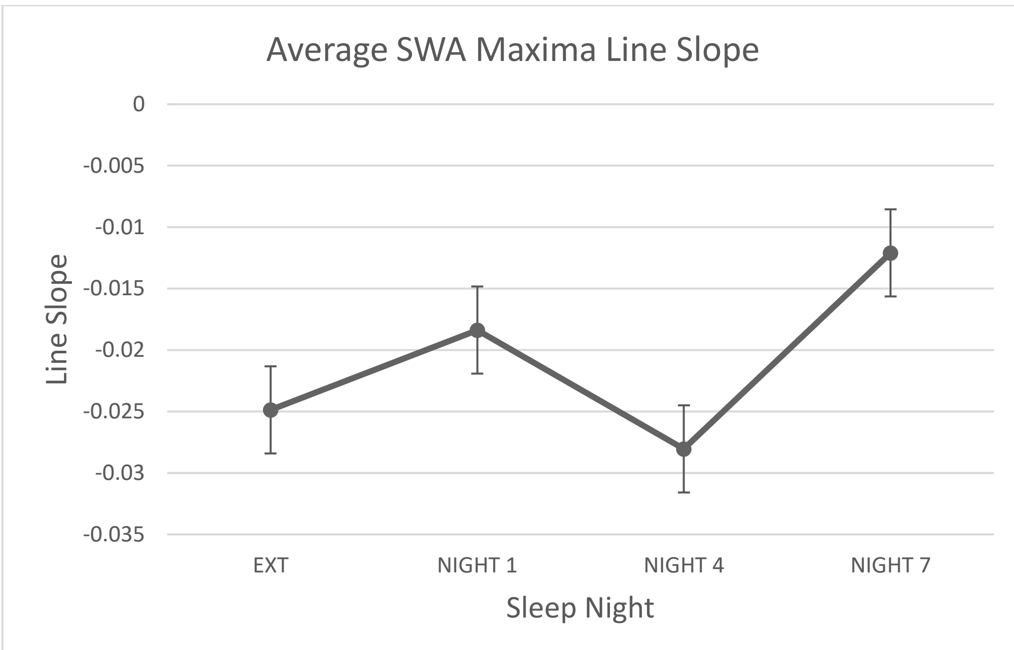


Table 23

Line Slope Descriptive Data

Night	N	Mean	SD	Kurtosis	Skewness
EXT	8	-0.0249	0.0259	2.9084	-1.6765
Night 1	8	-0.0184	0.0116	-1.4882	0.0366
Night 4	8	-0.0281	0.0237	0.7442	-0.8815
Night 7	8	-0.0121	0.0065	-0.3117	-0.7747

3.2.11 Single Variable Summary

After an analysis of each relevant variable, only PVT response time showed significance below 0.05. As expected, PVT response time was significantly slower the day after each of the three restricted sleep nights of interest compared to the day after a night of extended sleep. All three days' worth of response times had significance below 0.001. All other p-values are listed in Table 24 with extended sleep functioning as the comparison baseline.

Table 24

P-values of Single Variables Regressed with Sleep Night

	EXT	NIGHT 1	NIGHT 4	NIGHT 7
PVT AVG	X	0.72	0.42	0.74
ALL PVT	X	**<0.001**	**<0.001**	**<0.001**
MAX FIRST	X	0.51	0.4	0.61
MAX LAST	X	0.71	0.81	0.67
MAX DIFF	X	0.67	0.16	0.89
WIDTH FIRST	X	0.54	0.7	0.6
WIDTH LAST	X	0.22	0.92	0.69
WIDTH DIFF	X	0.6	0.32	0.83
AVG POWER FIRST	X	0.7	0.11	0.86
AVG POWER LAST	X	0.51	0.71	0.86
AVG POWER DIFF	X	0.12	0.97	0.69
SLOPE	X	0.49	0.74	0.18

3.3 Univariate Modeling

After checking to see which individual variables were sensitive to restricted sleep (response time), each variable was regressed individually to response time. The results of those analyses are seen in Table 25. With slowing response time (larger values), two sleep variables significantly changed and one trended toward significance. As response time slowed, the SWA maximum of the final full sleep cycle progressively increased. This indicates that as participants performed worse, sleep need later in the night's sleep period was higher. In other words, the slower response time indicates the body needs more sleep later in the night. Also of significance was the impact of slowing response time on the percentage difference between the first and last SWA maximum difference. As response time slowed, the gap between the first and last SWA maximum decreased. Both SWA maximum values increased, but a significant decrease in the difference between the two helps explain why the last SWA maximum increased significantly while the first SWA maximum increase was not significant. Initial analysis of the relationship with line slope indicates a trend, but not significance. As mentioned earlier, the neurobiology of SWA maxima allows for further one-tailed analysis, which halves the two-tailed process and brings line slope into significance. As response time increased, the slope of the line connecting the maxima decreased (flattened).

Table 25

P-values of Single Variables regressed with Response Time

Variable	P-Value
MAX FIRST	0.79
MAX LAST	**0.05**
MAX DIFF	**0.029**
WIDTH FIRST	0.59
WIDTH LAST	0.34
WIDTH DIFF	0.72
AVG POWER FIRST	0.75
AVG POWER LAST	0.25
AVG POWER DIFF	0.26
SLOPE	*0.08*
SLOPE (One-sided)	**0.04**

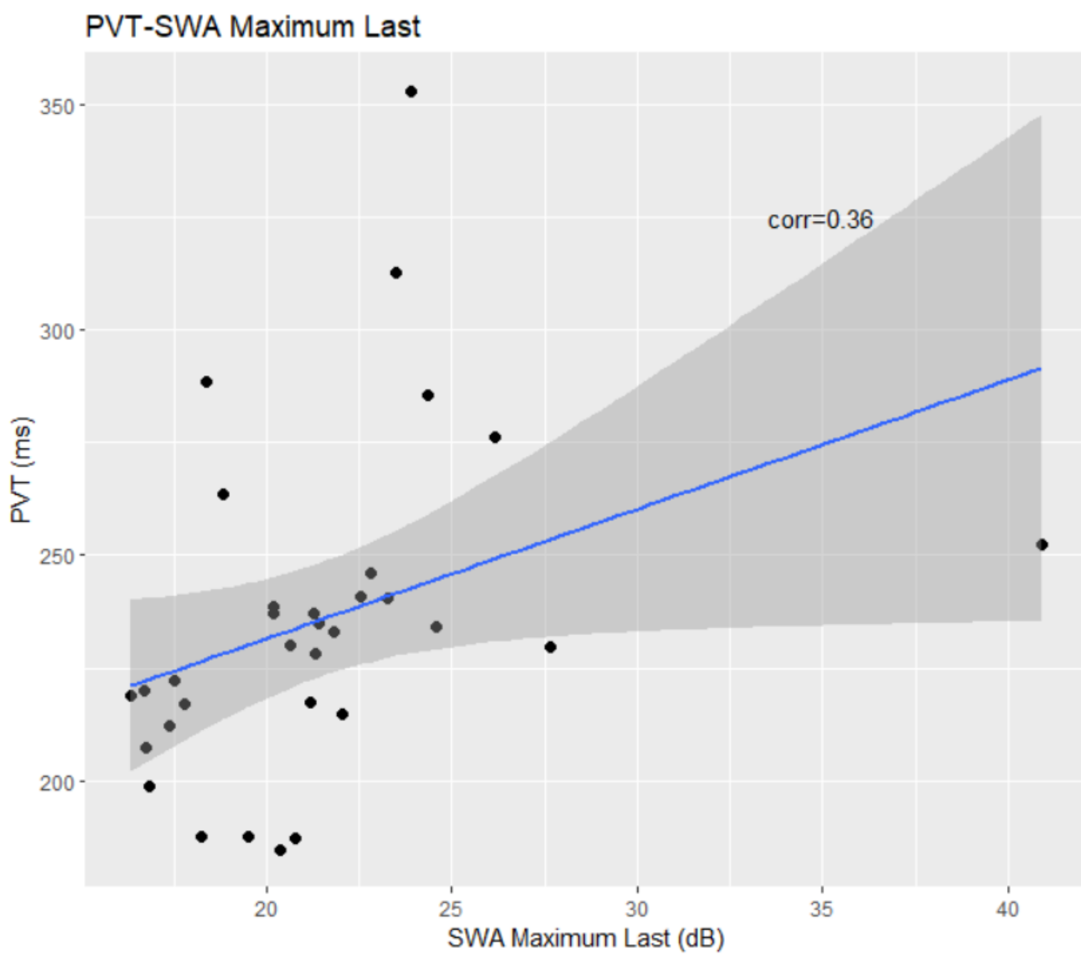
Note. Significant results in bold. Trending results in italics.

3.3.1 Last SWA Maximum and RT

Last SWA maximum values increased during the week of continual restricted sleep but was not statistically significant. As shown in the table above, regressing last SWA maximum values with next-day response time was statistically significant (p=0.05). Figure 15 plots all 32 (eight participants with four EEG tracked sleep nights each) data points measuring the last measured SWA maximum of a tracked night with the average PVT response time throughout that nest day. Figure 15 provides increasing average response time with higher prior night last SWA values. The correlation of that relationship is 0.36, indicating a moderate relationship between the two variables. This data indicates increasing response time follows higher previous night last SWA values.

Figure 15

Last SWA Maximum Regression with Response Time Scatterplot



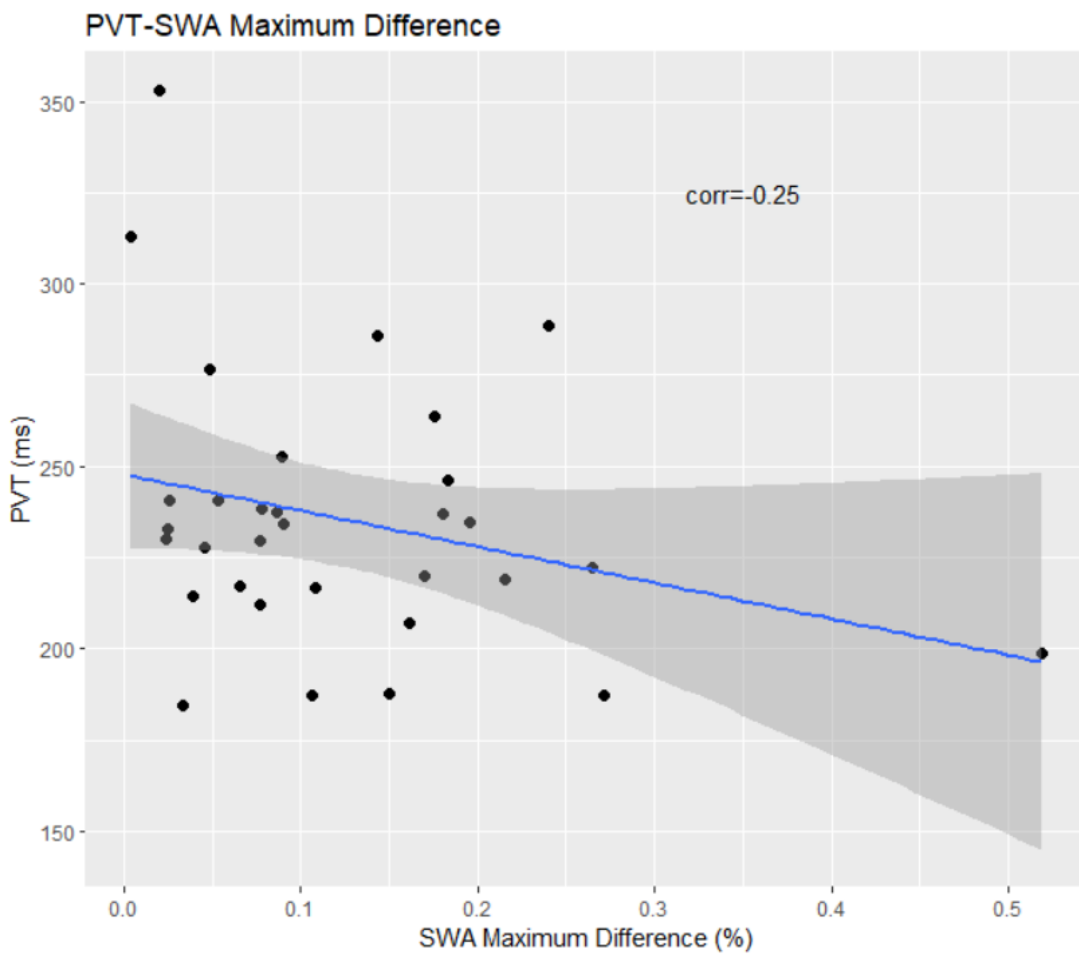
Note. A scatter plot of all 32 sleep nights. Each point is the cross-section of last SWA maximum and the following day's average response time. The blue line indicates the correlation between the two variables.

3.3.2 SWA Maximum Difference and RT

The percentage difference between the first and last SWA maxima in each sleep night is a second variable that was statistically significant regressed with response time, but not sleep night. The significance of the relationship between SWA maximum difference and response time is smaller than 0.05. Figure 16 is a scatterplot of all 32 sleep nights measuring the percentage difference between the first and last SWA maxima and the corresponding next-day PVT response time average. When the difference between the maxima decreases, the matching next-day response time tends to increase. The correlation between these two variables is 0.25, a modest relationship.

Figure 16

SWA Maximum Difference Regression with Response Time Scatterplot



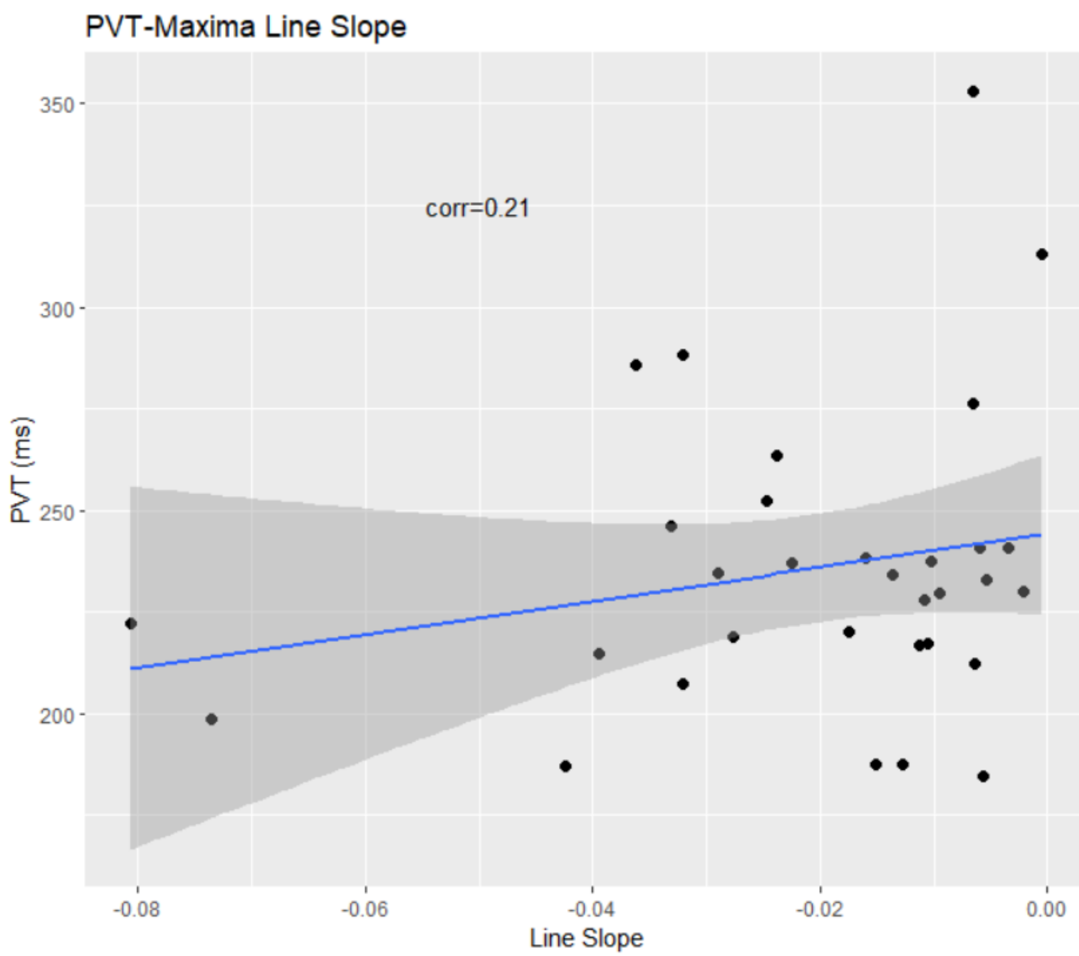
Note. A scatter plot of all 32 sleep nights. Each point is the cross-section of the difference between the first and last SWA maxima and the following day's average response time. The blue line indicates the correlation between the two variables.

3.3.3 Line Slope and RT

First analyses provided a trending, but not statistically significant relationship between the slope of the line connecting the first and last maxima and response time ($p=0.08$). If a participant sleeps for a portion of the intervention period, SWA values will diminish (see Illustration 1). These diminishing SWA values mean the slope of the line will always be negative. A positive slope would indicate the participant was awake during the intervention period, causing the data to be invalid. Since valid slopes must be one direction (or one-tailed), we are able to halve the current p-value to 0.04, allowing for statistical significance. Figure 17 is a scatterplot showing 32 data points of each participant's SWA maxima line slope and next-day average response time, regardless of which night the data point occurred. The less negative the line slope, the slower the next-day response time. The 0.21 correlation indicates a medium level relationship between these two variables. All non-significant scatterplots are in Appendix 3.

