POTENTIAL AGE MODERATION OF BLUE LIGHT THERAPY ON PTSD SLEEP SYMPTOM SEVERITY

By

KEILI HELENE OXLEY

A Thesis Submitted to The W.A. Franke Honors College

In Partial Fulfillment of the Bachelor's degree
With Honors in

Psychological Sciences

THE UNIVERSITY OF ARIZONA

MAY 2022

Approved by:

Dr. William D. "Scott" Killgore
Department of Psychiatry
Faculty Thesis Advisor

Abstract

One of the primary symptoms of PTSD is disruption of sleep. Effective treatment for sleep dysfunction in PTSD populations is lacking. There is a need for new therapeutic interventions because sleep is necessary for memory consolidation and emotion processing. Since sleep, memory, and mood are associated with one another, it is reasonably expected that if sleep in individuals with PTSD can be improved, enhanced memory and emotional processing will follow suit which may result in diminished or even disappearance of symptoms. A potential non-pharmacological approach for treating sleep problems is through morning blue light therapy (BLT). This thesis aimed to determine the effectiveness of BLT at treating PTSD symptoms, specifically through assessing subjective improvements in sleep quality and disturbances over six weeks, as measured by the Pittsburg Sleep Quality Index (PSQI). In addition, the impact of age on the effectiveness of light treatment was examined to see if changes in the eye physiology that arise from aging, specifically cataracts, negatively affect the capability for light to stimulate intrinsically photosensitive retinal ganglion cells (ipRGCs). It was hypothesized that daily administration of morning BLT would positively influence sleep quality more than the placebo condition of amber light therapy (ALT), but the extent of improvements in sleep would be moderated by older age. The results showed that sleep quality improved after six weeks of light treatment, regardless of light condition and age. However, there were trends towards better sleep in the younger population. This suggests that both wavelengths of light were effective at improving sleep in the PTSD population, but younger individuals may gain more improvements from light treatment than older individuals. These findings may inform further studies that investigate BLT at treating psychological disorders.

Introduction

Post-traumatic stress disorder (PTSD) is a mental health condition that is triggered by experiencing or witnessing a traumatic event. There are various events that can trigger PTSD symptoms including war and combat, sexual and physical abuse, accidents, near-death experiences, and other disasters. Among individuals who have been exposed to severe trauma, approximately 5.6% experience lifetime prevalence of PTSD symptoms. Additionally, PTSD is more common among populations that are minority, female, younger, uneducated, unemployed, and impoverished (Koenen et al., 2017). PTSD is associated with increased incidence of mood disturbances, poor emotion regulation abilities, and poor quality of sleep (Bisson et al., 2015). Sleep disturbances, insomnia, and treatment-resistant nightmares are diagnostic criteria for the disorder and are some of the most common symptoms or complaints for patients suffering from PTSD (Spoormaker, 2008). It is common for PTSD patients to have a fear of sleep that may be driven by anxiety about having a nightmare or being vulnerable while asleep. These concerns often cause avoidance of sleep and/or difficulty falling asleep (Richards et al., 2020). To compensate for the lack of sleep, many individuals with PTSD adopt maladaptive sleep behaviors and coping strategies, such as taking naps during the day, sleeping in, or increasing the total amount of time spent in bed, to attain a satisfactory amount of sleep. These strategies may lead to circadian misalignment, which can result in serious negative health implications later in life (Grandner, 2017).

It is well-documented that sleep plays a critical role in memory and emotion processing. Sleep disturbances may interfere with the processing of emotional memories following trauma and impact memory consolidation abilities that are necessary for fear extinction of the traumatic memories (Pace-Schott et al., 2015). In PTSD, the traumatic memory has been consolidated as a

fear memory and retrieval of the traumatic memories is associated with stress and fear responses. Some treatments use fear extinction to regulate fear memory through continuous reintroduction, retrieval, or re-exposure to the fear memory which gradually reduces the stress response over time (Kida, 2019). Fear extinction is a learning process that leads to inhibition of the fear response to the stressful or traumatic memory. Poor extinction memory in PTSD may lead to heightened fear expression during the day, which in turn causes decreased rapid eye movement (REM) sleep quality at night (Murkar & Koninck, 2018). Evidence indicates that REM is important for emotional memory processing and is vital for subsequent waking-day reactivity to emotional stimuli. Thus, disordered REM may be a significant contributor to the pathophysiology of emotion-based disorders, such as PTSD (Murkar & Koninck, 2018). These findings suggest improving sleep could be a meaningful target for therapeutic intervention for individuals diagnosed with PTSD.

It has also been confirmed that light is one of the most powerful regulators of our sleep-wake schedule (Wirz-Justice et al., 2021). A little over two decades ago, a novel class of photoreceptors was discovered, termed intrinsically photosensitive retinal ganglion cells (ipRGCs). These cells, which are not part of the image-forming visual system, relay information about light exposure to the suprachiasmatic nucleus (SCN) of the anterior hypothalamus which influence actions related to circadian rhythms, melatonin suppression, cognition, emotional processing, mood, and pupillary constriction (Touitou et al., 2017). These ipRGCs are maximally sensitive to blue light (λ = 460–480 nm) and less so to longer wavelengths including green, amber, and red light. Blue light can be used to induce wakefulness and promote circadian phase shifts, when administered in the morning (Tosini et al., 2016). Research indicates that blue light therapy (BLT) provides a non-pharmacological approach to decreasing daytime sleepiness and

fatigue, while improving sleep quality, duration, and latency (Killgore et al., 2020). Thus, administration of blue light at specific times of the day may be a promising intervention for sleep disruptions caused by PTSD and/or other disorders.

There is preliminary evidence that suggests that light, particularly blue light, helps to reset circadian rhythms which can aid in overall sleep (Wahl et al., 2019). The aim of this study was to evaluate the effectiveness of blue light therapy for improving symptoms of PTSD. The specific questions being raised are whether exposure to blue light therapy (BLT) improves sleep quality in patients who are diagnosed with PTSD better than amber light therapy (ALT) and if age moderates this relationship. With age, the lens in our eye becomes opaque and/or yellow which prevents light from passing through to the retina (Shiels & Hejtmancik, 2019). This phenomenon is known as cataracts. Therefore, it is proposed that older individuals may have greater expressions of cataracts and therefore may experience less stimulation of the ipRGCs. It was hypothesized that daily administration of morning BLT would positively influence sleep quality more than the placebo condition of amber light therapy (ALT), but the extent of improvements in sleep would be moderated by participant age.

Literature Review

Defining PTSD

Post-traumatic stress disorder (PTSD) is a mental health condition that is triggered by exposure to a traumatic event. This disorder can develop after a single distressing event or from prolonged exposure to trauma. Additionally, symptoms may present immediately following the event(s) or years later, and can persist for decades (Koenen et al., 2017). Various traumatic events can provoke the onset of PTSD, including direct or indirect exposure to combat, abuse, accidents, natural disasters, or other severe stressors. PTSD is characterized by clusters of

symptoms including, intrusive memories, avoidance behaviors, negative cognitions and mood, and hyperarousal (Bisson et al., 2015). Diagnosis of PTSD is based off the presence of these symptoms according to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5) or the International Classification of Diseases, 11th edition (ICD-11) guidelines. It is important to note that the diagnostic criteria between the DSM-5 and the ICD-11 are different, and cases that were diagnosed through one system may not be through the other. However, both diagnostic tools differentiate PTSD from similar disorders by emphasizing that the nature of the trauma resulted in at least one or two symptoms from each of the categorical criteria, persisted for at least one month, and impaired daily functioning (Bisson et al., 2015).

Pathogenesis of PTSD

The pathophysiology of PTSD is interdisciplinary in nature. PTSD arises from dysregulation of the mind-body interactions causing both psychological and physical implications on an individual. One theory is that enhanced neuroendocrine and immune responses to stress following traumatic experiences induces the onset of PTSD (Miao et al., 2018). The abnormal activation of the sympathetic nervous system disrupts normal functioning of the hypothalamic-pituitary-adrenal (HPA) axis (Miao et al., 2018). The excessive release of epinephrine and norepinephrine by the adrenal medulla stimulates the hypothalamus to produce elevated levels of corticotropin-releasing hormone (CRH) causing impaired downstream effects. The abnormal CRH levels ultimately result in diminished release of cortisol that serves to inhibit HPA axis activity through negative feedback mechanisms (Miao et al., 2018). Additionally, the overactivity of the autonomic nervous system and HPA axis disturb immune system regulation. There are elevated levels of pro-inflammatory cytokines in PTSD patients, thus leading to inappropriate inflammation that can cause damage to tissues (Neigh & Ali, 2016). If this

enhanced cell-mediated immune response is left untreated, it can further lead to the development of allergies and autoimmune diseases. Therefore, it is imperative that effective treatment and management of PTSD is adopted in a timely manner.