Figure 17

SWA Maximum Difference Regression with Response Time Scatterplot



Note. A scatter plot of all 32 sleep nights. Each point is the cross-section of the slope of the line connecting the first and last SWA maxima and the following day's average response time. The blue line indicates the correlation between the two variables.

3.4 Multivariate Modeling

Using mixed effect modeling with participant number as the random effect, all possible permutations of two variable combinations were performed. Each permutation was run in R by adding a second variable to the single variable mixed-effect model. Appendix 4 contains the p-values for all two sleep variables combinations regressed with response time.

Chapter 4: Discussion

The goal of this study was to identify a promising sleep biomarker that can be a potential useful predictor of next-day performance. This goal was accomplished by investigating brain dynamics during a week of restricted sleep and next-day psychomotor executive function performance. We observed a general increase in final SWA maxima, a decrease in the difference between the first and last SWA maxima, and a progressively less negative line slope connecting these maxima over the seven restricted nights that predicted increasing worsening next-day response time performance. This novel approach is a major step forward in helping better understand some of the intricacies of sleep architecture.

Using the most prominent brain region for capturing SWA (frontal, see Cajochen et al., 1999; Skorucak et al., 2018), this investigation indicated the final SWA maxima increased throughout the restricted sleep period in relation to the baseline night and as a predictor of increasing next-day response times. Along with higher sleep onset SWA values, indicating more tiredness upon starting sleep, restricting sleep tends to lead to higher waking SWA values. Higher waking SWA values can lead to degraded performance and an avalanche effect of increasing sleep onset SWA values that are hard to recover from. Skorucak et al. (2018) investigated SWA levels in 35 individuals during a week of six hours of restricted sleep by averaging SWA during the first half of the sleep period. This SWA averaging found non-significant frontal SWA increases, but there was no comparison to future performance or discrimination between the first or second sleep cycle (Skorucak et al., 2018). This study separates the different sleep cycles and compares final SWA maxima to next-day performance using PVT results, generally considered the gold standard of sleep-impacted performance measures. Future analyses could investigate if other EF performance measures are also sensitive to sleep interventions and SWA variability. Some of these measures include trail making on paper, memory tests, and go/no-go tasks.

Current findings suggest that the last SWA maxima is informative on its own, but comparisons to initial SWA maxima can inform more about the recuperative, or lack thereof, possibilities of restricted sleep. This investigation found that the gap (difference) between the first and last SWA maxima shrunk over the seven days of restricted sleep compared to baseline values and could predict increasing next-day response times. This shrinking difference could indicate that continually restricting sleep creates a less recuperative sleep process. Other research has measured multiple sleep cycles (Skorucak et al., 2018), but they focused on different aspects, like SWA rise rate. SWA rise rate can be conducive to length of different sleep stages but might focus more on early tiredness. This study highlights that, not only does restricting sleep create more waking and early tiredness, but that tiredness increases more than at sleep onset and affects performance. Following the avenues of research similar to Maatta and colleagues (2010), more investigations could be completed that focus on activities that focus on different brain regions, accomplished at different intensities at different times, and their impact on first and last SWA maxima, followed by performance measures throughout the following day.

An even more valuable way to conceptualize sleep integrity (or lack of) is the rate of potential sleep regression. This study found a SWA maxima line slope that is becoming less negative compared to baseline measures and associated with deteriorating next-day performance. This can be viewed by comparing the SWA levels in Appendix 2 to the preferred profile in Figure 1. The line slope allows for witnessing a potential accelerating of future tiredness and continued poor performance. Little is currently known about this sleep measure and its impact on performance, but its significance to predicting future performance struggles is a promising candidate for future studies, including other brain regions that might be impacted by different daytime activities. Though the frontal region seems to have the biggest reflection of sleep interventions, regions like the occipital and parietal can also be informative to better access the impact of different sleep interventions on SWA values and next-day performance.

Each of these significant findings have their merits and can help inform future research, but their totality indicates a more intricate relationship that warrants further exploration. This study also found some interesting multi-sleep variable significances (see Appendix 4) that would be enhanced with a larger participant pool. della Monica and colleagues (2018) showed that numerous metrics can be demonstrated to help explain the relationship between sleep and performance. They accomplished this by studying over 200 adults, ranging from 20-84, for one night of eight hours in bed and highlighted multiple relationships between sleep and performance. Adding to these findings can be done by including both the beginning and end of sleep, which can help fill some puzzle pieces in the mysteries of sleep.

There are some limitations inherent to any sleep study. Proper polysomnographic collection involves sleeping in a laboratory while wearing a gelled cap of electrodes. Not only is this uncomfortable, but participants are sleeping in a different room on a different mattress in an unfamiliar setting. Sleeping in a bed without your partner or pets or familiar background noise could enhance or hinder typical sleep habits. Trying to sleep in a quiet room may also be challenging for those used to falling asleep to a television or music or white noise machine.

Some limitations that are specific to this study include the worldwide COVID-19 pandemic. Some participants completed the study before the pandemic and others afterwards. Spending extended periods of time at a facility like WRAIR could cause extra anxiety in some and a sense of safety in others. Anxiety could result from returning to society after lockdowns and being disconnected from the outside world while so much could be changing with friends and family. A sense of security could result from spending time in a world-class facility that is probably safer and cleaner than being home or interacting with strangers. For some, spending days away from social media and news reports can allow for temporary relaxation and calm that is not possible during normal living conditions.

Every participant spent one week lying in bed for five hours per night and 62 hours completely deprived of sleep. With the cross-sectional study design, these two periods occurred at different portions of the 40-day study. Even though there were multiple nights of extended sleep before each period, sleep restriction early or late in the study might not be completely relatable.

Chapter 5: Conclusion

Sleep is such an important biological process for maintaining proper physical health, mental health, and performance that it needs to be better measured than subjective feelings or basic objective tests. Many people either minimize their fatigue levels or try to power through their tiredness. This study begins the process of linking previously unstudied objective measures (SWA maxima difference, SWA maxima line slope, etc.) with measures already validated to be sensitive to sleep loss (response time, first SWA levels, etc.). More research using these newer measures and including other neurobiological tests that test inhibition, motor behavior, planning, and working memory that could lead to a comprehensive panel of assorted metrics that can help better determine fatigue and tiredness in specialized populations, like the military and medical personnel. In this paper, restricting sleep to five hours per night for one week highlighted objective EEG measures showing participants were more tired later in the night with progressively slowing response time and this restricted seep appears to be less restorative than typical sleep.

Appendices

Appendix 1

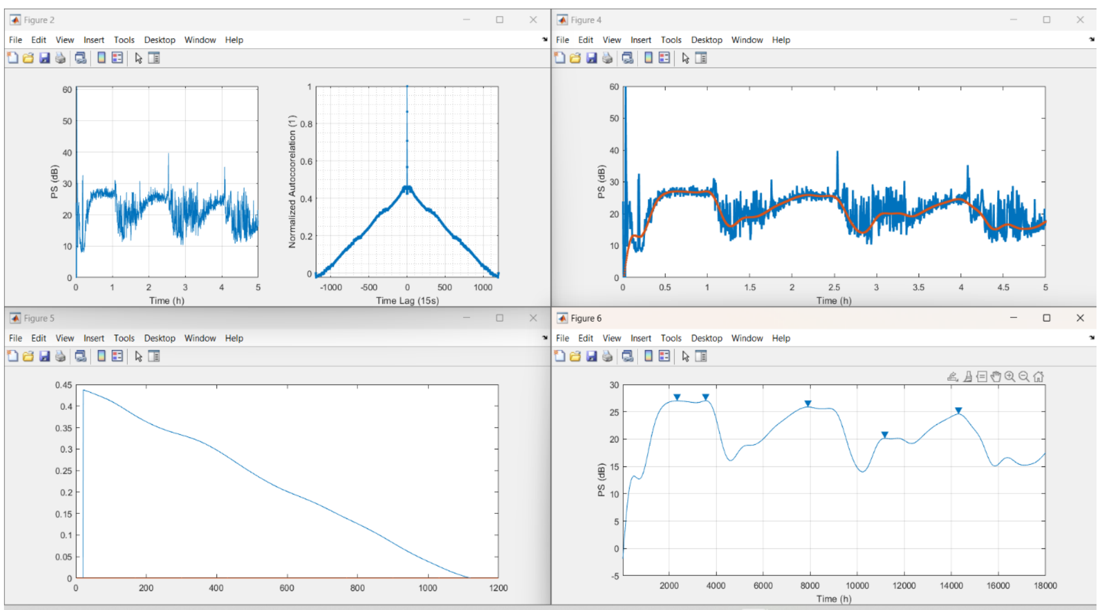
To qualify for this study, each participant must:

- Pass a pre-study standard medical exam
- Be between the ages of 18 and 39
- Be able to spend a week at a time in a sleep lab setting on multiple occasions
- Be able to go without items like cellphones for a week at a time on multiple occasions
- Not be on prescribed or non-prescribed psychoactive drugs, like anti-depressants
- Be on official leave (if employed by the military)
- Pass a comprehension assessment exam
- Habitually sleep between six and ten hours per night
- Sleep during traditional hours (start sleep after 2030 hours during the week and finish sleep before 1100 hours the next morning)
- Nap less than three times per week
- Not have a sleep disorder, like narcolepsy or apnea
- Not use any sleep aid medication the month prior to the start of the study
- Not consume over 600mg of caffeine on a daily basis (about 6 20oz caffeinated sodas, 4 16oz energy drinks or 30oz brewed coffee)
- Not have any neurological disorders, like epilepsy or amnesia
- Score below 14 on the Beck Depression Inventory
- Not have any organ disfunction, like heart, kidney, or liver disease

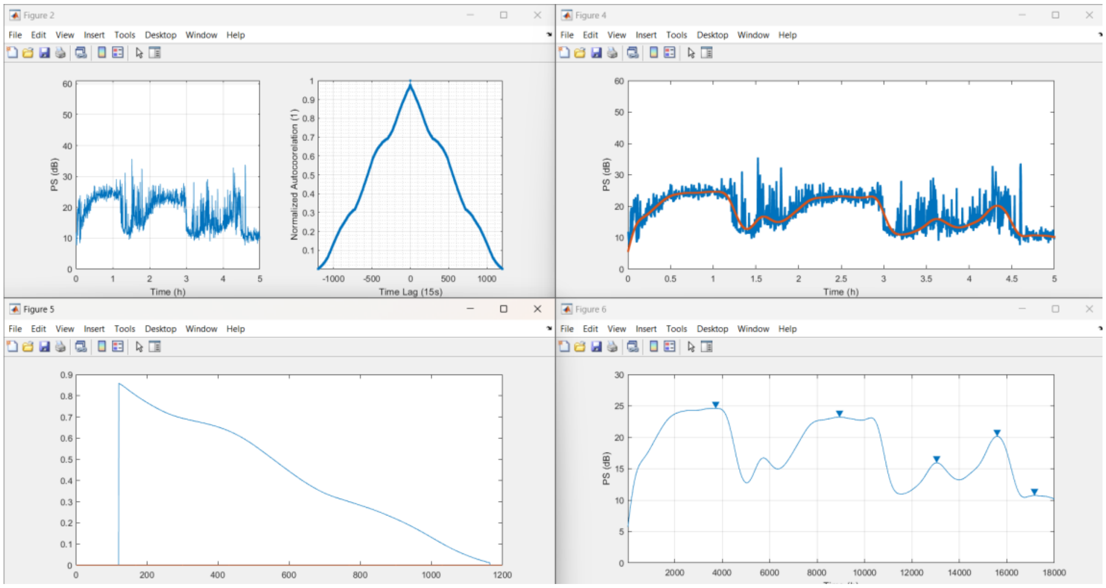
- Have a BMI under 30
- Have a resting blood pressure between 110/70 and 140/90
- Not have been hospitalized for a psychiatric disorder during the previous six months
- Not be a smoker or heavy drinker (14 or more drinks per week)
- Not use illicit drugs
- Women must not be pregnant or lactating

Appendix 2

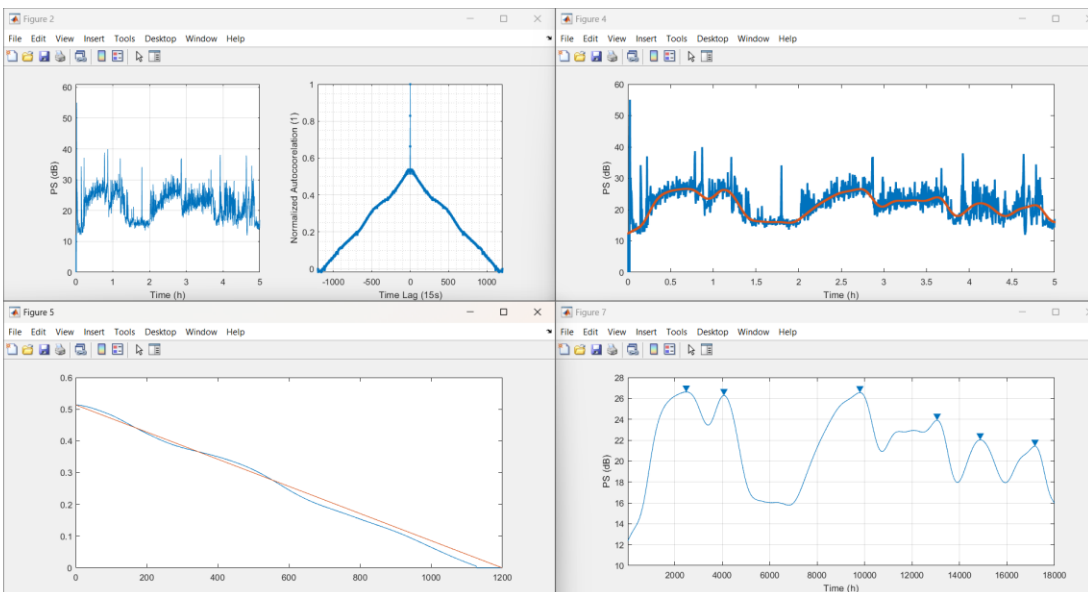
SWA graphs for all participants for all four nights



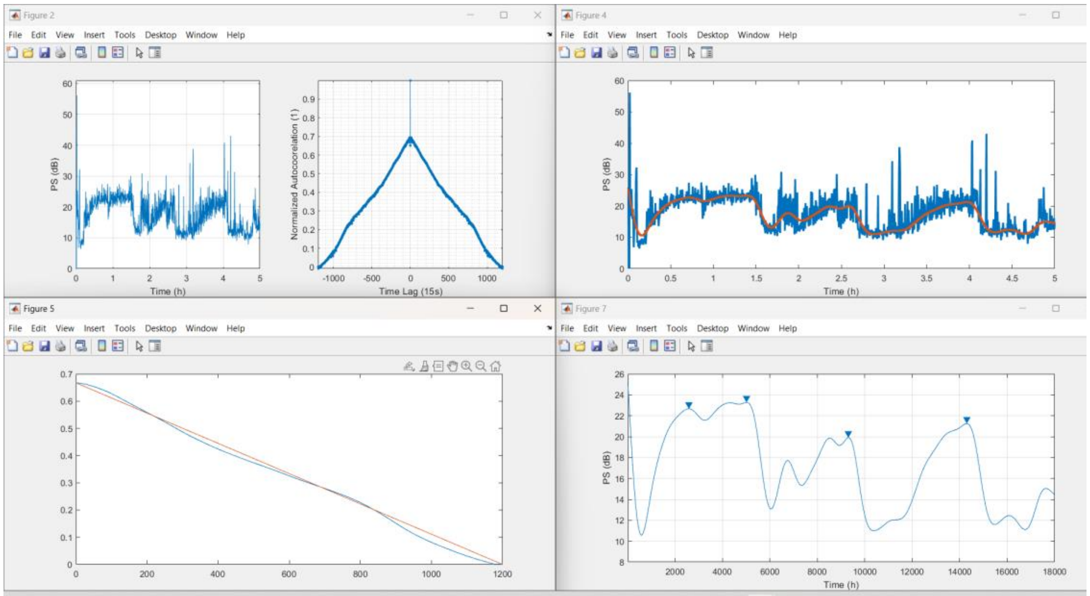
Participant 1-EXT Night



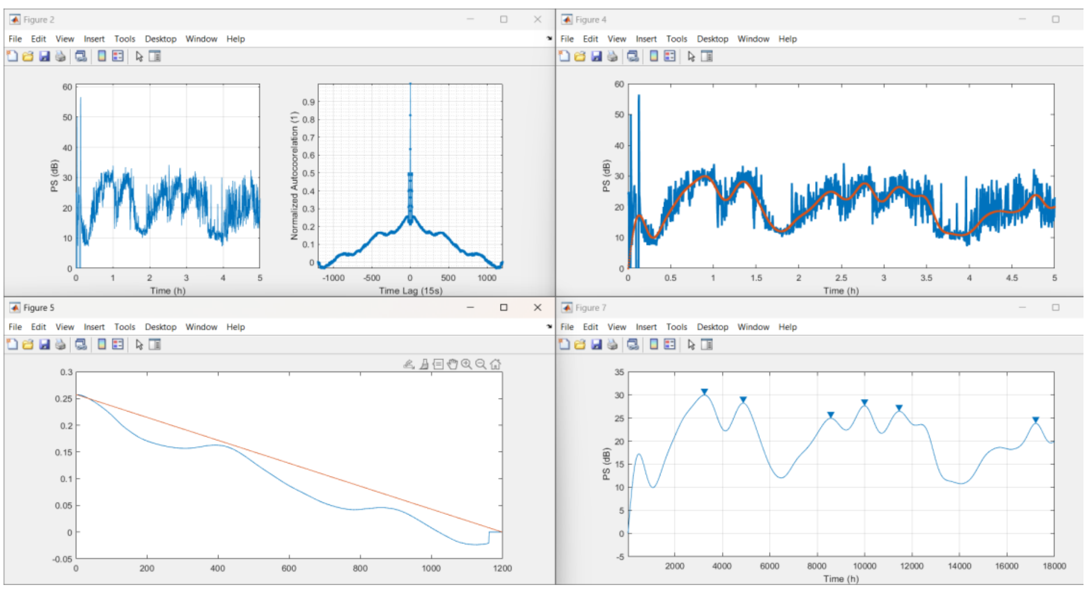
Participant 1-Night 1



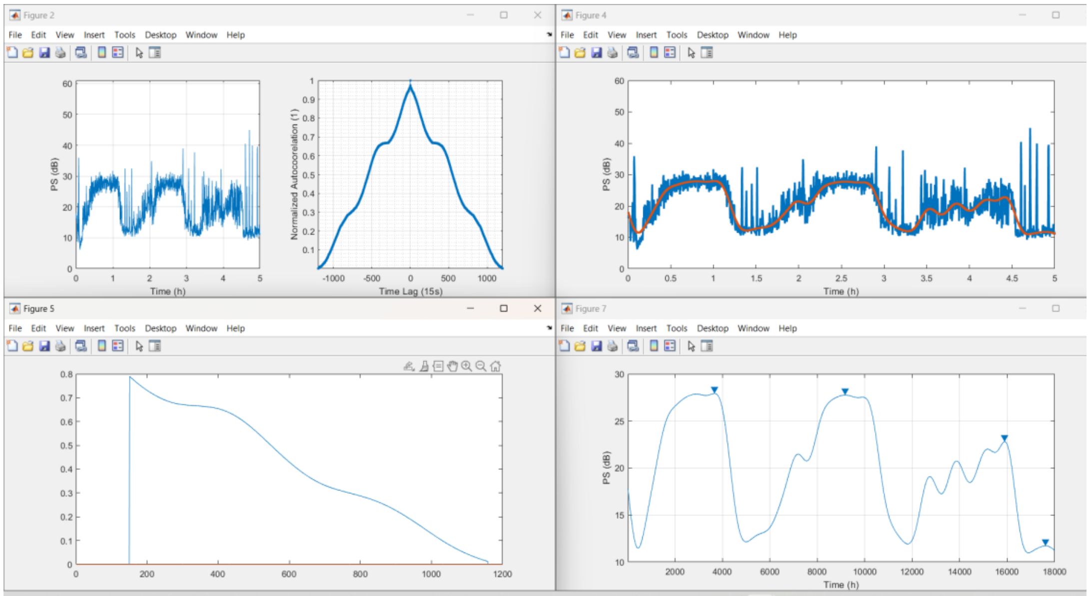
Participant 1-Night 4



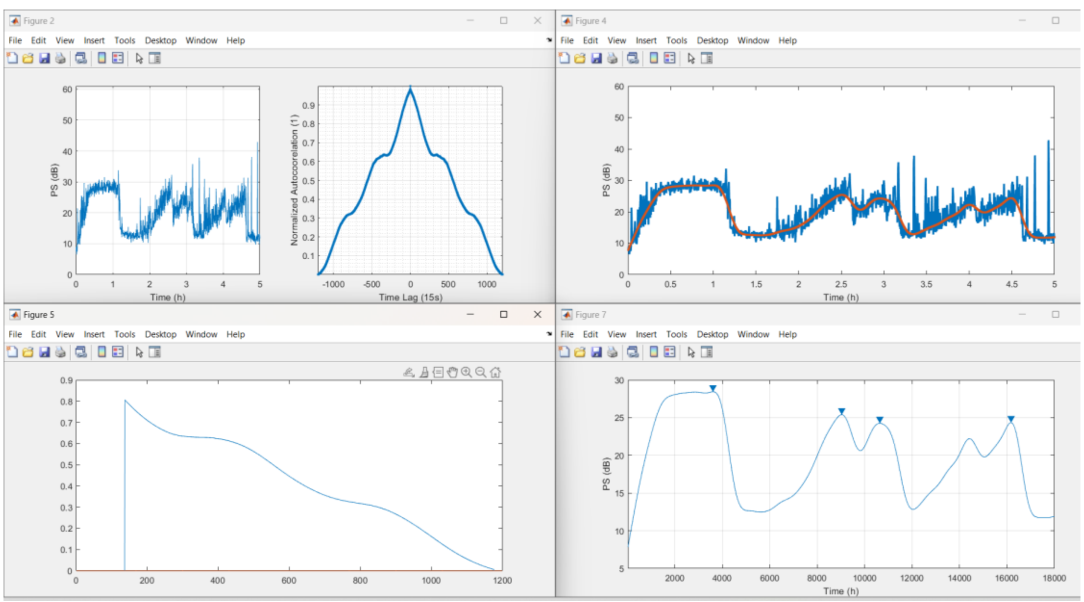
Participant 1-Night 7



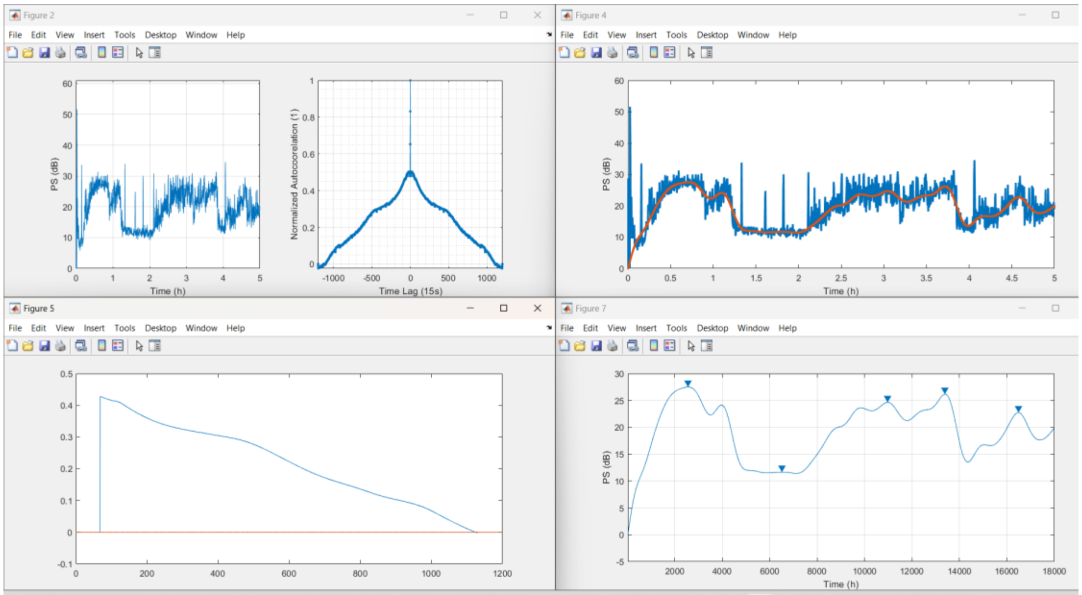
Participant 2-EXT Night



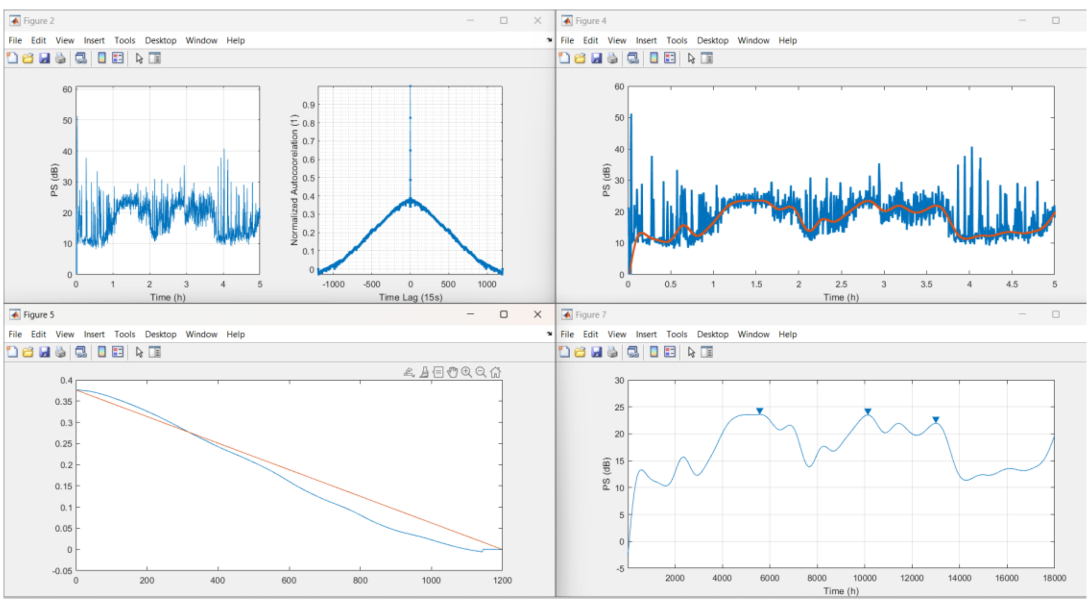
Participant 2-Night 1



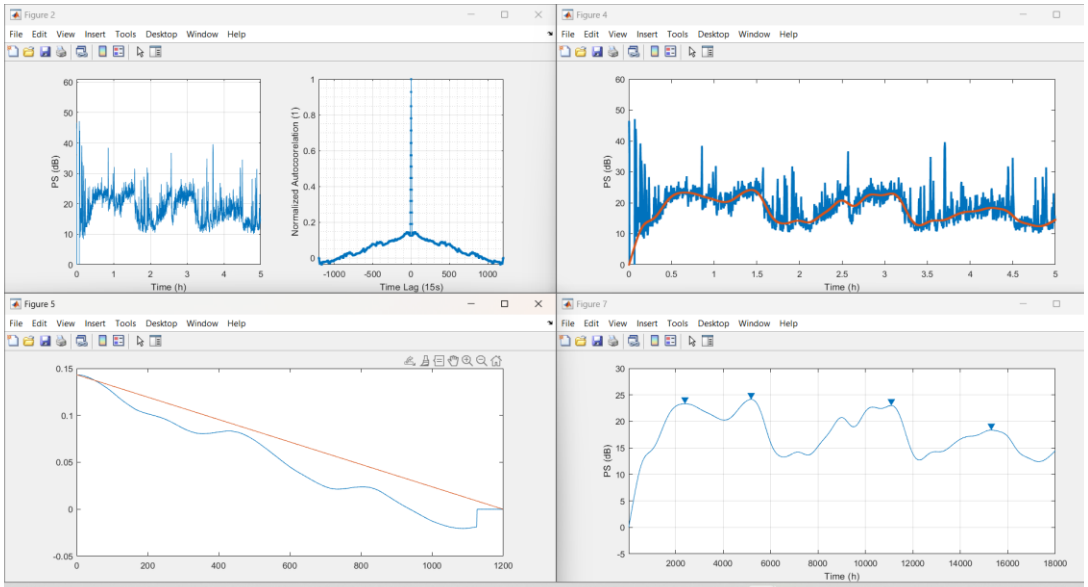
Participant 2-Night 4



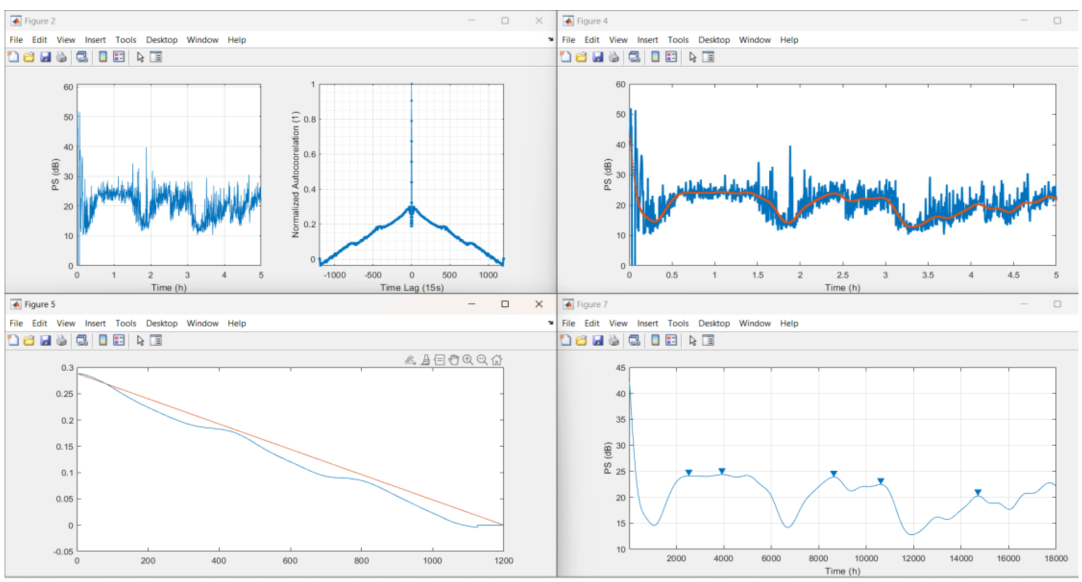
Participant 2-Night 7



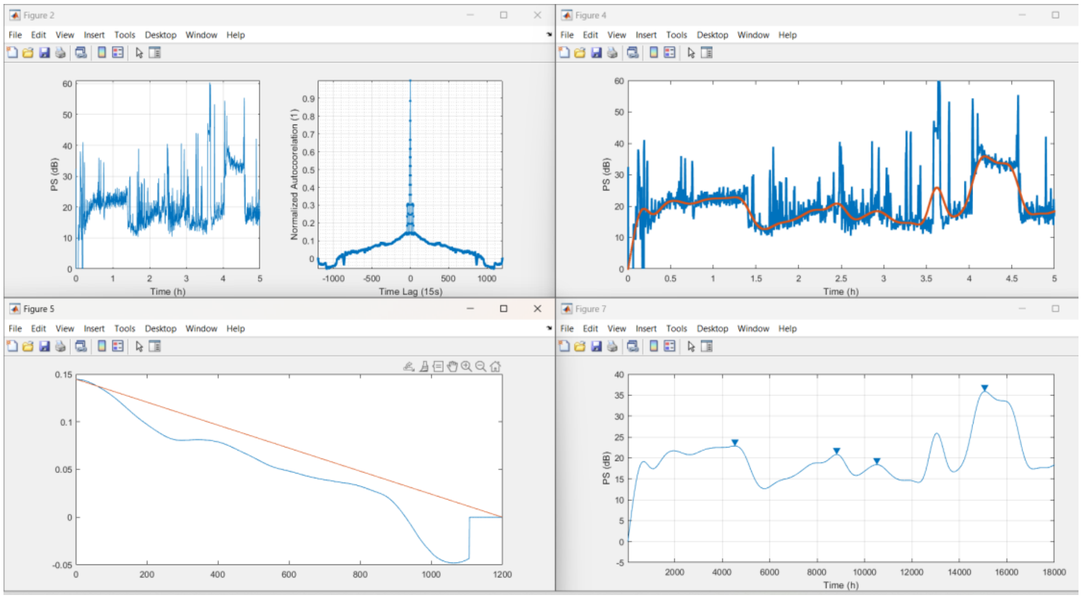