Evidence suggests genetics and epigenetics may predispose an individual to developing PTSD. It is possible that structural variations in the brain affect PTSD vulnerability. Studies have determined that alterations in certain genes are highly correlated with PTSD development. DNA methylation and histone methylation/acetylation of genes related to the physiological stress response, learning and memory, mood, and pain perception are associated with neural phenotypes of PTSD (Lebois et al., 2016). For example, the FKBF5 gene expressed in the hippocampus is responsible for modulating HPA-axis reactivity, specifically by maintaining proper glucocorticoid receptor (GR) sensitivity. A genetic polymorphism of this specific gene reduces GR sensitivity to binding glucocorticoids, thus suppressing cortisol release, and allowing the stress response to be maintained. The fact that this gene resides in the hippocampus suggests its potential to affect memory formation and recall. Additionally, evidence indicates that exposure to high stress can epigenetically alter gene expression in offspring, suggesting that the propensity for developing PTSD after trauma may be hereditary (Miao et al., 2018).

Sleep After Trauma Exposure

It is well-documented that exposure to trauma negatively impacts sleep. Individuals that have been exposed to trauma experience a high prevalence of sleep disturbances consisting of trouble falling and staying asleep, recurring nightmares, panicked awakenings, and disruptive sleep behaviors (Richards et al., 2020). It is theorized that traumatic experience(s) cause an increase in arousal that is affecting overall sleep quality and quantity. Trauma-related sleep disturbances predict physical and psychiatric symptoms and are now deemed to be primary

contributors to PTSD progression and maintenance (Miller et al., 2020). Therefore, attenuating sleep disturbances could be a viable means of preventing trauma-exposed individuals from developing PTSD. Currently, there is a lack of effective interventions that focus on improving sleep in this population.

Sleep Disorders in PTSD

Common sleep disorders have been identified in PTSD, such as insomnia, obstructive sleep apnea (OSA), and other circadian rhythm sleep disorders. Insomnia is described as a sleep disorder that is characterized by difficulties in initiating and maintaining sleep that results in in daytime impairment and/or distress (Richards et al., 2020). Excessive rumination and worry around sleep leads to increased arousal, leaving the individual awake and alert. In addition, more time spent in bed in the absence of sleep causes frustration which perpetuates wakefulness and further reduces the likelihood for sleep to occur. Inadequate sleep duration brings about compensatory mechanisms that lead to shifts in circadian rhythms (Miller et al., 2020). However, it is necessary to mention that cognitions about sleep differ between insomnia and PTSD. In insomnia disorder, individuals have a desire for sleep, but individuals with PTSD often tend to avoid sleep altogether (Richards et al., 2020).

Some studies have found a positive relationship between PTSD symptom severity and the risk for developing OSA. It has been proposed that trauma-related sleep disturbances increase vulnerability for OSA which further leads to more severe PTSD symptomology (Miller et al., 2020). Additionally, the mechanism by which OSA may present more frequently in PTSD populations is unclear and unconvincing but could be explained by the rising rates of obesity in people with PTSD (Richards et al., 2020). It is also likely that increased obesity prevalence in this population is due to improper sleep hygiene.

Poor Sleep Quality in PTSD

Sleep disturbances and recurrent intrusive nightmares are diagnostic criteria for the disorder and are some of the most common symptoms and/or complaints for patients diagnosed with PTSD (Spoormaker, 2008). This further supports the argument that sleep is a significant symptom of concern in the disorder. It is also common for PTSD patients to have a fear of sleep that may be driven by anxiety about having a nightmare or being vulnerable while sleeping. These worries often cause avoidance of sleep and/or difficulty falling asleep. To compensate for the lack of nighttime sleep, many individuals with PTSD adopt maladaptive sleep behaviors and coping strategies to try to attain a satisfactory amount of sleep by taking naps during the day, sleeping in, and increasing the total amount of time spent in bed (Richards et al., 2020). However, this leads to circadian misalignment and leaves them in a vicious cycle of nighttime wakefulness and daytime sleepiness which can result in serious negative health implications later in life (Grandner, 2017). It seems that these trauma-related symptoms contribute significantly to the poor subjective sleep quality reported in PTSD. There is evidence to suggest that mitigating PTSD-related sleep symptoms could be a strong target for intervention.

Sleep, Memory, and Emotion Regulation

Past findings indicate that sleep plays a critical role in memory consolidation and the processing of emotions. More specifically, rapid-eye-movement (REM) sleep is known to be important for emotional memory processing and is vital for subsequent waking-day reactivity to emotional stimuli (Tempesta et al., 2018). Evidence suggests REM sleep enhances memory for emotional stimuli while also suppressing reactivity to emotional stimuli. (Murkar & Koninck, 2018). Thus, disordered REM may contribute significantly to the pathophysiology of emotion-based disorders, such as PTSD. If an individual experiences disrupted sleep patterns and

behaviors, then it is viable to infer that the memory of the traumatic event(s) is altered and causing hyperarousal. Additionally, sleep impacts the fear memory formation, modification, and its extinction (Pace-Schott et al., 2015). The literature demonstrates a link between memory, emotion, and sleep.

Memory and Emotion Regulation Dysfunction in PTSD

There is a high likelihood that fear memory disruption among PTSD patients is impaired due to issues with sleep. In PTSD, the traumatic memory has been consolidated as a fear memory and retrieval of the traumatic memories is associated with stress and fear responses. Sleep disturbances may interfere with the processing of emotional memories following trauma and impact memory consolidation abilities that are necessary for fear extinction of the traumatic memories (Pace-Schott et al., 2015). This further demonstrates the notion that disruptions in sleep may contribute to PTSD development. Poor extinction memory in PTSD may lead to heightened fear expression during the day, which in turn causes decreased REM sleep quality at night (Murkar & Koninck, 2018). There is evidence that suggests improving sleep will promote both fear memory and extinction memory consolidation (Pace-Schott et al., 2015). These findings support the idea that alleviating PTSD-related sleep symptoms could be a strong target for treatment intervention.

Prevention and Treatment of PTSD

Currently, there are a few different approaches to prevention and treatment of PTSD, but none that effectively target and improve sleep in this population. Interventions for PTSD are psychological or pharmacological in nature. It is difficult to appropriate primary prevention interventions for trauma exposure and PTSD in the general population. Luckily, there are some measures in place for particular groups of individuals, such as military personnel, firefighters,

and other first responders, who may be at high risk for exposure to traumatic events. Training and education regarding stress management, relaxation techniques, healthy coping mechanisms, and emotional intelligence components may serve as potential strategies for psychological prevention (Miao et al., 2018). However, the efficacy of psychoeducation and training is still inconclusive. There is evidence suggesting that administration of brief and trauma-focused cognitive behavioral interventions may help reduce symptom severity in individuals showing early symptoms of PTSD due to a single traumatic event (Bisson et al., 2015). Pharmacological prevention measures have targeted the influence of stress on memory formation. Some studies have indicated that primary prevention of PTSD is most effective when sympatholytic drug treatment is started before and early after the traumatic event (Miao et al., 2018). However, there is a lack of convincing evidence because it is difficult to obtain IRB approval for studies of this nature since there are many potential side effects and ethical issues that arise when administering drugs to people who do not have valid diagnoses.

Research on effective treatment for PTSD continues to grow. The primary goal of treatment is to see improvements in PTSD symptom severity, reduction of comorbid medical or psychiatric conditions, enhanced quality of life, and diminished disability/functional impairment. First-line treatment options are usually trauma-focused psychological interventions. Some examples include cognitive therapies, exposure therapies, eye movement desensitization and reprocessing (EMDR), and hypnotherapies (Bisson et al., 2015). Studies have demonstrated that these psychological treatments effectively improve PTSD symptoms and provide long lasting effects (Kline et al., 2018). Drug treatment is a second-line treatment option. PTSD patients are often prescribed either antidepressants or sympatholytic drugs. Among these types of medications, fluoxetine, paroxetine, sertraline, and venlafaxine showed significant reduction of

PTSD symptom severity when compared with placebos (Miao et al., 2018). It should also be mentioned that treating PTSD using a combination of psychological and pharmacological interventions resulted in insufficient and inconclusive evidence of improvement (Bisson et al., 2015).

The lack of research and literature for effective treatment of sleep dysfunction in PTSD populations demonstrates the need for new therapeutic strategies and experimentation. Since sleep, memory, and mood are associated with one another, it is reasonably expected that if sleep in individuals with PTSD can be improved, enhanced memory and emotional processing will follow suit and may result in diminished or even disappearance of symptoms.