Participant 3-EXT Night



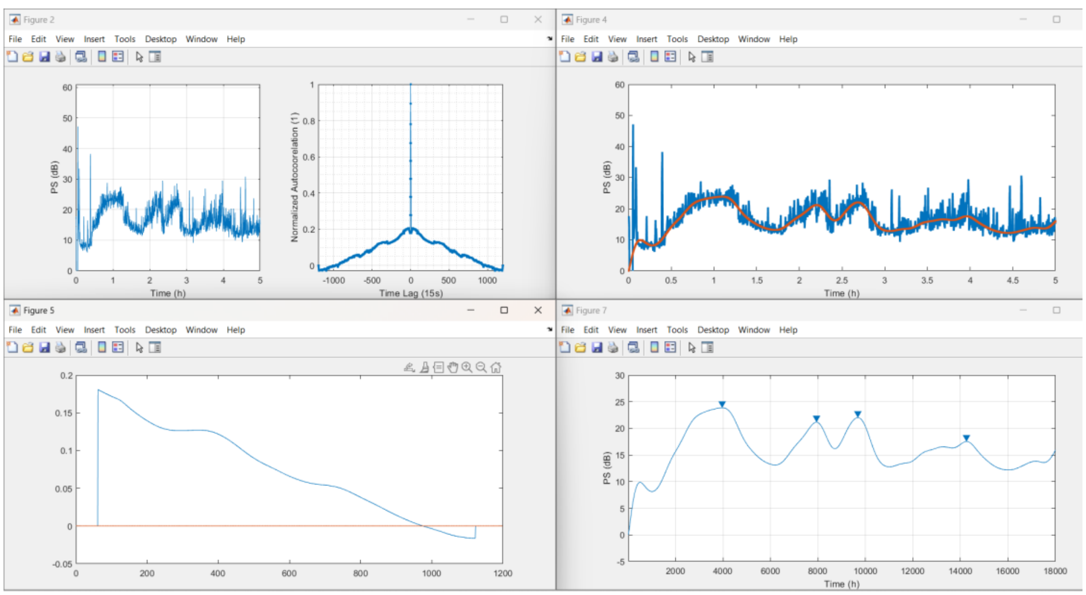
Participant 3-Night 1



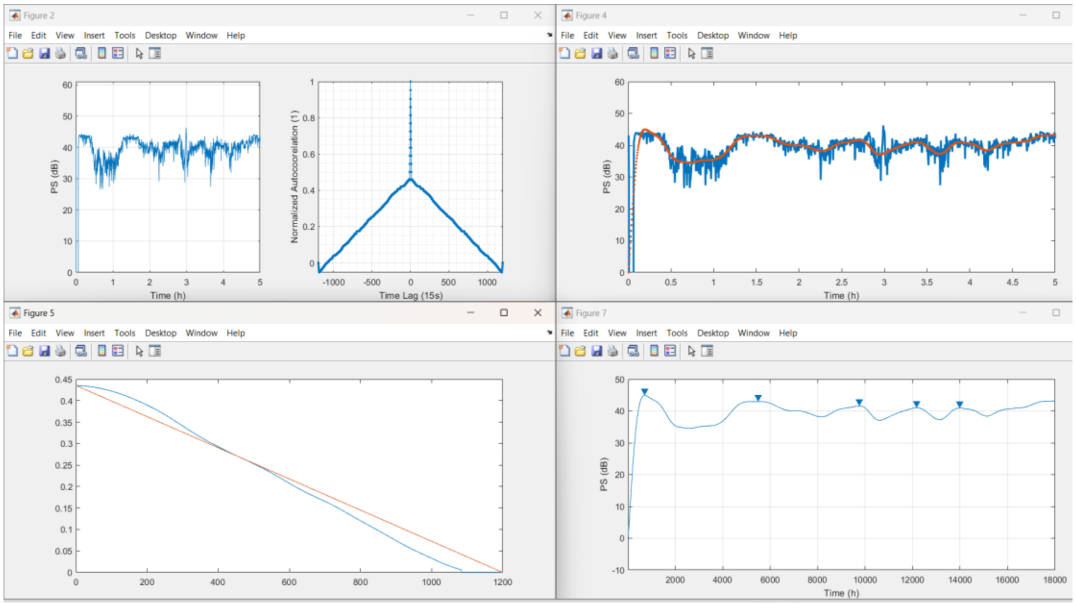
Participant 3-Night 4



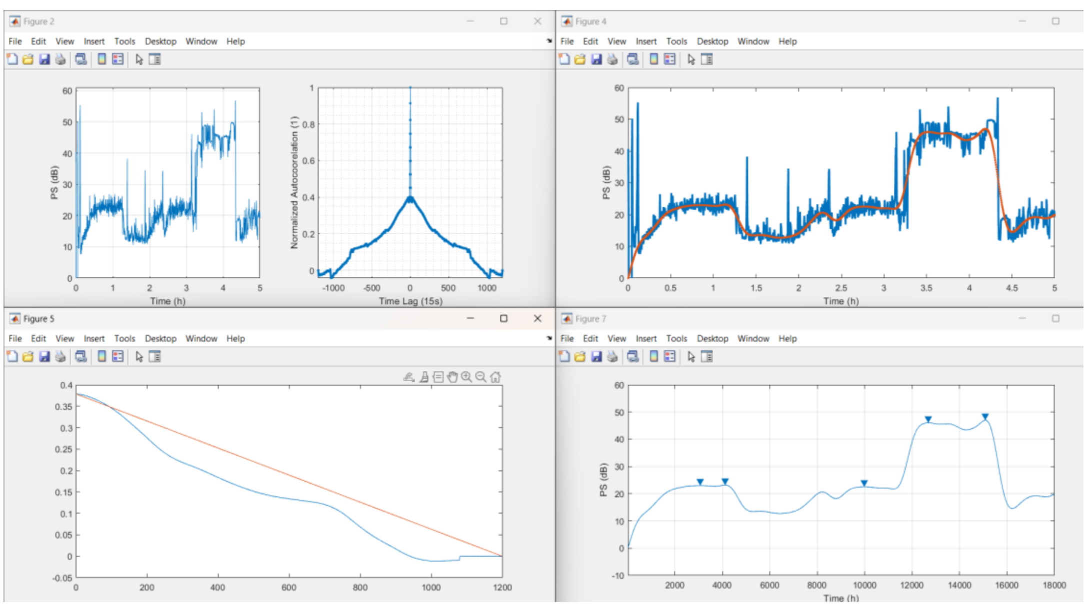
Participant 3-Night 7



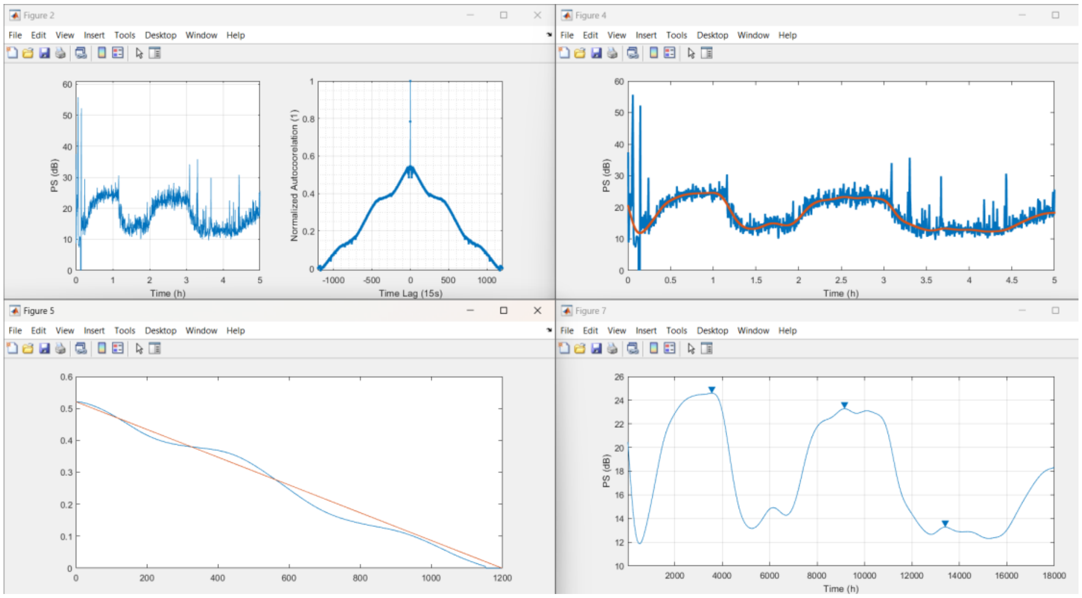
Participant 4-EXT Night



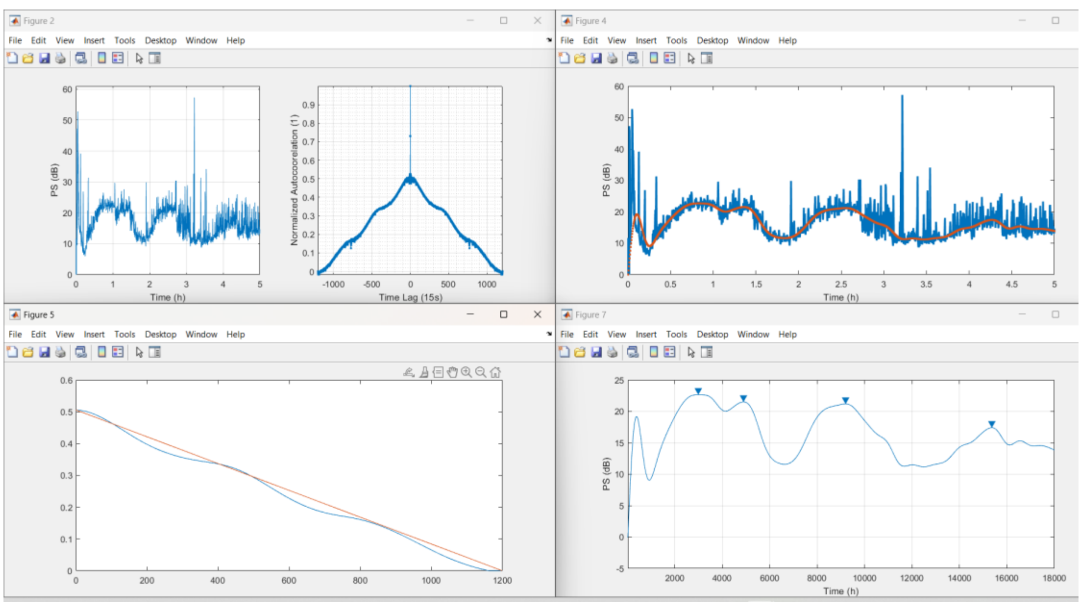
Participant 4-Night 1



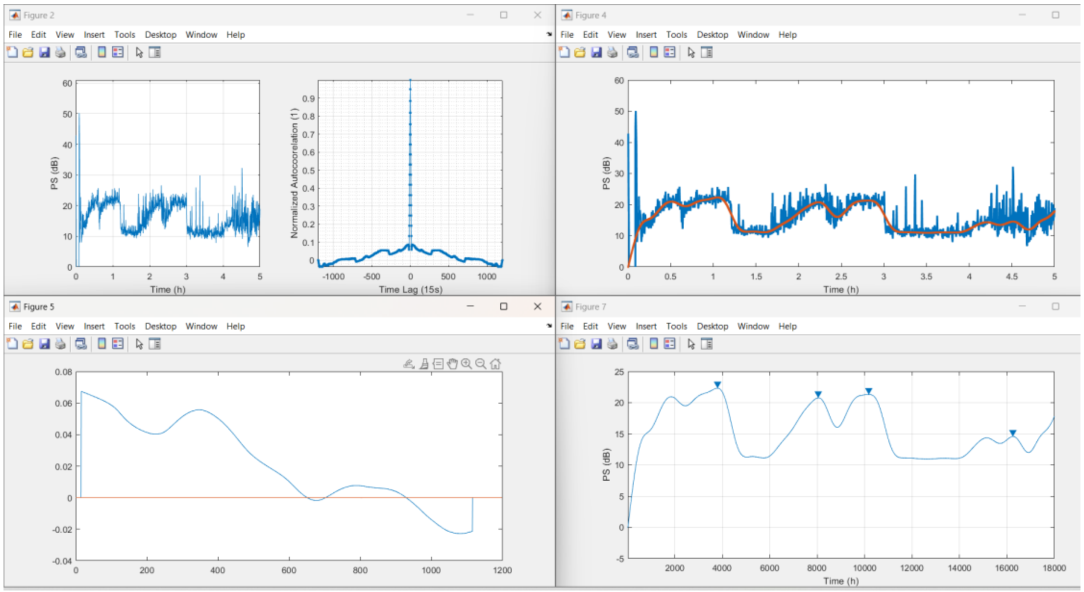
Participant 4-Night 4



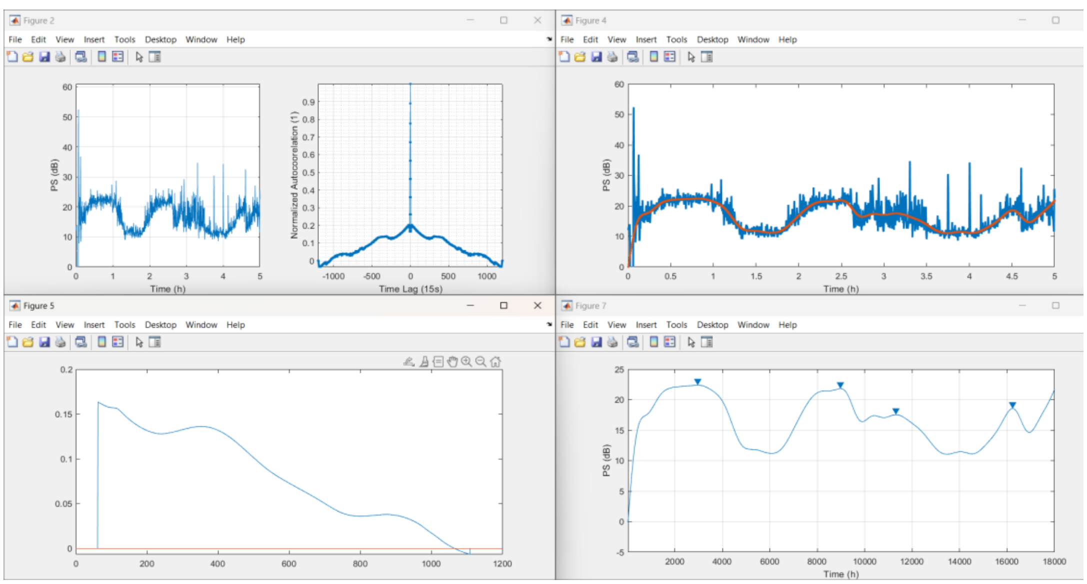
Participant 4-Night 7



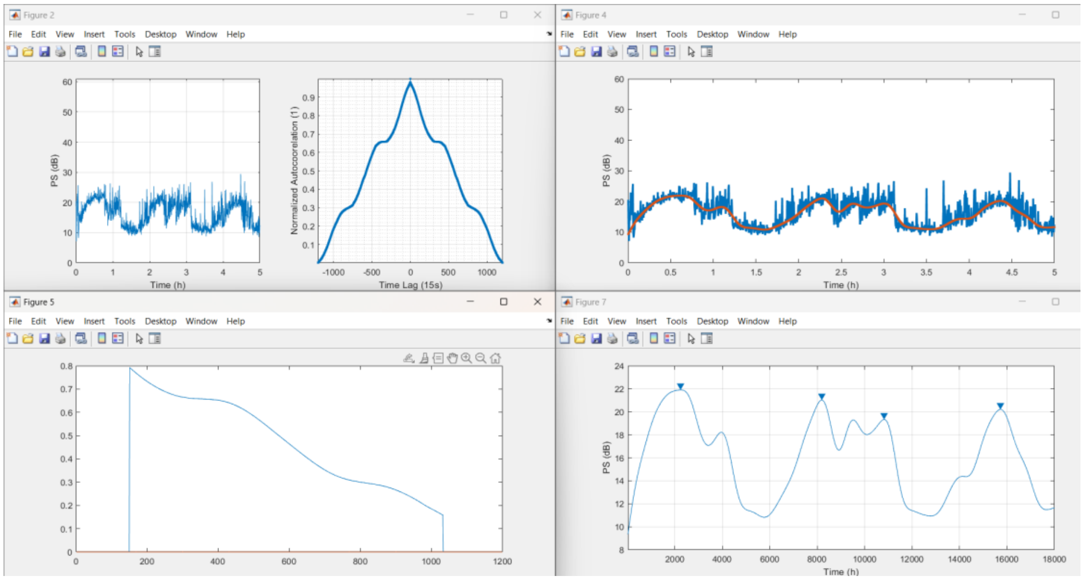
Participant 5-EXT Night