Unknowns

There are still many unknowns regarding PTSD. It is a complex condition and there a great number of factors that could influence the onset of PTSD. For one, the diagnostic criteria for PTSD are somewhat ambiguous likely because the fundamental understanding and definition of the disorder is still uncertain. Additionally, in many cases, it is difficult to ascertain whether the discussed variations in anatomy, physiology, and/or behaviors existed before trauma exposure or if they are direct acquisitions of PTSD development. It is likely a combination of vulnerability factors and consequences of trauma exposure that produce the symptoms present in PTSD. Overall, future research focusing on this area of study is necessary to gain more familiarity with the concepts previously discussed.

Blue Light Therapy

The literature suggests that memory and emotion processing are associated with sleep, but there is a lack of effective treatments that alleviate these common impairments evident in PTSD. The link between these vital components of human functioning provides a potential basis

for treatment of PTSD. Since non-pharmacological therapies, such as cognitive behavioral therapy for insomnia (CBT-I) and imagery rehearsal therapy (IRT), demonstrate effectiveness in improving sleep and symptom severity in veterans diagnosed with PTSD, similar non-drug therapies hold the most promise for PTSD symptom relief (Miller et al., 2020).

A potential non-pharmaceutical treatment option for PTSD is blue light therapy (BLT). Light is one of the most powerful regulators of our sleep-wake schedule (Wirz-Justice et al., 2021). Blue light is visible light that is comprised of emission wavelengths in the range of 400-490 nm. A little over two decades ago, a novel class of photoreceptors was discovered, termed intrinsically photosensitive retinal ganglion cells (ipRGCs). These cells, which are not part of the image-forming visual system, relay information about light exposure to the suprachiasmatic nucleus (SCN) of the anterior hypothalamus which is deemed to be the brain's master clock (Wahl et al., 2019). They influence actions related to circadian rhythms, melatonin suppression, cognition, emotional processing, mood, and pupillary constriction (Touitou et al., 2017). These ipRGCs are maximally sensitive to blue light (λ = 460–480 nm) and less so to longer wavelengths including green, amber, and red light.

There is preliminary evidence that suggests when administered in the morning, blue light can be used to induce circadian phase shifts and wakefulness during the day to promote nighttime sleep that will aid in cognition, memory, and emotional processing and regulation (Touitou et al., 2017). BLT has been used as an effective treatment for certain mood disorders such as major depressive disorder (MDD) and seasonal affective disorder (SAD) (Maruani & Geoffroy, 2019). In addition, BLT has been used to effectively treat sleep and mood disturbances in individuals with mild traumatic brain injury (mTBI) by decreasing daytime sleepiness and fatigue that lead to improvements in sleep quality, duration, and latency (Killgore et al., 2020).

Although, it should be noted that blue light, particularly later in the day or evening, can also hinder sleep because of its ability to shift circadian rhythms. Due to the increasing prominence of blue light in society, a large segment of the world's population is subjected to daily exposure of artificial light from electronics during the hours approaching sleep (Tosini et al., 2016). Even low intensity light found in recent technologies and electronics can act on the biological clock that can causes phase delays and slowing of melatonin secretion (Touitou et al., 2017). Circadian rhythms are coordinated by internal/biological and external/astronomical signals of time. When these components are no longer in tune, then the entire body system experiences desynchronization resulting in alteration of circadian parameters (Foster et al., 2013). Clinical symptoms, such as persistent fatigue and sleep disorders that could lead to chronic insomnia and mood disorders, develop with circadian misalignment.

To maintain a healthy circadian system, it is imperative that the proportion of artificial blue light exposure encountered during the day is accompanied by a reduction of the same proportion during the evening hours (Wahl et al., 2019). It is critical that exposure to blue light be administered in an appropriate time frame to experience beneficial effects. Thus, administration of BLT in the morning, before beginning the day's activities, may be an effective non-pharmacological treatment intervention for sleep disruptions caused by PTSD and/or other disorders.

Cataracts

As we age, the lens in our eyes becomes opaque and/or yellow, and we develop cataracts. The lens is responsible for transmitting and focusing light onto the retina where the ipRGCs reside. Cataracts arise due to disorganization of the precise arrangement of lens fiber cells and/or accumulation of denatured lens crystallin proteins which disrupt the normal optical density

(Shiels & Hejtmancik, 2019). Cataracts influence how light is transmitted through the lens by scattering light which may reduce ipRGC stimulation. Therefore, cataracts may reduce the effectiveness for BLT to treat sleep symptoms. It is proposed that the older population in the study may have some cataract formation that impacts blue light's ability to treat sleep problems, so they will experience weakened improvements from treatment compared to the younger population.

In summary, there is evidence to suggest that BLT has a beneficial effect on mood, cognition, and sleep in individuals with major depressive disorder (MDD), seasonal affective disorder (SAD), and mild traumatic brain injury (mTBI). However, it is currently unknown whether BLT may lead to similar beneficial effects in individuals with PTSD. In addition, there is evidence that suggests exposure to BLT impacts the processing of memories and emotional stimuli, but how these components affect individuals with PTSD is unclear. The goal is to assess the impact of daily BLT in comparison to a daily amber light therapy (ALT) placebo condition on trauma-related sleep symptoms associated with PTSD and determine if age acts as a moderator. The proposed hypothesis is if PTSD patients are stimulated with BLT in the morning, then their circadian rhythms will be re-set which will help them sleep better in the evening and allow them to process their traumatic memories and emotions, but older individuals will experience improvements in sleep of a lesser extent because of age-related cataracts that may diminish ipRGC sensitivity.

Methods

Participants

Research participants aged 18 to 50 years who have been diagnosed with PTSD based off the Structured Clinical Interview for the DSM-5 Axis I Disorders (SCID-5) were recruited for

the study through Internet advertisements and flyers that had been placed around businesses and organizations within the Tucson community. Eligibility criteria are listed below.

Inclusion Criteria:

1) Age 18-50 years; 2) Right handedness as assessed by the Edinburgh Handedness Inventory (EHS) (necessary to avoid mixed lateralization on brain imaging); 3) Clinical diagnosis of PTSD based on the Structured Clinical Interview for DSM-5 (SCID-5).

Exclusion Criteria:

1) History of head injury with loss of consciousness or post-traumatic amnesia, or major neurological illness; 2) Medical or neurologic conditions (e.g., diabetes, cancer, epilepsy) that would confound interpretation of results; 3) Mixed or left-handedness; 4) Abnormal visual acuity that cannot be corrected by contact lenses (necessary to see stimuli in scanner environment); 5) Less than a 9^{th} grade education or IQ estimate less than 80; 6) Metal within the body, pregnancy, or other contraindication for MRI procedures; 7) Previous formal treatment with light therapy; 8) History of light-induced migraine or epilepsy; medical complications that could elevate the risk of discomfort associated with light-therapy; 9) Use of medications that could affect functional neuroimaging and other results (e.g., ACE inhibitors, betablockers, mood stabilizers). Patients currently taking acceptable medications (e.g., SSRIs, SNRIs) must take them consistently for six weeks prior to participation; 10) Participants were excluded if they were currently taking or anticipated the need to take sleep-inducing medications (e.g., doxepin and trazodone) or supplements that have known effects on sleep (e.g., melatonin) throughout the course of the study; 11) Current alcohol abuse (more than 2 instances of a 5+ drink intake (men) or a 4+ drink intake (women) while drinking in the past two months, and/or on average

drinking >2 drinks per day (men) and >1 drinks per day (women) during the past two months; 12) Significant use of illicit drugs; 13) History of marijuana usage within the past 4 weeks, use of marijuana prior to the age of 16, and/or a history of greater than moderate marijuana usage throughout the participant's lifetime; 14) History of a substance use disorder or alcoholism; 15) Night work, shiftwork, or anyone who has a significantly desynchronized work-sleep schedule (i.e., sleeping later than 10:30 a.m. more than once per week); 16) Onset of PTSD before 18 years of age; 17) PTSD acquired 10 years or longer prior to participation in the study.

The present study and all its procedures obtained approval from the University of Arizona Institutional Review Board (IRB) and the U.S. Army's Human Research Protection Office (HRPO).

Procedures

Interested individuals completed a screening visit to determine their eligibility for the study. During this visit, participants consented to participate in the study and completed a full SCID, along with cognitive assessments and demographics questionnaires, which are listed below (chart 1). Eligible participants were provided a wrist-worn accelerometer, also known as an actiwatch (Philips Respironics Actiwatch Spectrum) to quantify sleep and were instructed on how to fill out daily sleep diaries. At this time, participants were scheduled for a baseline visit that would occur approximately one week later.

Visit 1: (Screening)

- Full SCID-5
- Cognitive Assessments
  - Wechsler Abbreviated Scale of Intelligence (WASI-II)
  - Wide Range Achievement Test (WRAT)
- Questionnaires
  - Alcohol Use Disorders Identification Test (AUDIT)
  - Combat Exposure Scale (CES)
  - Edinburgh Handedness Inventory (EHI)
  - Marijuana Use (M/USE)
  - Morningness-Eveningness Questionnaire (MEQ)
  - Rivermead Post-Concussion Symptom Questionnaire (RPCSQ)
  - Trauma History Screen (THS)
- Participant provided actiwatch and instructed on sleep diaries
- Schedule visit 2 (baseline)

Chart 1: Comprehensive List of Tasks, Assessments, and Questionnaires Completed in Visit 1

During the baseline visit, participants performed a variety of tasks, including an MRI scan, cognitive assessments, and emotion-based, computerized, and sleep questionnaires. Participants were randomly assigned to either the blue light treatment (BLT; ~480 nm) group or placebo amber light treatment (ALT; ~530 nm) group and were provided the appropriate light box (BLT: Philips goLITE BLU®, Philips Electronics, Stanford, CT; ALT: Custom lightbox provided by Philips Electronics). Participants were instructed to complete the 30-minute light treatment sessions within two hours of waking each morning, between 8:00 AM and 10:00 AM, for six weeks. Participants were also instructed to position the light box approximately one arm's length away and 45 degrees from their primary direction of gaze. Participants were told that they may perform any activity, but they must remain in front of the light box. Participants were expected to self-report their light box use start-time and duration each day via REDCap surveys.

After six weeks of light treatment, participants came in for a post-treatment visit where they completed tasks similar to those administered in the baseline visit, but with an additional fear conditioning portion. Once all the tasks were completed, the participant was debriefed and compensated for their participation. Listed below are charts that illustrate the study timeline for

participants (chart 2) and a comprehensive list of tasks and activities the participant completed during visits 2 and 3 (chart 3).

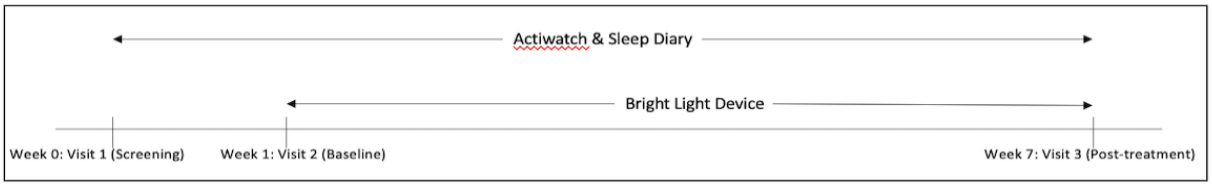


Chart 2: Study Timeline for Participants

Visit 2 & 3: (Baseline/Post-Treatment)

- Bright Light Therapy Device (Blue/Amber)
- Heart Rate Device
- MRI Screening Form
- Fear Conditioning (Visit 3)
- Critical Assessments
  - Clinician-Administered PTSD Scale for DSM-5 (CAPS-5)
  - Multiple Sleep Latency Test (MSLT)
- MRI Scan
  - Anticipation Task
  - BMAT (Fear/Happy)
  - DTI
  - Fear Conditioning Task (Visit 3)
  - Field Map
  - Flair Axial
  - Magnetic Resonance Spectroscopy (MRS)
  - MPRAGE
  - Resting MRI
- Cognitive Assessments
  - Repeatable Battery for the Assessment of of Neuropsychological Status (RBANS A/B)
- Computerized Assessments
  - Balloon Analogue Risk Task (BART)
  - Psychomotor Vigilance Test (PVT)
- Questionnaires
  - Beck Anxiety Inventory (BAI)
  - Beck Depression Inventory (BDI)
  - Connor-Davidson Resilience Scale (CD-RISC)
  - Day of Scan Information Questionnaire (DSIQ)
  - Distressing Dreams & Nightmare Severity Index (DDNSI)
  - Evaluation of Risks (EVAR)
  - Functional Outcome of Sleep Questionnaire (FOSQ)
  - Gratitude Questionnaire (GQ-6)
  - Insomnia Severity Index (ISI)
  - M/USE Calendar
  - Patient Health Questionnaire (PHQ-9)
  - Pittsburg Sleep Quality Index (PSQI)
  - PTSD Checklist for DSM-5 (PCL-5)
  - Satisfaction with Life Scale (SWLS)
  - Spielberger State-Trait Anxiety Inventory (STAI)
  - Stanford Sleepiness Scale (SSS)
  - Therapy Questionnaire

Chart 3: Comprehensive List of Tasks, Assessments, and Questionnaires Completed in Visit 2/3

Thesis-Focused Procedures

The focus of this thesis was on examining the main effects and interaction of BLT and age on PTSD-related sleep symptom severity. The Pittsburg Sleep Quality Index (PSQI) was chosen to quantify sleep quality and disturbance among the population under study. Scores from the baseline visit and the post-treatment visit were included for analysis.

Sleep Symptom Severity – Measured via PSQI

The Pittsburg Sleep Quality Index (PSQI) is a questionnaire that assesses an individual's subjective sleep quality and sleep disturbance within a one-month time interval (Buysse et al., 1989). It consists of 19 individual items that comprise 7 components: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction. Each component is assigned a score based off the self-rated items in the questionnaire, ranging from 0-3 points where "0" indicates no difficulty and "3" indicates severe difficulty. The sum of the component scores results in one global score between 0-21 points where "0" indicates no difficulties and "21" indicates severe difficulties across all components. In other words, lower scores on this assessment imply better quality of sleep and a reduction in component and/or global scores over time suggests improvements in sleep have occurred.

Statistical Analysis

All data analyses were performed using the IBM Statistical Package for Social Sciences (SPSS) program, Version 28. Both descriptive and frequency statistics were executed to determine demographic characteristics of the sample population. To test for internal consistency among the items in the PSQI, reliability statistics were calculated. Cronbach's alpha for the seven components in the questionnaire revealed an internal consistency reliability of 0.538 for

the baseline visit and 0.633 for the post-treatment visit, indicating low to moderate internal reliability. A three-way mixed ANOVA test (2 between-subjects factors: 2 light treatments (ALT versus BLT) and 2 age groups (under age 30 versus age 30 and up); 1 within-subjects factor: PSQI scores over time) was used to assess whether there were group differences for sleep quality over time. This analysis was performed to understand how sleep quality may change over time depending on light condition and age. In addition, analyses using age as a covariate were performed to examine the effect of light treatment on time-based sleep improvements while controlling for age. These tests were performed for each PSQI component individually and globally.

Results

Demographic Characteristics

A total of 84 participants completed the study, but data from 4 participants were omitted from this analysis due to missing either pre- or post-light treatment PSQI scores/responses. Of the 80 participants (n=80), 37 participants received amber light treatment (ALT; λ = 530 nm) and 43 participants received blue light treatment (BLT; λ = 480 nm). As a whole, the population was mostly composed of individuals under age 30 (56.3%) and the mean age was 30.69 years (M = 30.69, SD = 8.642). The population under study was primarily female (66.3%) and white (65%). There were 45 participants in the "under age 30" group (M = 24.36, SD = 2.909) and 35 participants in the "aged 30 and up" group (M = 38.83, SD = 6.959). The statistical groupings of participants are shown in the table below.