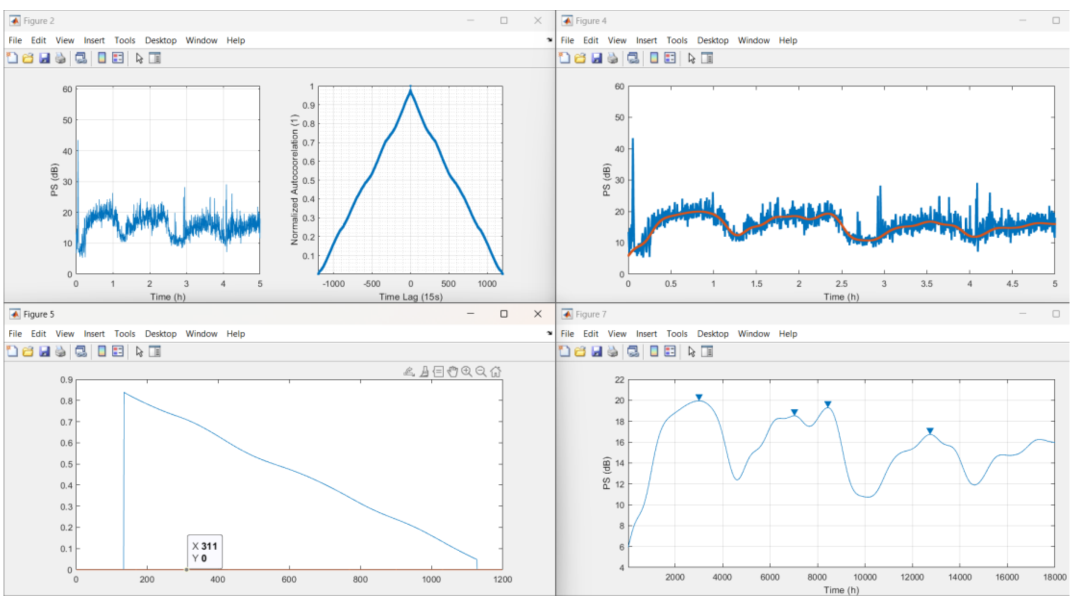
Participant 5-Night 1



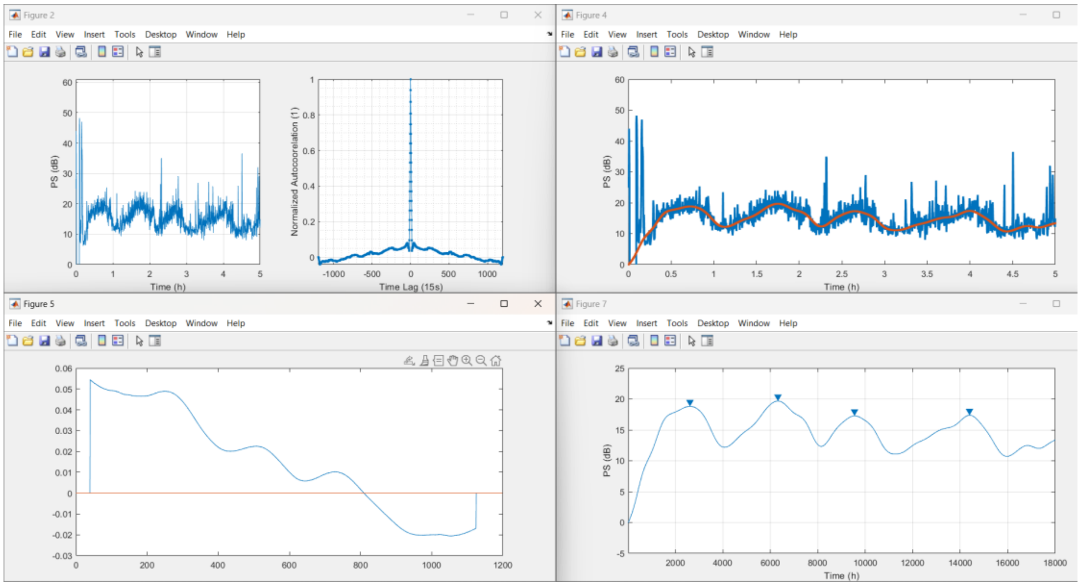
Participant 5-Night 4



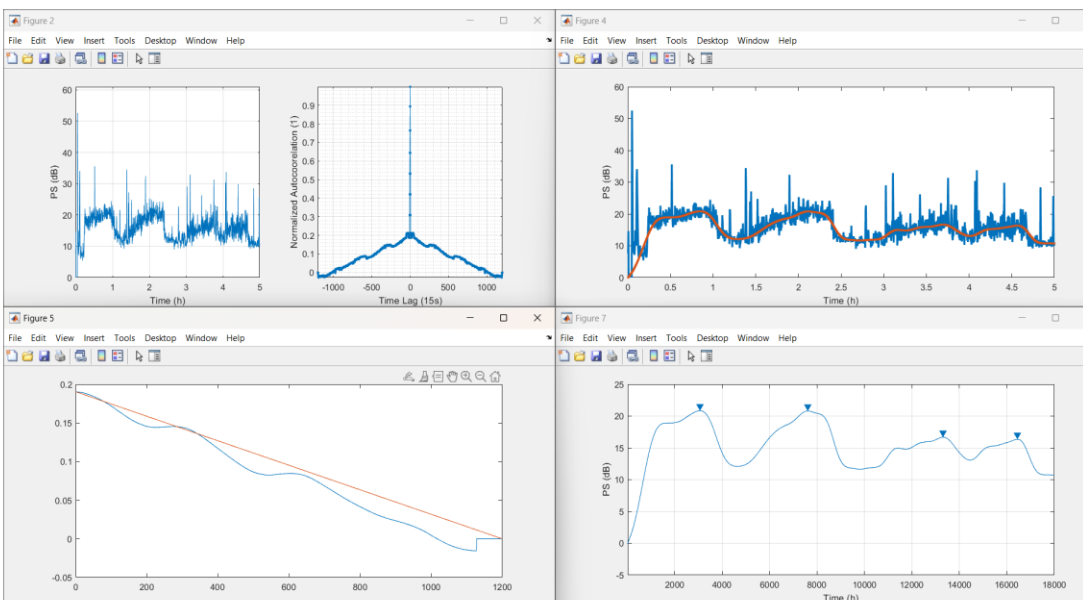
Participant 5-Night 7



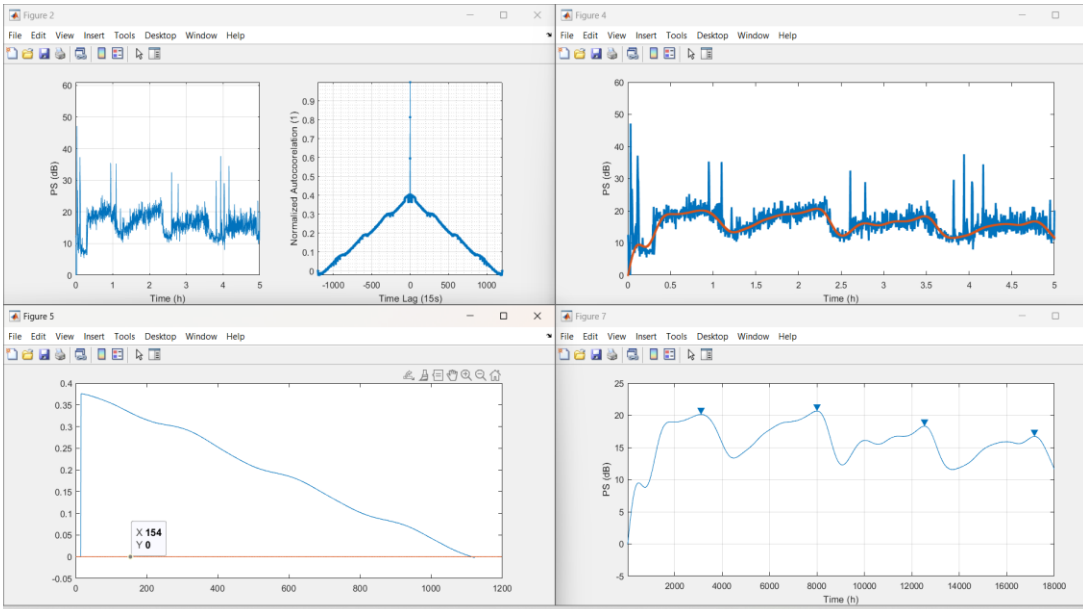
Participant 6-EXT Night



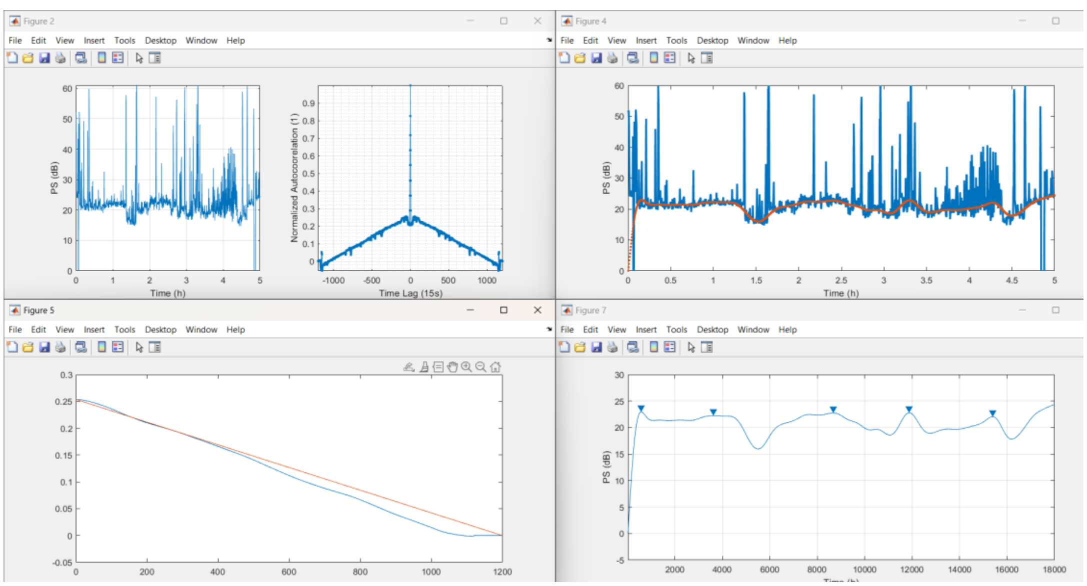
Participant 6-Night 1



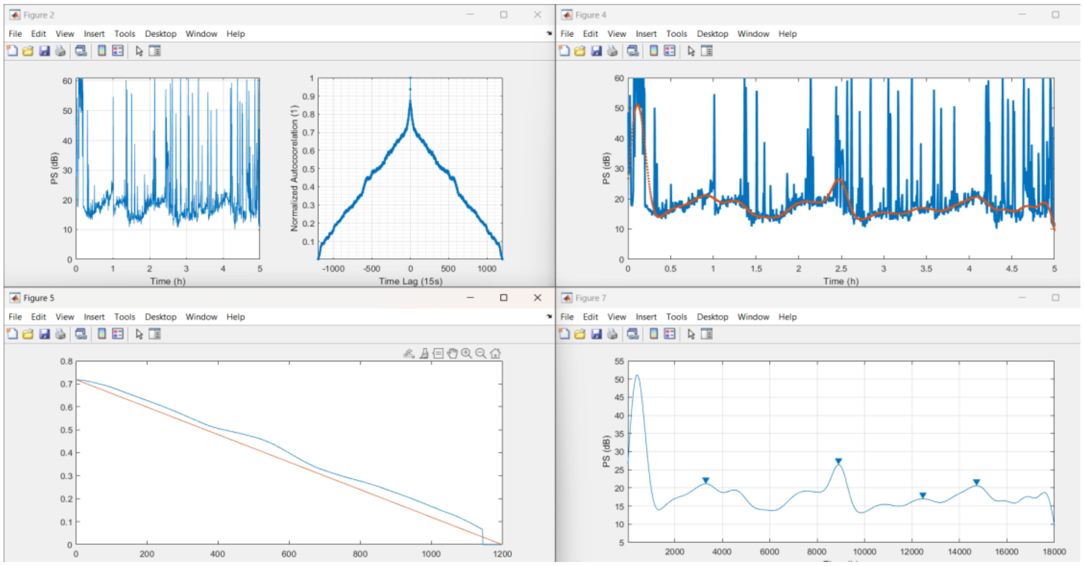
Participant 6-Night 4



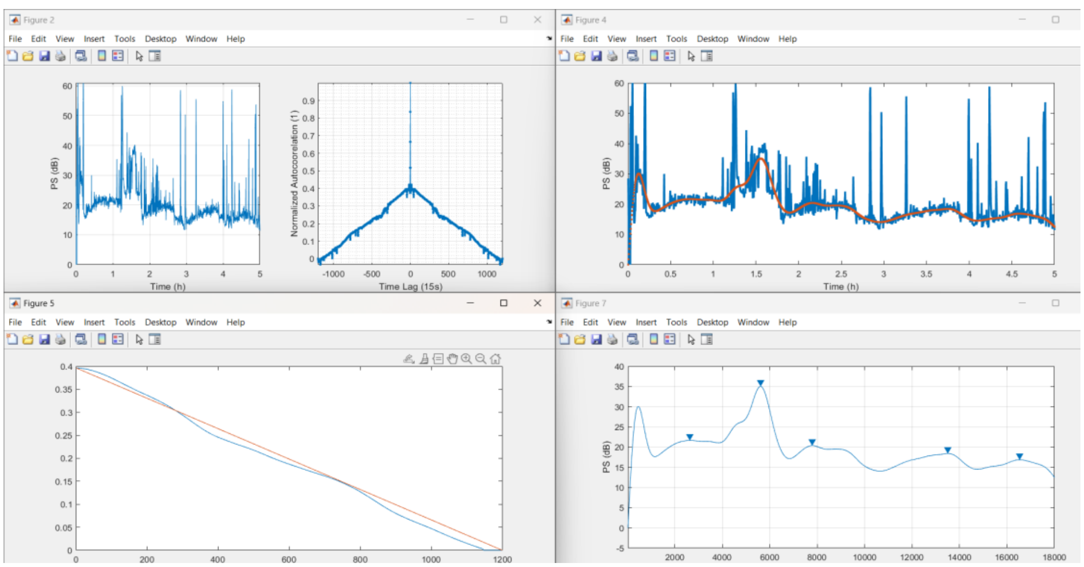
Participant 6-Night 7



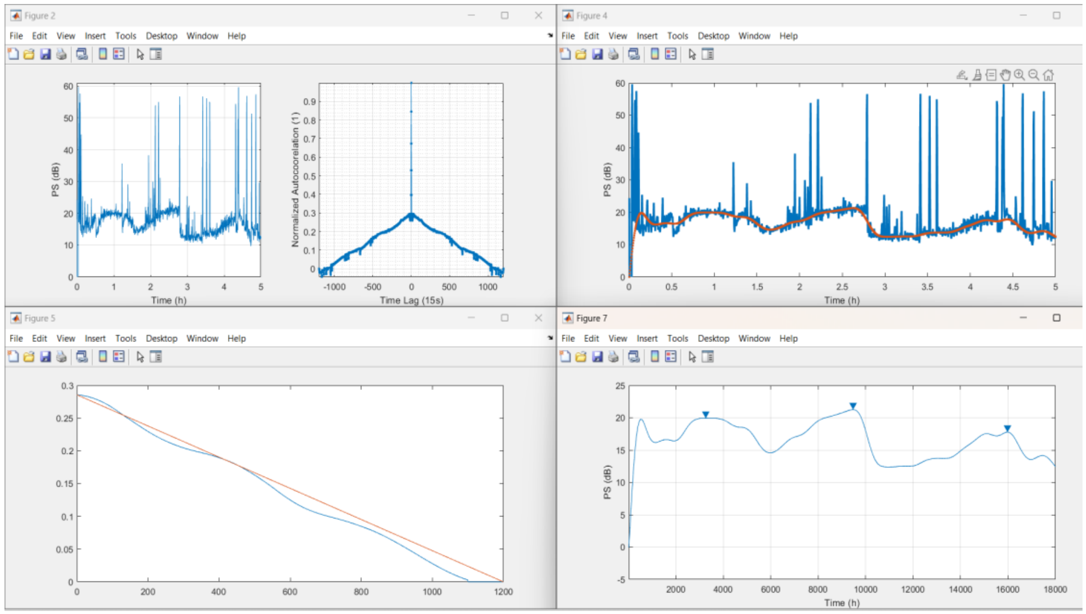
Participant 7-EXT Night



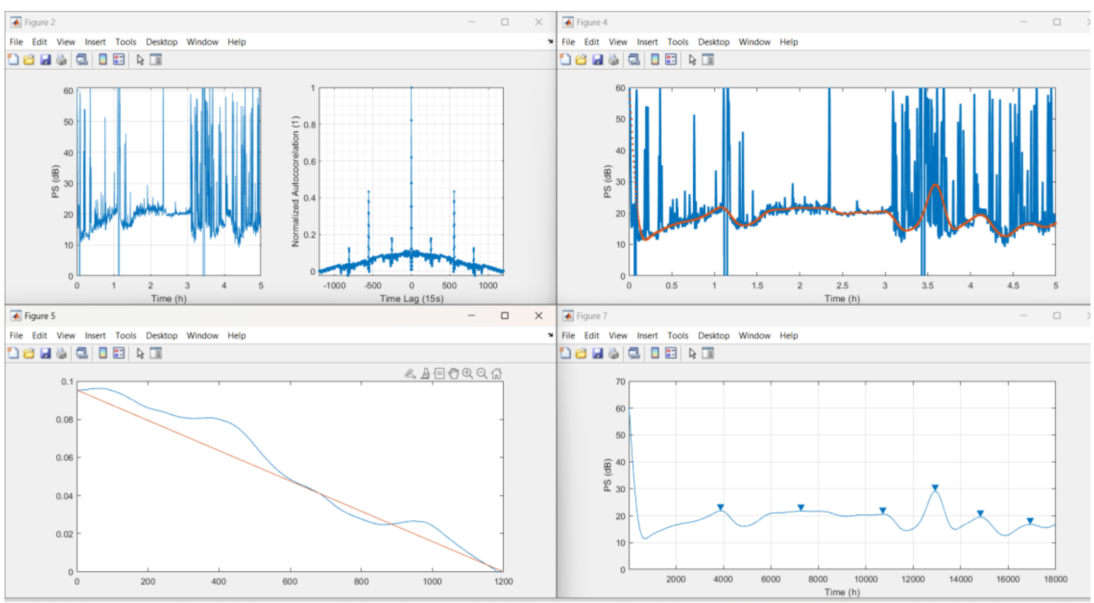