Age * Light Group Crosstabulation
Statistics

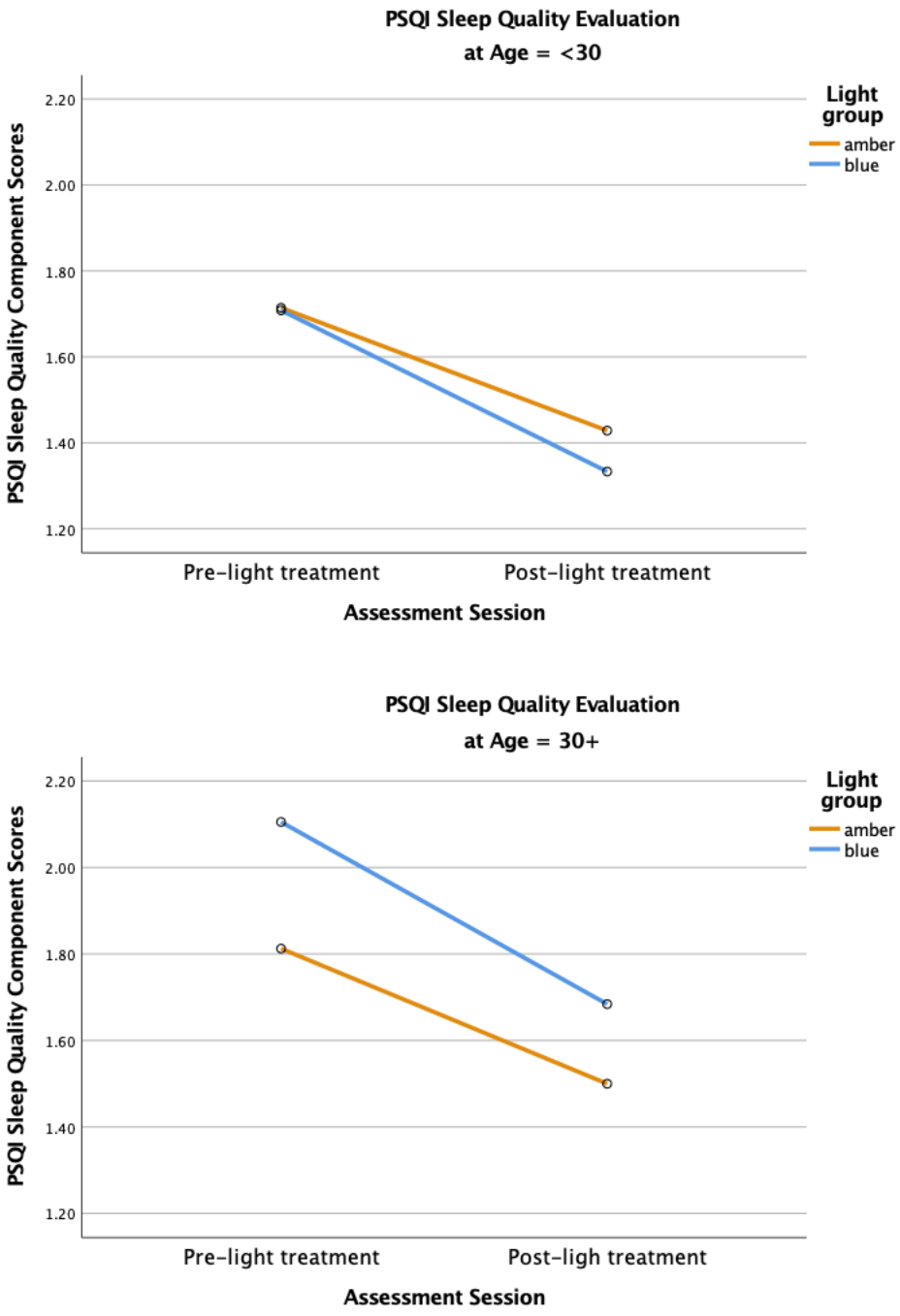
Count

		Light group		
		amber	blue	Total
Age	<30	21	24	45
	30+	16	19	35
Total		37	43	80

Table 1: Contingency table of age and light treatment participant groupings

Subjective Sleep Quality Evaluation

The three-way mixed ANOVA analysis revealed a significant main effect (*at the $p < .05$ level*) of time, F (1,76) = 18.078, p = <.001, but no significant main effects for the light condition, F (1,76) = .486, p = .488, nor the age group, F (1,76) = 2.894, p = .093. There were no significant interactions between light condition and age, F (1,76) = 1.149, p = .287, between time and light condition, F (1,76) = .364, p = .548, between time and age, F (1,76) = .049, p = .825, and there was no significant 3-way interaction between time x light condition x age, F (1,76) = .003, p = .953. These results mean there was a decline in PSQI sleep quality component scores from pre- to post-treatment, regardless of light color and age. Sleep quality improved over the six weeks for each group. The average component scores did not differ depending on age group or light condition, but there was a trend (p = .093) toward better sleep quality in the younger participants. This suggests that the younger participants were getting more satisfactory sleep, in general, than the older participants. There were no significant interactions between the independent variables on sleep quality improvements over time.



Figures 1-2 Three-way mixed ANOVA graphs for PSQI Sleep Quality Component

When controlling for age, the analysis revealed no significant main effects (*at the p < .05 level*) of time, F (1,77) = 1.406, p = .239, light condition, F (1,77) = .32, p = .574, nor age group

F (1,77) = 3.196, p = .078. There were no significant interactions between time and light condition, F (1,77) = .366, p = .547, nor between time and age, F (1,77) = .052, p = .82. These results demonstrate that there was not much difference in average sleep quality scores from pre- to post-light treatment. The PSQI sleep quality component scores did not significantly differ depending on age group or light condition, but there was a trend (p = .078) toward better sleep in the younger participants. This suggests that younger individuals may gain more improvement in sleep quality from light treatment than older individuals. There were no significant interactions between the independent variables on sleep quality improvements over time.

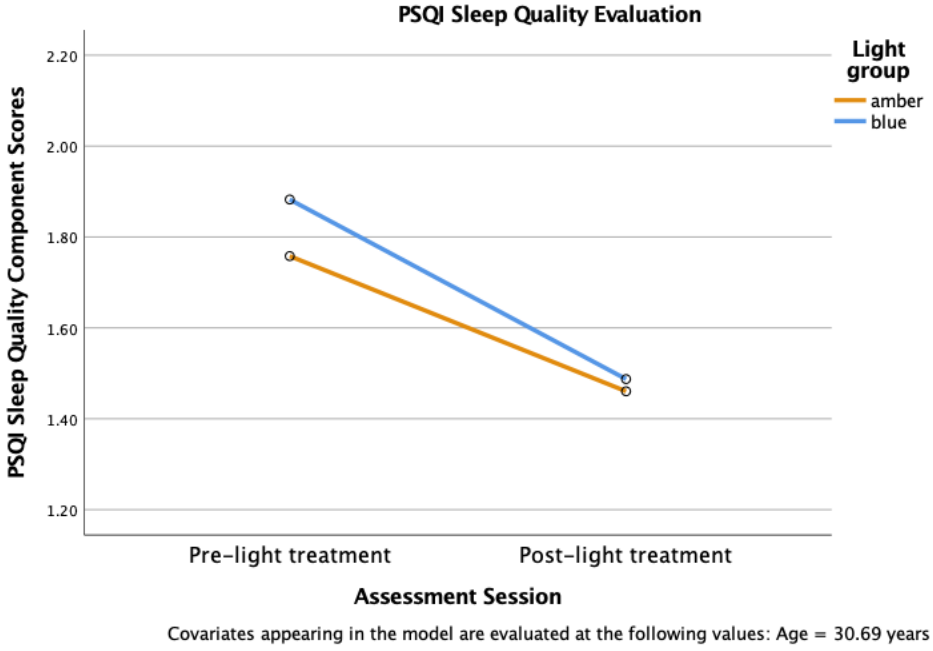
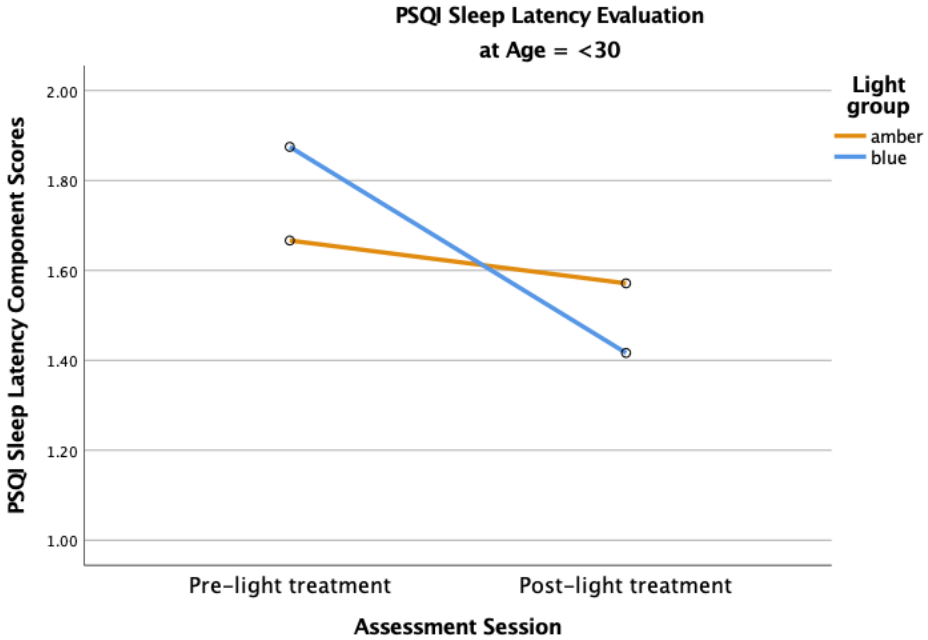