Participant 7-Night 1



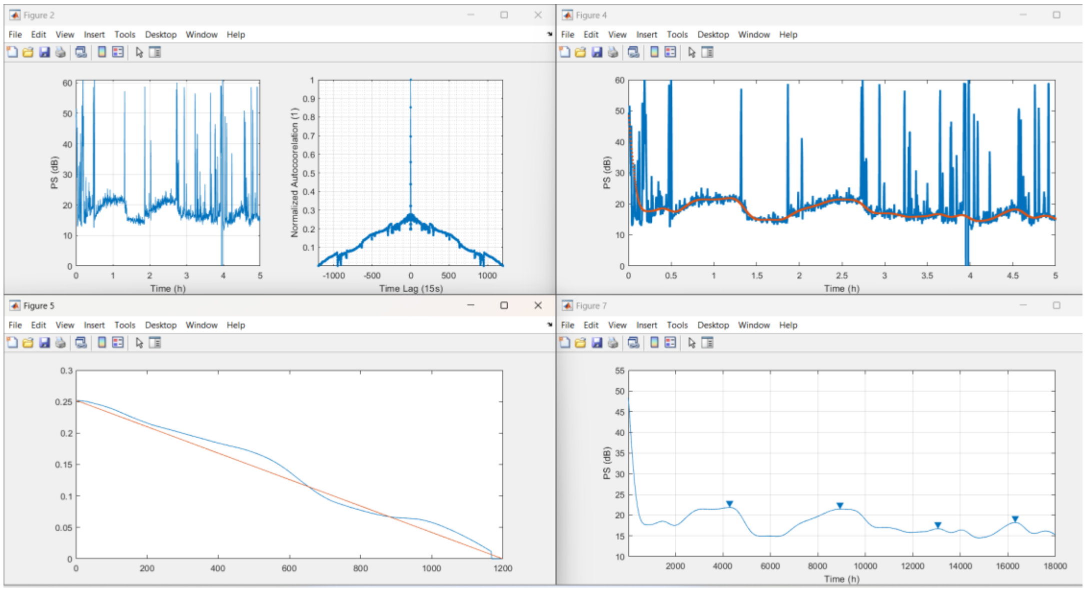
Participant 7-Night 4



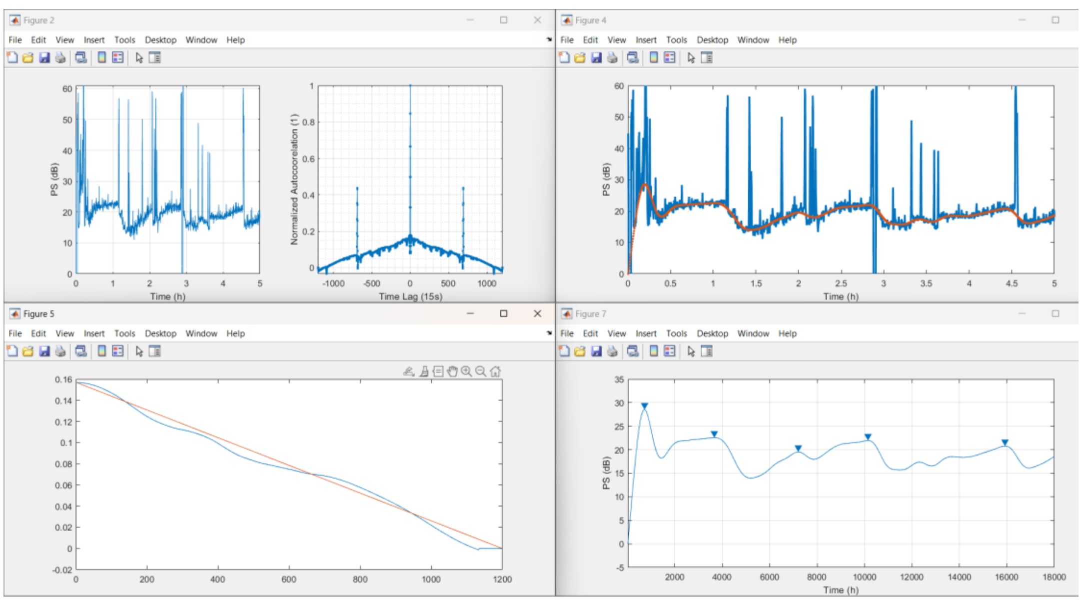
Participant 7-Night 7



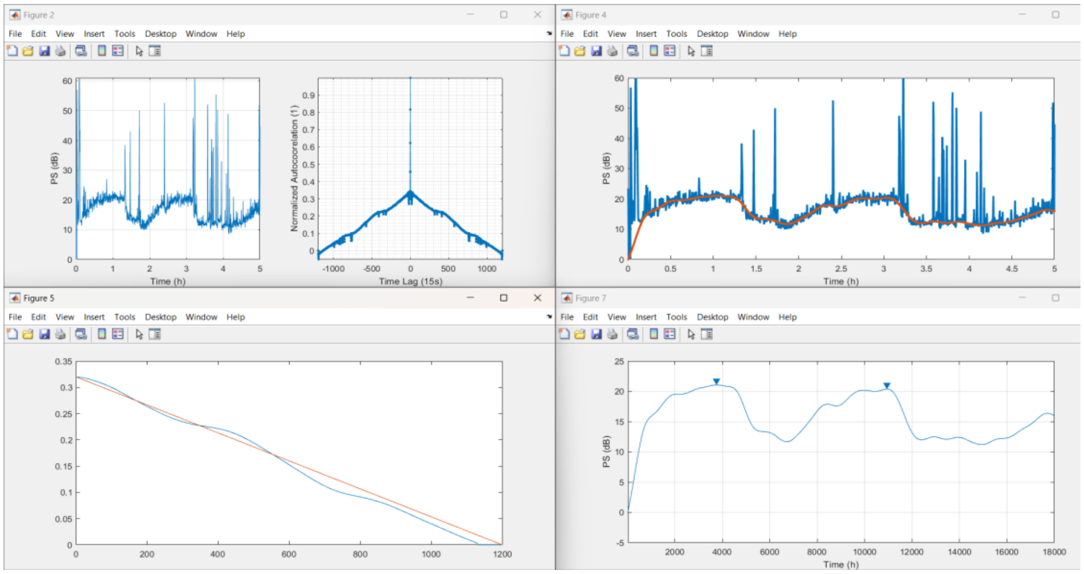
Participant 8-EXT Night



Participant 8-Night 1



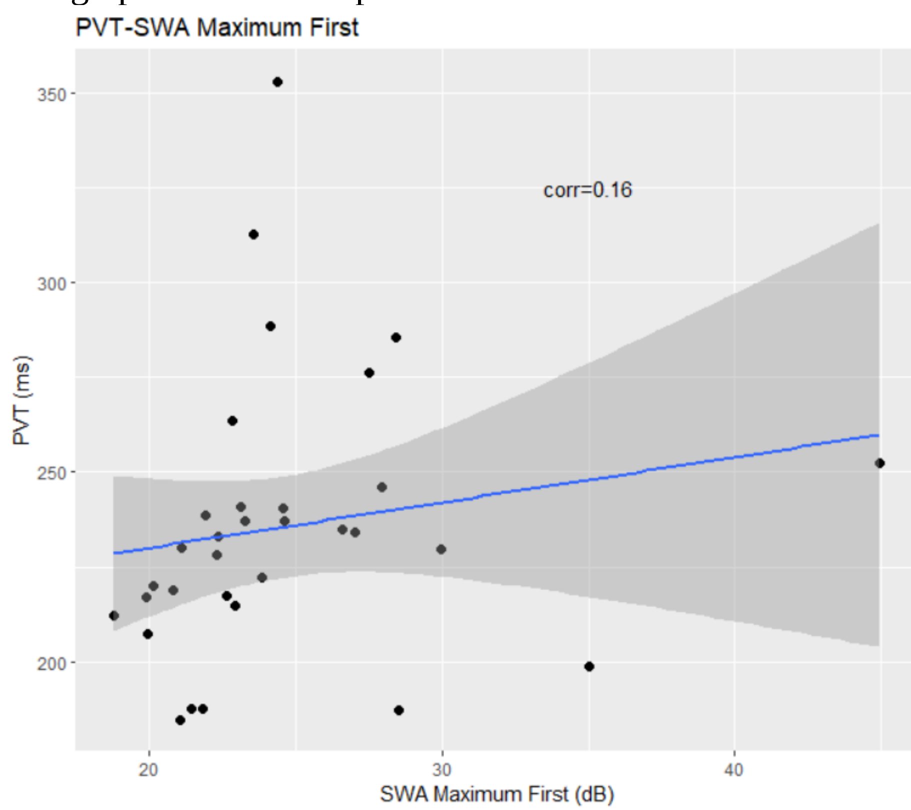
Participant 8-Night 4

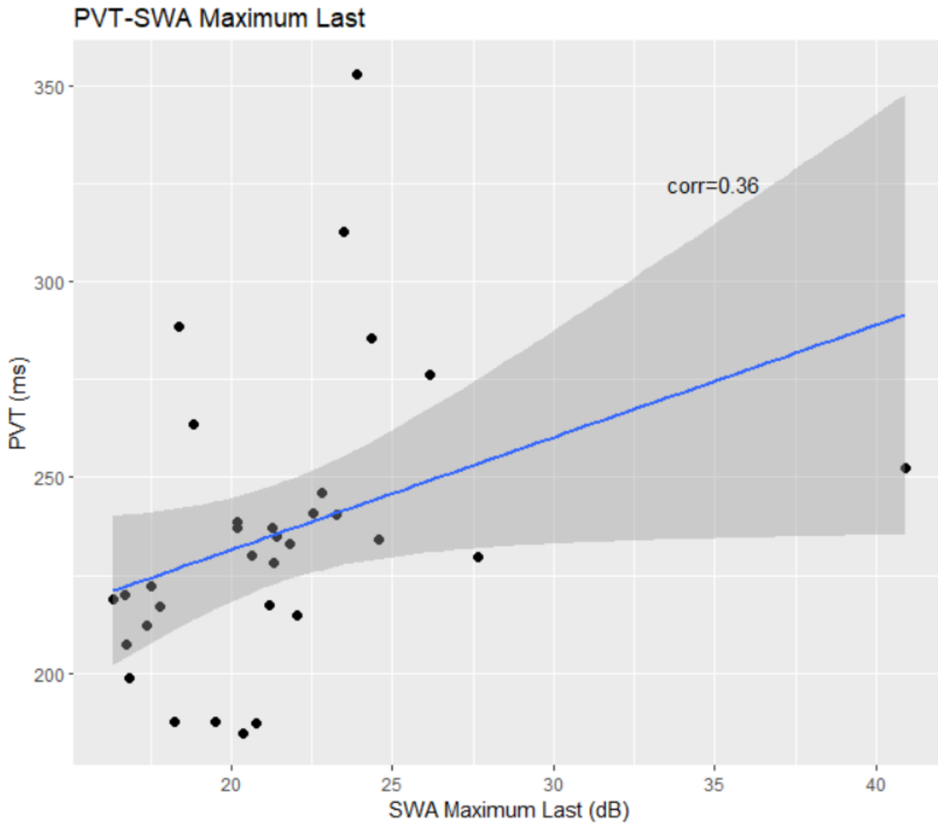


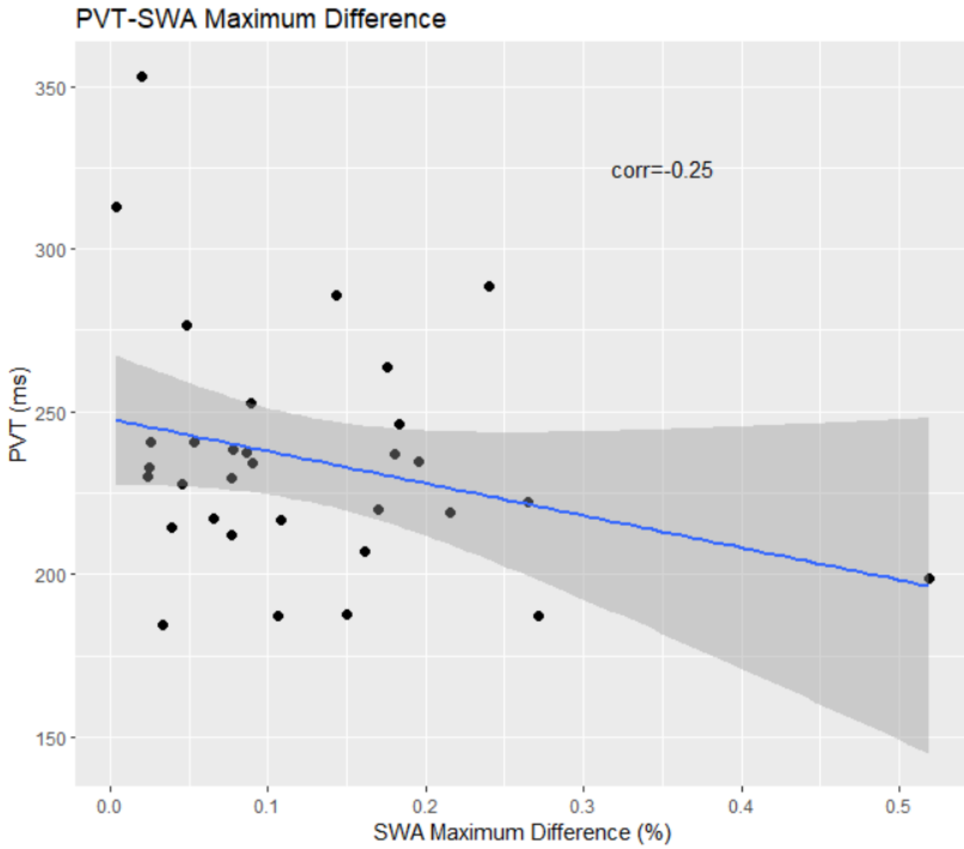
Participant 8-Night 7

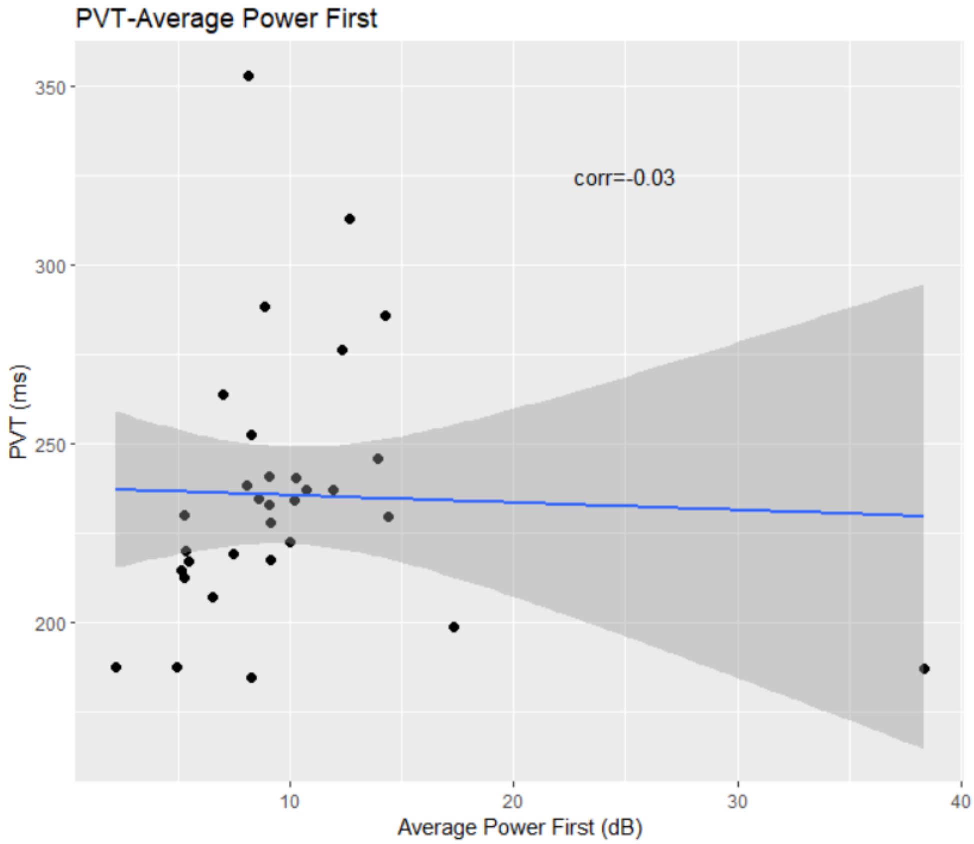
Appendix 3

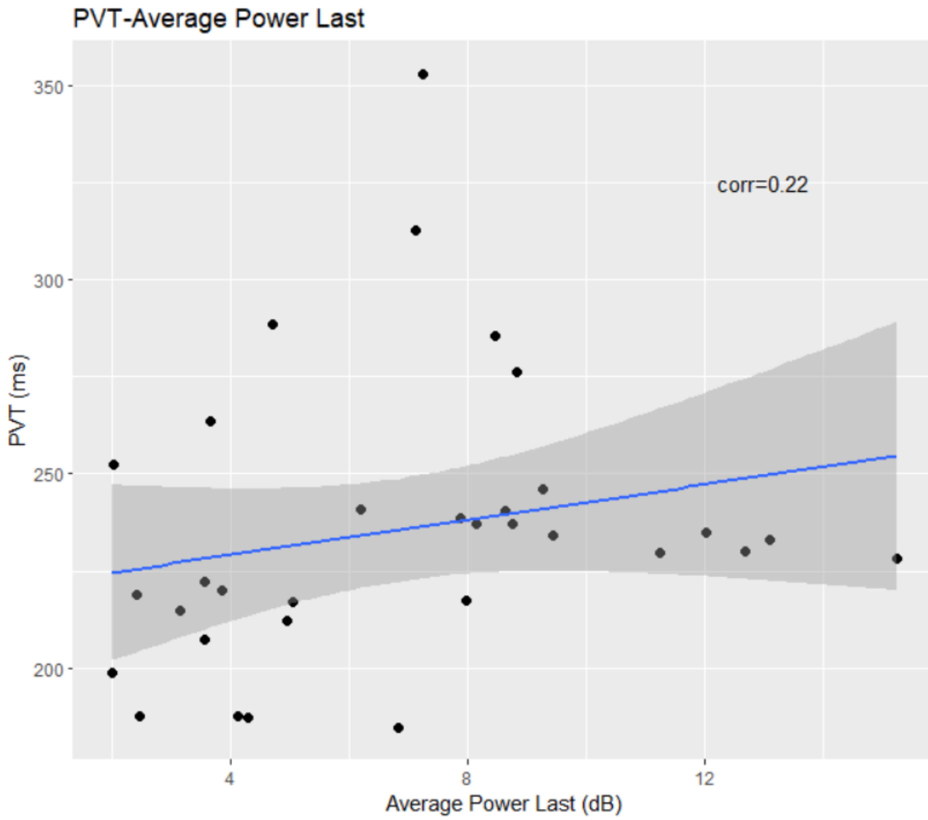
Plot graphs of each sleep measure with PVT

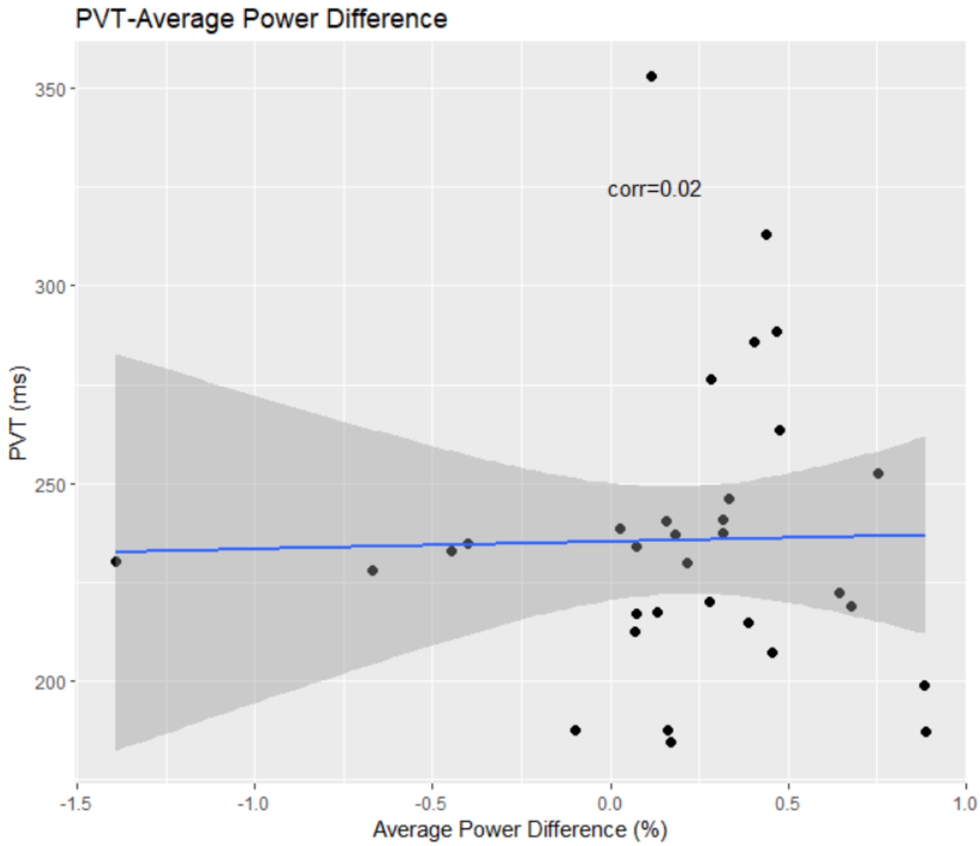



PVT-SWA Maximum Last
corr=0.36
PVT (ms)
350
300
250
200
20
25
30
35
40
SWA Maximum Last (dB)

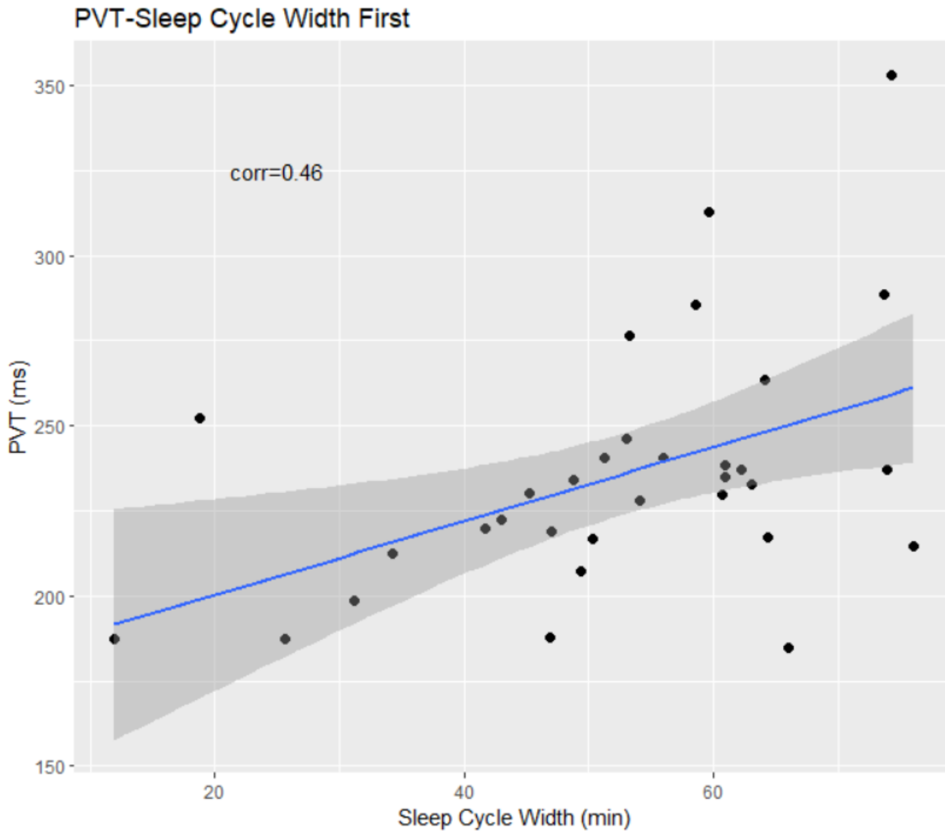

PVT-SWA Maximum Difference
corr=-0.25
PVT (ms)
350
300
250
200
150
0.0
0.1
0.2
0.3
0.4
0.5
SWA Maximum Difference (%)

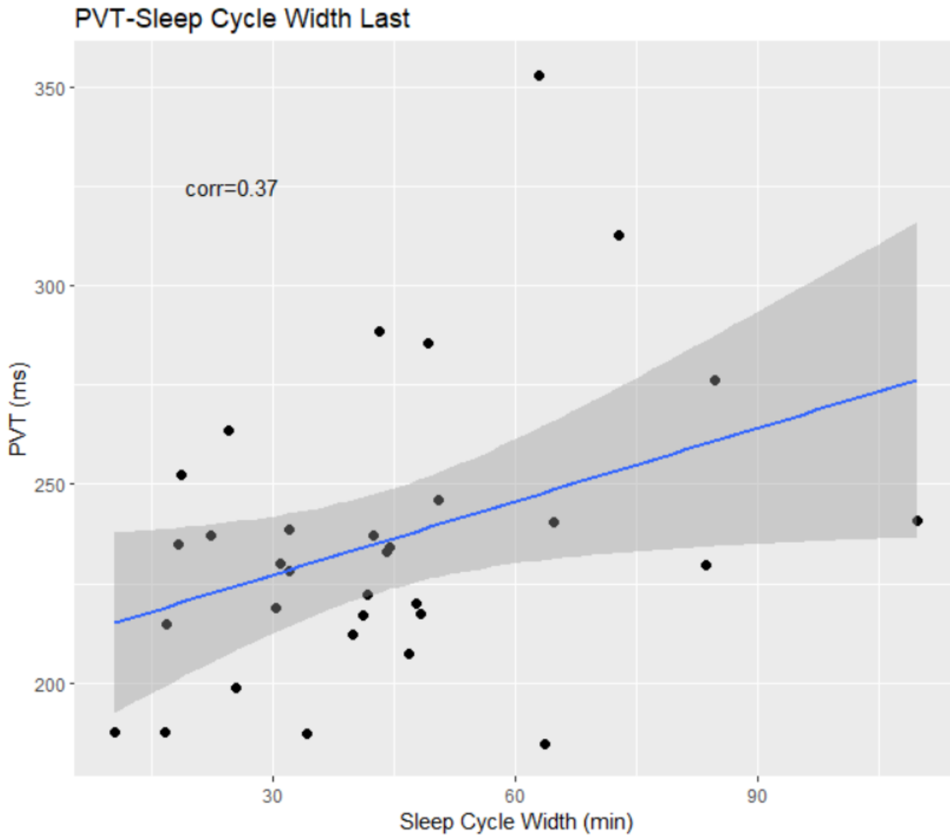

PVT-Average Power First
corr=-0.03
PVT (ms)
350
300
250
200
10
20
30
40
Average Power First (dB)

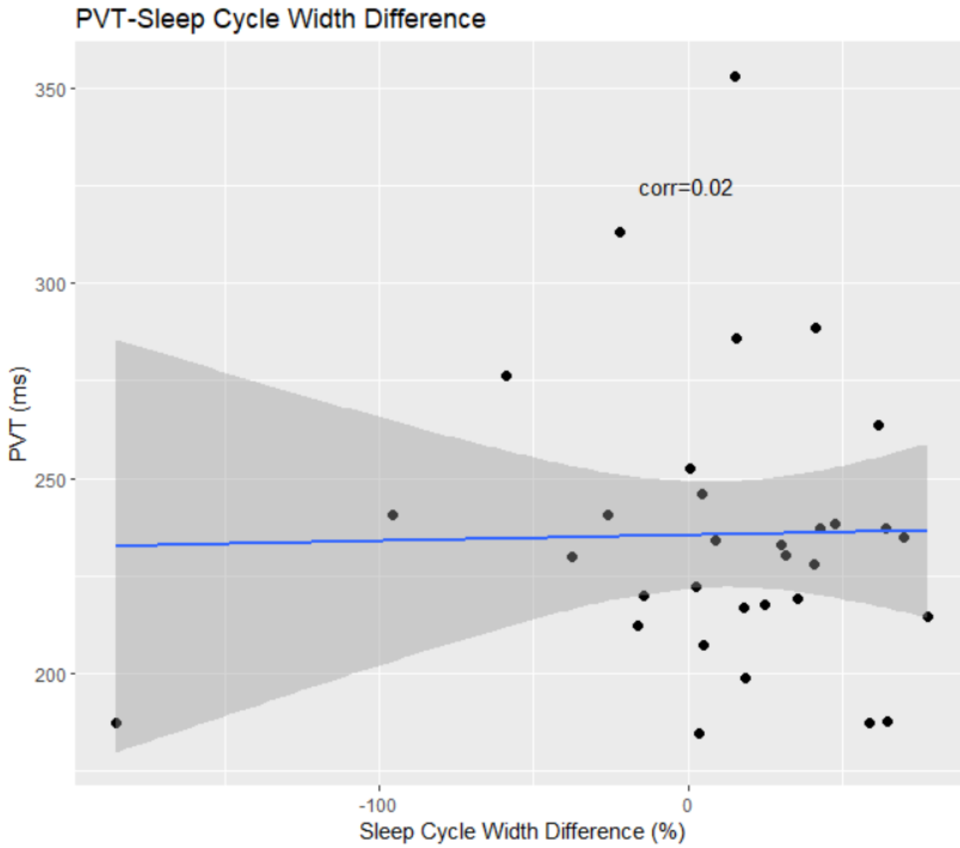

PVT-Average Power Last
corr=0.22
PVT (ms)
350
300
250
200
4
8
12
Average Power Last (dB)

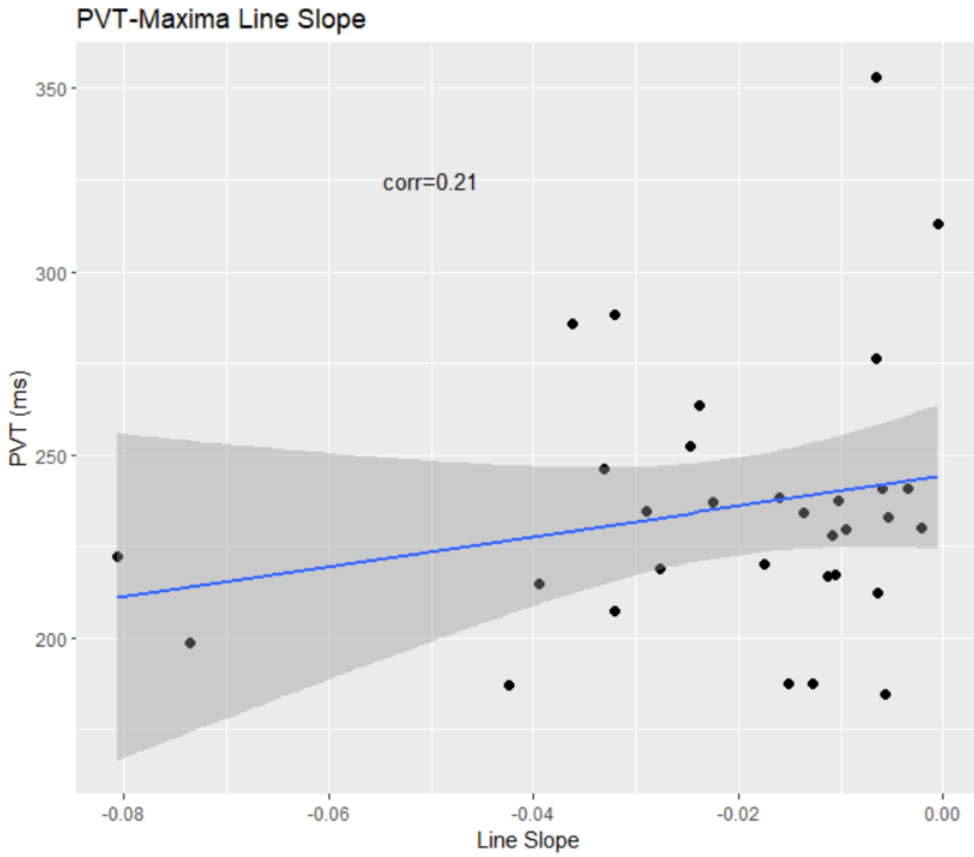


PVT-Average Power Difference
corr=0.02
PVT (ms)
350
300
250
200
-1.5
-1.0
-0.5
0.0
0.5
1.0
Average Power Difference (%)


PVT-Sleep Cycle Width First
corr=0.46
350
300
250
200
150
PVT (ms)
20
40
60
Sleep Cycle Width (min)


PVT-Sleep Cycle Width Last
corr=0.37
PVT (ms)
350
300
250
200
30
60
90
Sleep Cycle Width (min)


PVT-Sleep Cycle Width Difference
corr=0.02
350
300
250
200
PVT (ms)
-100
0
Sleep Cycle Width Difference (%)



Appendix 4

P-values for all two and three variable combinations regressed with response time.

		p-values		
1	2	1	2	1:2
MAX1	MAXL	0.47	**0.047**	0.17
MAX1	ΔMAX	0.56	0.39	0.68
MAX1	ENERGY1	**0.039**	**0.048**	**0.044**
MAX1	ENERGYL	0.24	0.24	0.33
MAX1	ΔENERGY	0.67	0.99	0.87
MAX1	WIDTHL	0.63	0.76	0.9
MAX1	WIDTH2	0.26	0.29	0.36
MAX1	ΔWIDTH	0.84	0.48	0.5

MAX1	SLOPE	0.45	0.97	0.72
MAXL	ΔMAX	0.22	0.64	0.51
MAXL	ENERGY1	0.61	0.97	0.95
MAXL	ENERGYL	0.12	0.55	0.67
MAXL	ΔENERGY	0.19	0.6	0.51
MAXL	WIDTHL	0.96	0.25	0.15
MAXL	WIDTH2	0.2	0.68	0.8
MAXL	ΔWIDTH	0.09	0.29	0.3
MAXL	SLOPE	0.5	0.87	0.99
ΔMAX	ENERGY1	0.2	0.56	0.45
ΔMAX	ENERGYL	0.39	0.78	0.77
ΔMAX	ΔENERGY	0.1	0.69	0.44
ΔMAX	WIDTHL	0.77	0.66	0.24
ΔMAX	WIDTH2	0.98	0.64	0.44
ΔMAX	ΔWIDTH	**0.036**	0.96	0.79
ΔMAX	SLOPE	0.14	0.91	0.41
ENERGY1	ENERGYL	0.64	0.38	0.58
ENERGY1	ΔENERGY	0.7	0.87	0.74
ENERGY1	WIDTHL	0.82	0.92	0.8
ENERGY1	WIDTH2	0.64	0.41	0.55
ENERGY1	ΔWIDTH	0.57	0.41	0.86
ENERGY1	SLOPE	0.94	0.5	0.86
ENERGYL	ΔENERGY	0.6	0.51	0.52
ENERGYL	WIDTHL	0.49	0.5	0.34
ENERGYL	WIDTH2	0.32	0.42	0.52
ENERGYL	ΔWIDTH	0.52	0.6	0.62
ENERGYL	SLOPE	0.92	0.24	0.53
ΔENERGY	WIDTHL	0.33	0.23	0.21
ΔENERGY	WIDTH2	0.9	0.42	0.68
ΔENERGY	ΔWIDTH	0.28	0.68	0.89
ΔENERGY	SLOPE	0.86	0.44	0.93
WIDTHL	WIDTH2	0.24	0.2	0.14
WIDTHL	ΔWIDTH	0.45	0.91	0.58
WIDTHL	SLOPE	0.58	0.92	0.55
WIDTH2	ΔWIDTH	0.23	0.71	0.44
WIDTH2	SLOPE	0.97	0.47	0.79
ΔWIDTH	SLOPE	0.69	0.09	0.59

Bibliography

Achermann, P., & Borbély, A. A. (1990). Simulation of human sleep: ultradian dynamics of electroencephalographic slow-wave activity. *Journal of Biological Rhythms*, *5*(2), 141-157.

Anderson, C., & Platten, C. R. (2011). Sleep deprivation lowers inhibition and enhances impulsivity to negative stimuli. *Behavioural Brain Research*, *217*(2), 463-466.

Ayas, N. T., Barger, L. K., Cade, B. E., Hashimoto, D. M., Rosner, B., Cronin, J. W., ... & Czeisler, C. A. (2006). Extended work duration and the risk of self-reported percutaneous injuries in interns. *JAMA*, *296*(9), 1055-1062.

Baddeley, A. (2010). Working memory. *Current Biology*, *20*(4), R136-R140.

Barenberg, J., Berse, T., & Dutke, S. (2011). Executive functions in learning processes: do they benefit from physical activity?. *Educational Research Review*, *6*(3), 208-222.

Basner, M., & Dinges, D. F. (2011). Maximizing sensitivity of the psychomotor vigilance test (PVT) to sleep loss. *Sleep*, *34(5)*, 581-591.

Baumeister, R. F. (2014). Self-regulation, ego depletion, and inhibition. *Neuropsychologia*, *65*, 313-319.

Belenky, G. (1997, January). Sleep, sleep deprivation, and human performance in continuous operations. In Joint Services Conference on Professional Ethics-JSCOPE (Vol. 97).