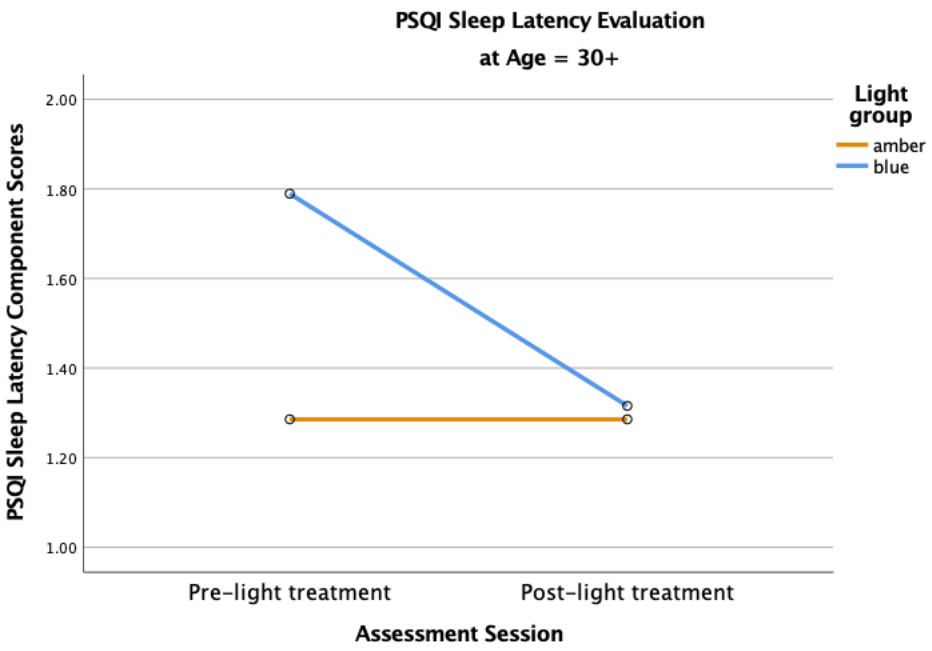
Figure 3 ANCOVA graph for PSQI Sleep Quality Component without consideration for age

Sleep Latency Evaluation

The three-way mixed ANOVA analysis revealed a significant main effect (*at the p < .05 level*) of time, F (1,74) = 9.036, p = .004., but no significant main effects for the light condition,

F (1,74) = .843, p = .362, nor the age group, F (1,74) = 1.778, p = .187. There was no significant interaction between light condition and age, F (1,74) = .563, p = .455. There was a significant interaction between time and light condition, F (1,74) = 5.996, p = .017, but no significant interaction between time and age, F (1,74) = .055, p = .816. This suggests that light condition, but not age, influenced sleep latency scores over time. There was no significant 3-way interaction between time x light condition x age, F (1,74) = .105, p = .747. These results mean there was a decline in PSQI sleep latency scores from pre- to post-treatment, regardless of light color and age. This suggests that sleep latency improved over the six weeks for each group. The average scores for this component did not differ depending on age group or light condition. There were significant differences in sleep latency scores over time depending on the assigned light condition, suggesting that six weeks of treatment with blue light resulted in greater improvements in sleep latency than amber light, for both age groups. There were no other significant interactions between the independent variables on sleep latency improvements over time.





Figures 4-5 Three-way mixed ANOVA graphs for PSQI Sleep Latency Component

When controlling for age, the analysis revealed no significant main effects (*at the p < .05 level*) of time, F (1,75) = 1.479, p = .228, light condition, F (1,75) = .654, p = .421, nor age group, F (1,75) = 1.584, p = .212. There was a significant interaction between time and light condition, F (1,75) = 5.968, p = .017, but no significant interaction between time and age, F (1,75) = .04, p = .843. These results suggest that there was not much difference in PSQI sleep latency component scores from pre- to post-treatment. Additionally, the average scores for this component did not significantly differ depending on age group nor light condition. For both age groups, the blue light condition improved sleep latency significantly more than the amber light condition. This suggests light condition affects sleep latency scores over time, but age does not.

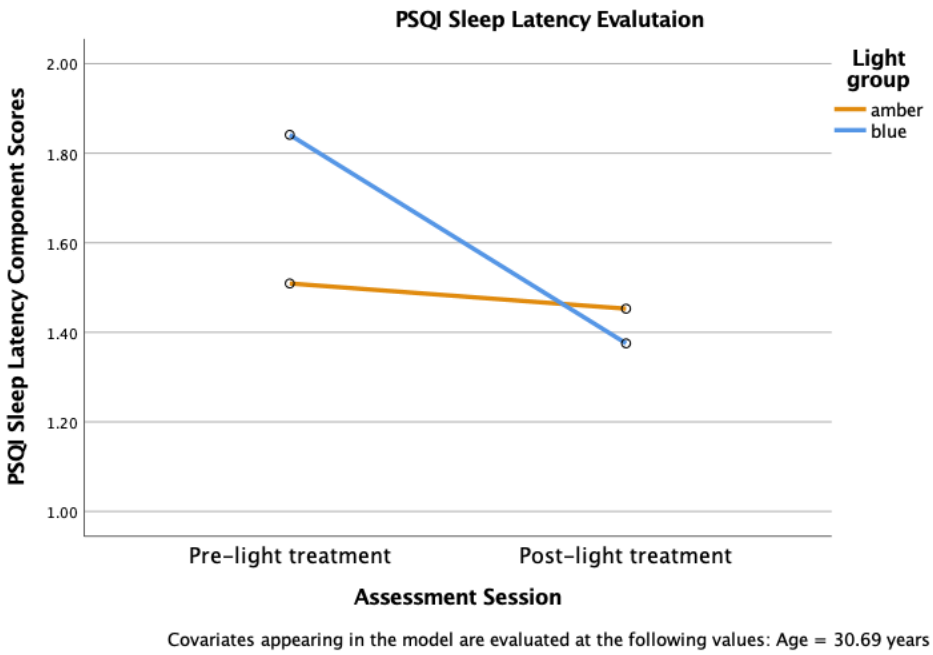
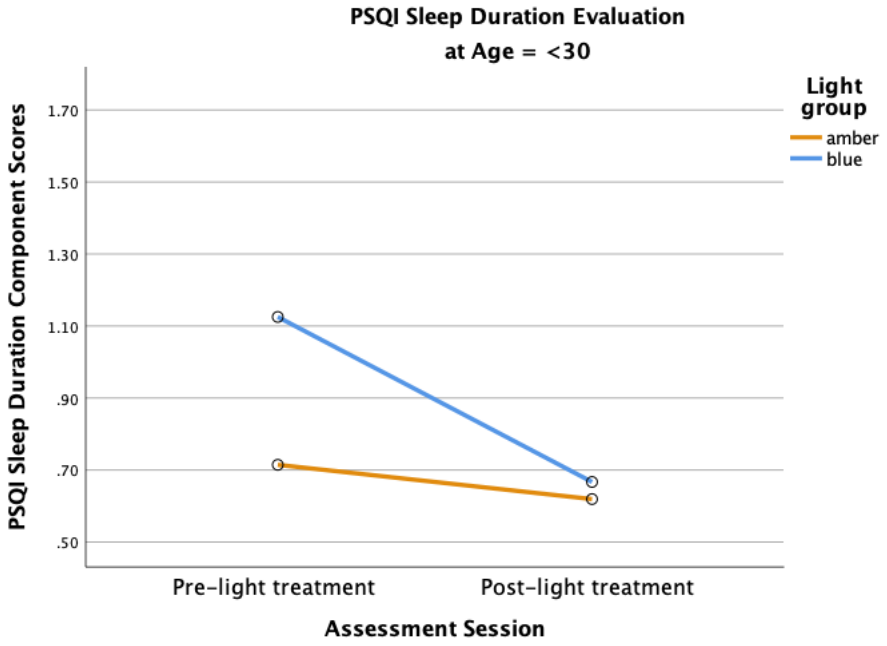