Belenky, G., Wesensten, N. J., Thorne, D. R., Thomas, M. L., Sing, H. C., Redmond, D. P., ... & Balkin, T. J. (2003). Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: A sleep dose-response study. *Journal of Sleep Research*, *12*(1), 1-12.

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

Cajochen, C., Foy, R., & Dijk, D. J. (1999). Frontal predominance of a relative increase in sleep delta and theta EEG activity after sleep loss in humans. *Sleep Research Online*, *2*(3), 65-69.

Caldwell, J. A. (2005). Fatigue in aviation. *Travel Medicine and Infectious Disease*, *3*(2), 85-96.

Centers for Disease Control and Prevention (CDC). (2011). Effect of short sleep duration on daily activities--United States, 2005-2008. *MMWR. Morbidity and Mortality Weekly Report*, *60*(8), 239-242.

Chaisilprungraung, T., Stekl, E. K., Thomas, C. L., Blanchard, M. E., Hughes, J. D., Balkin, T. J., & Doty, T. J. (2022). Quantifying the effects of sleep loss: relative effect sizes of the psychomotor vigilance test, multiple sleep latency test, and maintenance of wakefulness test. *Sleep Advances*, *3*(1), zpac034.

Collins, A., & Koechlin, E. (2012). Reasoning, learning, and creativity: frontal lobe function and human decision-making. *PLoS Biology*, *10*(3), e1001293.

Davis, C. L., Tomporowski, P. D., Boyle, C. A., Waller, J. L., Miller, P. H., Naglieri, J. A., & Gregoski, M. (2007). Effects of aerobic exercise on overweight children's cognitive functioning: a randomized controlled trial. *Research Quarterly for Exercise and Sport*, *78*(5), 510-519.

Dawson, D., & Reid, K. (1997). Fatigue, alcohol and performance impairment. *Nature*, *388*(6639), 235-235.

Dayarian, N., & Khadem, A. (2024). A hybrid boundary element-finite element approach for solving the EEG forward problem in brain modeling. *Frontiers in Systems Neuroscience*, *18*, 1327674.

della Monica, C., Johnsen, S., Atzori, G., Groeger, J. A., & Dijk, D. J. (2018). Rapid eye movement sleep, sleep continuity and slow wave sleep as predictors of cognition, mood, and subjective sleep quality in healthy men and women, aged 20–84 years. *Frontiers in Psychiatry*, *9*, 255.

Diamond, A. (2013). Executive functions. *Annual Review of Psychology*, *64*, 135-168.

Diep, C., Ftouni, S., Manousakis, J. E., Nicholas, C. L., Drummond, S. P., & Anderson, C. (2020). Acoustic slow wave sleep enhancement via a novel, automated device improves executive function in middle-aged men. *Sleep*, *43*(1), zsz197.

Drummond, S. P., Paulus, M. P., & Tapert, S. F. (2006). Effects of two nights sleep deprivation and two nights recovery sleep on response inhibition. *Journal of Sleep Research*, *15*(3), 261-265.

Edwards, L. M., Murray, A. J., Holloway, C. J., Carter, E. E., Kemp, G. J., Codreanu, I., ... & Clarke, K. (2011). Short-term consumption of a high-fat diet impairs whole-body efficiency and cognitive function in sedentary men. *The FASEB Journal*, *25*(3), 1088-1096.

Eggermont, L. H., Milberg, W. P., Lipsitz, L. A., Scherder, E. J., & Leveille, S. G. (2009). Physical activity and executive function in aging: the MOBILIZE Boston Study. *Journal of the American Geriatrics Society*, *57*(10), 1750-1756.

Eling, P., Derckx, K., & Maes, R. (2008). On the historical and conceptual background of the Wisconsin Card Sorting Test. *Brain and Cognition*, *67*(3), 247-253.

Esterman, M., & Rothlein, D. (2019). Models of sustained attention. *Current Opinion in Psychology*, *29*, 174-180.

Fabbri, M., Beracci, A., Martoni, M., Meneo, D., Tonetti, L., & Natale, V. (2021). Measuring subjective sleep quality: a review. *International Journal of Environmental Research and Public Health*, *18*(3), 1082.

Fabrigoule, C., Rouch, I., Taberly, A., Letenneur, L., Commenges, D., Mazaux, J. M., ... & Dartigues, J. F. (1998). Cognitive process in preclinical phase of dementia. *Brain: A Journal of Neurology*, *121*(1), 135-141.

Fell, J. C., & Voas, R. B. (2014). The effectiveness of a 0.05 blood alcohol concentration (BAC) limit for driving in the United States. *Addiction*, *109*(6), 869-874.

Giller, F., & Beste, C. (2019). Effects of aging on sequential cognitive flexibility are associated with fronto-parietal processing deficits. *Brain Structure and Function*, *224*(7), 2343-2355.

Gottlieb, D. J., Redline, S., Nieto, F. J., Baldwin, C. M., Newman, A. B., Resnick, H. E., & Punjabi, N. M. (2006). Association of usual sleep duration with hypertension: The Sleep Heart Health Study. *Sleep*, *29*(8), 1009-1014.

Greene, R. W., Bjorness, T. E., & Suzuki, A. (2017). The adenosine-mediated, neuronal-glial, homeostatic sleep response. *Current Opinion in Neurobiology*, *44*, 236-242.

Haack, M., & Mullington, J. M. (2005). Sustained sleep restriction reduces emotional and physical well-being. *Pain*, *119*(1-3), 56-64.

Harvey, A. G., Murray, G., Chandler, R. A., & Soehner, A. (2011). Sleep disturbance as transdiagnostic: Consideration of neurobiological mechanisms. *Clinical Psychology Review*, *31*(2), 225–235. https://doi.org/10.1016/j.cpr.2010.04.003

Hillman, C. H., Erickson, K. I., & Kramer, A. F. (2008). Be smart, exercise your heart: exercise effects on brain and cognition. *Nature Reviews Neuroscience*, *9*(1), 58-65.

Hirshkowitz, M., Whiton, K., Albert, S. M., Alessi, C., Bruni, O., DonCarlos, L., ... & Hillard, P. J. A. (2015). National Sleep Foundation's sleep time duration recommendations: Methodology and results summary. *Sleep Health*, *1*(1), 40-43.

Horne, J. A., & Reyner, L. A. (1995). Sleep related vehicle accidents. *BMJ*, *310*(6979), 565-567.

Huber, R., Felice Ghilardi, M., Massimini, M., & Tononi, G. (2004). Local sleep and learning. *Nature*, *430*(6995), 78-81.

Iber, C., Ancoli-Isreal, S., Chesson, A., & Quan, S. (2007). The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology and Technical Specification, 1st edition: Westchester, Illinois: American Academy of Sleep Medicine.

Kalmijn, S., Van Boxtel, M. P. J., Ocke, M., Verschuren, W. M. M., Kromhout, D., & Launer, L. J. (2004). Dietary intake of fatty acids and fish in relation to cognitive performance at middle age. *Neurology*, *62*(2), 275-280.

Kamijo, K., & Takeda, Y. (2010). Regular physical activity improves executive function during task switching in young adults. *International Journal of Psychophysiology*, *75*(3), 304-311.

Kramer, A. F., Hahn, S., Cohen, N. J., Banich, M. T., McAuley, E., Harrison, C. R., ... & Colcombe, A. (1999). Ageing, fitness and neurocognitive function. *Nature*, *400*(6743), 418-419.

Lamond, N., & Dawson, D. (1999). Quantifying the performance impairment associated with fatigue. *Journal of Sleep Research*, *8*(4), 255-262.

Lara, T., Madrid, J. A., & Correa, Á. (2014). The vigilance decrement in executive function is attenuated when individual chronotypes perform at their optimal time of day. *PloS One*, *9*(2), e88820.

Lentino, C. V., Purvis, D. L., Murphy, K. J., & Deuster, P. A. (2013). Sleep as a component of the performance triad: The importance of sleep in a military population. *US Army Medical Department Journal*, *4*, 98-108.

Lim, S. (2018). Eating a balanced diet: a healthy life through a balanced diet in the age of longevity. *Journal of Obesity & Metabolic Syndrome*, *27*(1), 39.

Määttä, S., Landsness, E., Sarasso, S., Ferrarelli, F., Ferreri, F., Ghilardi, M. F., & Tononi, G. (2010). The effects of morning training on night sleep: a behavioral and EEG study. *Brain Research Bulletin*, *82*(1-2), 118-123.

Macera, C. A., Aralis, H. J., Rauh, M. J., & MacGregor, A. J. (2013). Do sleep problems mediate the relationship between traumatic brain injury and development of mental health symptoms after deployment?. *Sleep*, *36*(1), 83-90.

Mah, C. D., Mah, K. E., Kezirian, E. J., & Dement, W. C. (2011). The effects of sleep extension on the athletic performance of collegiate basketball players. *Sleep*, *34*(7), 943-950.

Mahajan, K., & Velaga, N. R. (2020). Effects of partial sleep deprivation on braking response of drivers in hazard scenarios. *Accident Analysis & Prevention*, *142*, 105545.

Maskevich, S., Jumabhoy, R., Dao, P. D., Stout, J. C., & Drummond, S. (2017). Pilot validation of ambulatory activity monitors for sleep measurement in Huntington’s disease gene carriers. *Journal of Huntington's Disease*, *6*(3), 249-253.

Miles, S., Howlett, C. A., Berryman, C., Nedeljkovic, M., Moseley, G. L., & Phillipou, A. (2021). Considerations for using the Wisconsin Card Sorting Test to assess cognitive flexibility. *Behavior Research Methods*, *53*(5), 2083-2091.

Miller, N. L., Shattuck, L. G., & Matsangas, P. (2011). Sleep and fatigue issues in continuous operations: A survey of US Army officers. *Behavioral Sleep Medicine, 9(1),* 53-65.

Miyake, A., Friedman, N. P., Emerson, M. J., Witzki, A. H., Howerter, A., & Wager, T. D. (2000). The unity and diversity of executive functions and their contributions to complex “frontal lobe” tasks: A latent variable analysis. *Cognitive Psychology*, *41*(1), 49-100.

Mochón-Benguigui, S., Carneiro-Barrera, A., Castillo, M. J., & Amaro-Gahete, F. J. (2021). Role of physical activity and fitness on sleep in sedentary middle-aged adults: the FIT-AGEING study. *Scientific Reports*, *11*(1), 1-12.

Mullen, T. R., Kothe, C. A., Chi, Y. M., Ojeda, A., Kerth, T., Makeig, S., ... & Cauwenberghs, G. (2015). Real-time neuroimaging and cognitive monitoring using wearable dry EEG. *IEEE Transactions on Biomedical Engineering*, *62*(11), 2553-2567.

Müller, N. G., & Knight, R. T. (2006). The functional neuroanatomy of working memory: contributions of human brain lesion studies. *Neuroscience*, *139*(1), 51-58.

Owen, A. M., McMillan, K. M., Laird, A. R., & Bullmore, E. (2005). N-back working memory paradigm: A meta-analysis of normative functional neuroimaging studies. *Human Brain Mapping*, *25*(1), 46-59.

Park, B. S., Kim, I. H., & Park, K. M. (2020). Top 100 cited articles on sleep medicine: a bibliometric analysis. *European Neurology*, *83*(1), 111-120.

Patel, A. K., Reddy, V., & Araujo, J. F. (2021). Physiology, sleep stages. In *StatPearls [Internet]*. StatPearls Publishing.

Peever, J, & Fuller, P. (2017) The Biology of REM Sleep. *Current Biology*, 27(22), R1237-R1248.

Pion-Tonachini, L., Kreutz-Delgado, K., & Makeig, S. (2019). ICLabel: An automated electroencephalographic independent component classifier, dataset, and website. *NeuroImage*, *198*, 181-197.

Poisson, R. J., & Miller, M. E. (2014). Spatial disorientation mishap trends in the US Air Force 1993–2013. *Aviation, Space, and Environmental Medicine*, *85*(9), 919-924.

Pontifex, M. B., Hillman, C. H., Fernhall, B. O., Thompson, K. M., & Valentini, T. A. (2009). The effect of acute aerobic and resistance exercise on working memory. *Medicine & Science in Sports & Exercise*, *41*(4), 927-934.

Purves, D., Augustine, G. J., Fitzpatrick, D., Hall, W. C., LaMantia, A. S., & White, L. E. (2011). Neuroscience, 5th edition. Sunderland, MA.

Ritland, B. M., Simonelli, G., Gentili, R. J., Smith, J. C., He, X., Mantua, J., ... & Hatfield, B. D. (2019). Effects of sleep extension on cognitive/motor performance and motivation in military tactical athletes. *Sleep Medicine*, *58*, 48-55.

Roca, J., Castro, C., López-Ramón, M. F., & Lupiánez, J. (2011). Measuring vigilance while assessing the functioning of the three attentional networks: The ANTI-Vigilance task. *Journal of Neuroscience Methods*, *198*(2), 312-324.

Rundo, J. V., & Downey III, R. (2019). Polysomnography. *Handbook of Clinical Neurology*, *160*, 381-392.

Rupp, T. L., Wesensten, N. J., Bliese, P. D., & Balkin, T. J. (2009). Banking sleep: Realization of benefits during subsequent sleep restriction and recovery. *Sleep*, *32*(3), 311-321.

Sanders, J., Johnson, K. A., Garavan, H., Gill, M., & Gallagher, L. (2008). A review of neuropsychological and neuroimaging research in autistic spectrum disorders: Attention, inhibition and cognitive flexibility. *Research in Autism Spectrum Disorders*, *2*(1), 1-16.

Sarter, M., Givens, B., & Bruno, J. P. (2001). The cognitive neuroscience of sustained attention: where top-down meets bottom-up. *Brain Research Reviews*, *35*(2), 146-160.

Schwartz, J., & Simon Jr, R. D. (2015). Sleep extension improves serving accuracy: A study with college varsity tennis players. *Physiology & Behavior*, *151*, 541-544.

Skorucak, J., Arbon, E. L., Dijk, D. J., & Achermann, P. (2018). Response to chronic sleep restriction, extension, and subsequent total sleep deprivation in humans: adaptation or preserved sleep homeostasis?. *Sleep*, *41*(7), zsy078.

Stickgold, R. (2005). Sleep-dependent memory consolidation. *Nature*, *437*(7063), 1272-1278.

Stuss, D., Shallice, T., Alexander, M., & Picton, T. (1995). A Multidisciplinary Approach to Anterior Attentional Functions. *Annual New York Academy of Sciences,* (769), 191-211.

Taylor, M. K., Hernández, L. M., Sessoms, P. H., Kawamura, C., & Fraser, J. J. (2020). Trauma exposure and functional movement characteristics of male tactical athletes. *Journal of Athletic Training*, *55*(4), 384-389.

Tobler, I. (1995). Is sleep fundamentally different between mammalian species? *Behavioural Brain Research*, *69*(1-2), 35-41.

United States Army. (n.d.). *Performance Triad (P3).* https://p3.amedd.army.mil/

United States Army. (2021, July 14). *The Synergy of the Performance Triad!* https://p3.amedd.army.mil/news/what-leaders-need-to-know/what-is-the-performance-triad

Van Dongen, H., Maislin, G., Mullington, J. M., & Dinges, D. F. (2003). The cumulative cost of additional wakefulness: Dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep, 26*(2), 117-126.

van Schie, M. K., Lammers, G. J., Fronczek, R., Middelkoop, H. A., & van Dijk, J. G. (2021). Vigilance: discussion of related concepts and proposal for a definition. *Sleep Medicine, 83*, 175-181.

van Schooten, K. S., Duran, L., Visschedijk, M., Pijnappels, M., Lord, S. R., Richardson, J., & Delbaere, K. (2019). Catch the ruler: concurrent validity and test–retest reliability of the ReacStick measures of response time and inhibitory executive function in older people. *Aging Clinical and Experimental Research, 31*, 1147-1154.

Venter, R. (2014). Perceptions of team athletes on the importance of recovery modalities. *European Journal of Sport Science*, 14 (Suppl. 1), S69–S7.

Walker, M. P., Brakefield, T., Morgan, A., Hobson, J. A., & Stickgold, R. (2002). Practice with sleep makes perfect: sleep-dependent motor skill learning. *Neuron*, *35*(1), 205-211.

Whelehan, D. F., Alexander, M., & Ridgway, P. F. (2020). Would you allow a sleepy surgeon operate on you? A narrative review. *Sleep Medicine Reviews*, *53*, 101341.

Wilckens, K. A., Erickson, K. I., & Wheeler, M. E. (2012). Age-related decline in controlled retrieval: the role of the PFC and sleep. *Neural Plasticity*, *2012*, 1-15.

Wilckens, K. A., Ferrarelli, F., Walker, M. P., & Buysse, D. J. (2018). Slow-wave activity enhancement to improve cognition. *Trends in Neurosciences*, *41*(7), 470-482.

Williamson, A. M., & Feyer, A. M. (2000). Moderate sleep deprivation produces impairments in cognitive and motor performance equivalent to legally prescribed levels of alcohol intoxication. *Occupational and Environmental Medicine*, *57*(10), 649-655

Williamson, A. M., Feyer, A. M., Mattick, R. P., Friswell, R., & Finlay-Brown, S. (2001). Developing measures of fatigue using an alcohol comparison to validate the effects of fatigue on performance. *Accident Analysis & Prevention*, *33*(3), 313-326.

Yamazaki, E. M., Antler, C. A., Lasek, C. R., & Goel, N. (2021). Residual, differential neurobehavioral deficits linger after multiple recovery nights following chronic sleep restriction or acute total sleep deprivation. *Sleep*, *44*(4), zsaa224.

Yu, S., Mückschel, M., & Beste, C. (2021). Event-related synchronization/desynchronization and functional neuroanatomical regions associated with fatigue effects on cognitive flexibility. *Journal of Neurophysiology*, *126*(2), 383-397.