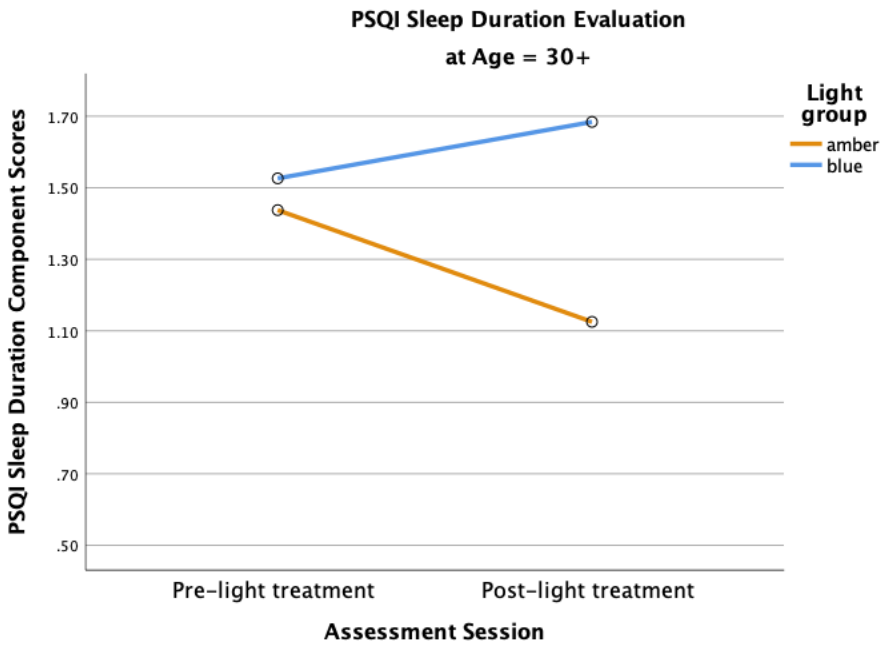
Figure 6 ANCOVA graph for PSQI Sleep Latency Component without consideration for age

Sleep Duration Evaluation

The three-way mixed ANOVA analysis revealed non-significant main effects (*at the p < .05 level*) of time, F (1,76) = 3.122, p = .081 and light condition, F (1,76) = 2.374, p = .128, but there was a significant age group main effect, F (1,76) = 13.598, p = <.001. There were no significant interactions between light condition and age, F (1,76) = .07, p = .792, between time and light condition, F (1,76) = .072, p = .79, nor between time and age, F (1,76) = .991, p = .323. There was a significant 3-way interaction between time x light condition x age, F (1,76) = 4.324, p = .041. These results suggest there was not a significant difference in sleep duration component scores from pre- to post-treatment, but there was a trend (p = .081) towards a decline in scores over time suggesting sleep duration improved slightly over the six weeks. There was no significant difference in component scores according to light condition, but there was a significant difference based off age group, showing that the younger population generally had

more satisfactory durations of sleep than the older population. There appears to be an interaction among light condition and age that significantly affects the sleep duration PSQI component score over time, such that the blue light therapy, only in the older population, resulted in higher scores over the six weeks of treatment. This suggests that older individuals experienced declines in sleep duration over the six weeks of treatment with the blue light. There were no other significant interactions between the independent variables on sleep duration improvements with time.





Figures 7-8 Three-way mixed ANOVA graphs for PSQI Sleep Duration Component

When controlling for age, the analysis revealed no significant main effects (*at the p < .05 level*) of time, F (1,77) = 2.814, p = .097, nor light condition, F (1,77) = 2.337, p = .13, but there was a significant age group main effect, F (1,77) = 14.001, p = <.001. There were no significant interactions between time and light condition, F (1,77) = .000, p = .996, nor between time and age, F (1,77) = 1.289, p = .26. These results indicate that there was not a significant difference in sleep duration component scores from pre- to post-treatment, but there was a trend (p = .097) towards a decline in scores over time suggesting sleep durations got better over the six weeks, regardless of light condition and age. There was no significant difference in scores according to light condition, but there was a significant difference in the scores based off age group showing that the younger population had lower scores than the older population which suggests they generally had more satisfactory durations of sleep than the older population. There were no significant interactions between the independent variables on sleep improvements over time.

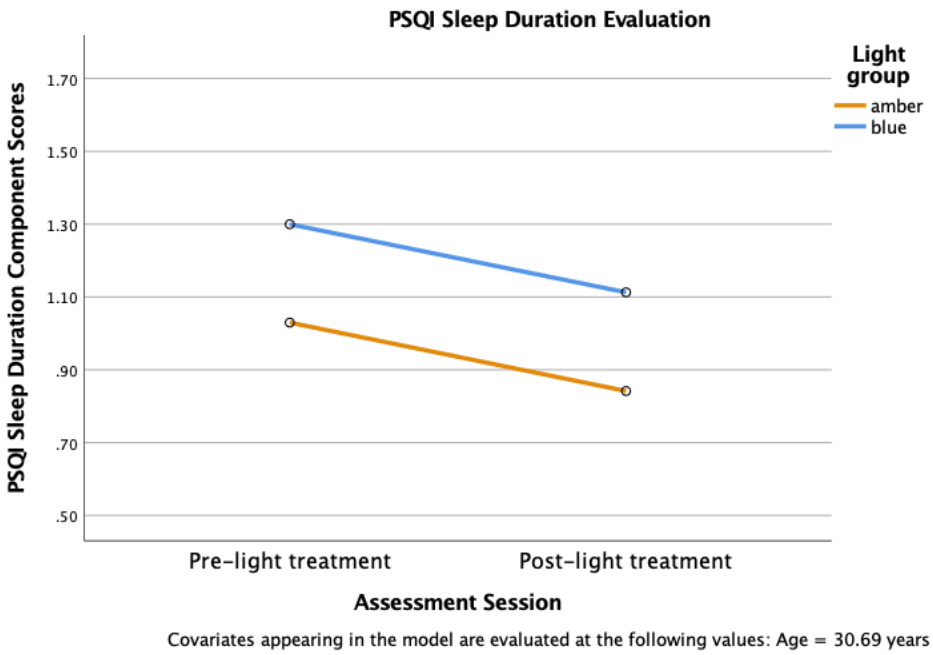
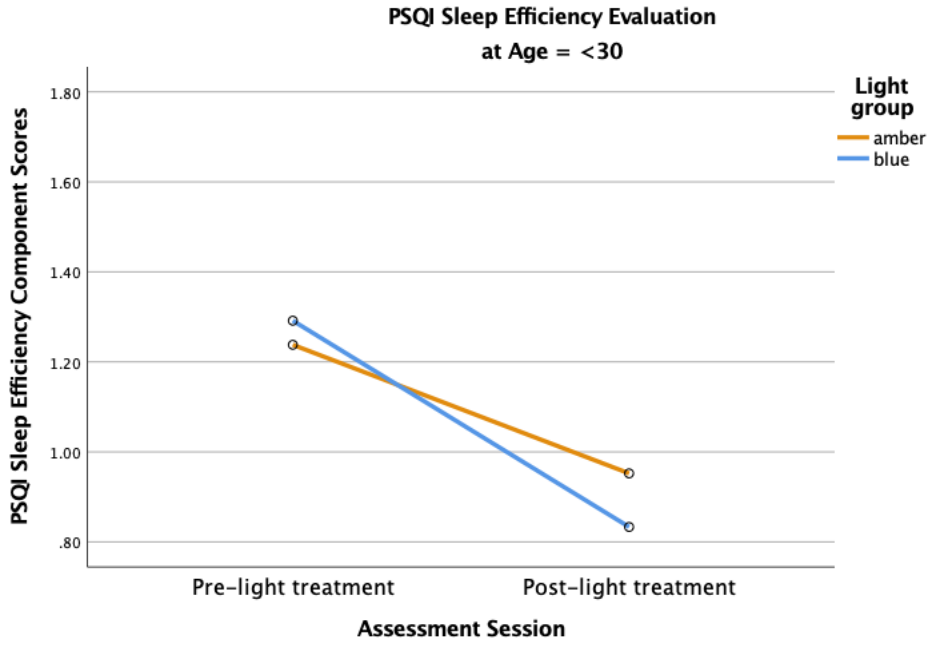


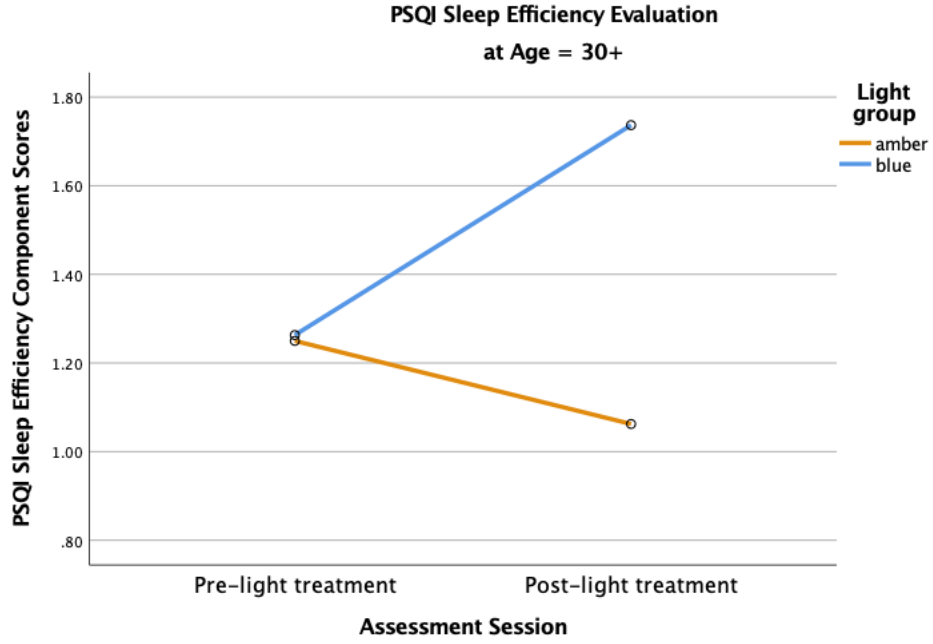
Figure 9 ANCOVA graph for PSQI Sleep Duration Component without consideration for age

Habitual Sleep Efficiency Evaluation

The three-way mixed ANOVA analysis revealed no significant main effects (*at the p < .05 level*) of time, F (1,76) = .602, p = .44, light condition, F (1,76) = .456, p = .501, nor age group, F (1,76) = 1.172, p = .282. Additionally, there were no significant interactions between light condition and age, F (1,76) = .669, p = .416, between time and light condition, F (1,76) = .686, p = .41, between time and age, F (1,76) = 3.049, p = .085, and there was no significant 3-way interaction between time x light condition x age, F (1,76) = 1.997, p = .162. These results indicate that there was not much difference in sleep efficiency component scores over time, therefore suggesting there were minimal changes in sleep efficiency that occurred over the six weeks. The average component scores did not significantly differ depending on age group or light condition. There were no significant interactions between the independent variables on sleep efficiency improvements over time, but there was a trend (p = .085) toward better sleep

efficiency with time in the younger participants. This suggests the younger population may gain more improvements in sleep efficiency over six weeks of light treatment.





Figures 10-11 Three-way mixed ANOVA graphs for PSQI Sleep Efficiency Component

When controlling for age, the analysis revealed a significant main effect (*at the p < .05 level*) of time, F (1,77) = 4.282, p = .042. There were no significant main effects for the light condition, F (1,77) = .334, p = .565, nor age group, F (1,77) = 1.327, p = .253. There were no significant interactions between time and light condition, F (1,77) = .423, p = .517, nor between time and age, F (1,77) = 3.419, p = .068. These results mean there was a decline in sleep efficiency component scores from pre- to post-treatment, regardless of light color and age. This suggests that sleep efficiency improved over the six weeks for each group. The average component scores did not significantly differ depending on age group or light condition. There were no significant interactions of the between-subjects factors on sleep efficiency improvements over time, but there was a trend (p = .068) toward greater improvements in sleep efficiency among the younger population. This suggests that over six weeks of light treatment, the younger participants may gain more improvements in sleep efficiency than the older participants.

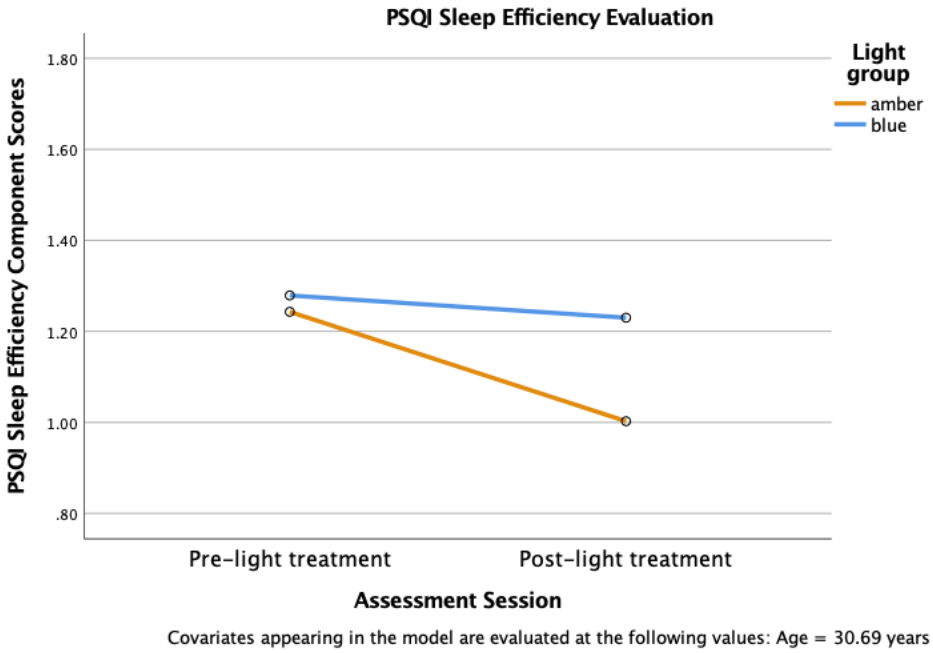
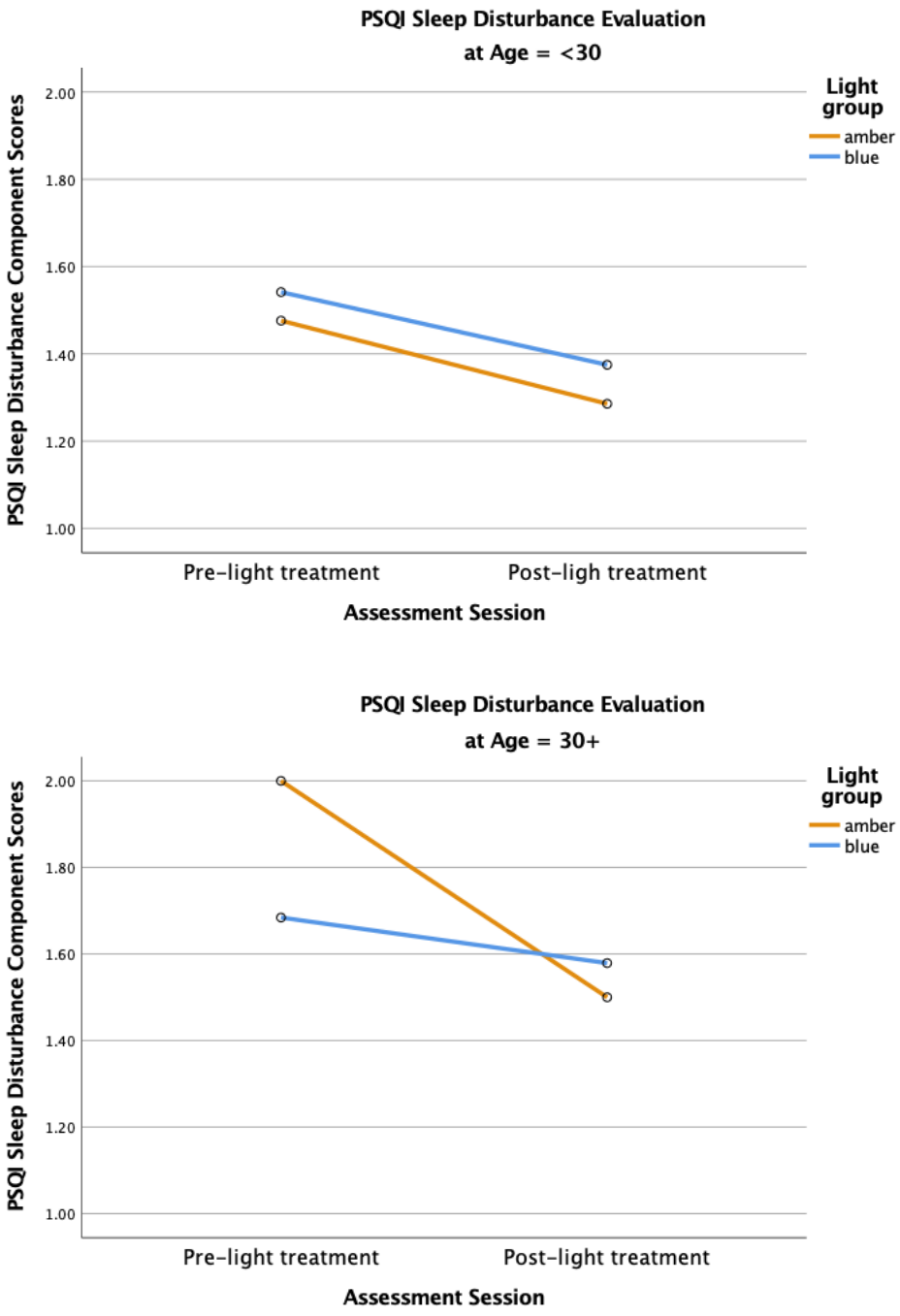


Figure 12 ANCOVA graph for PSQI Sleep Efficiency Component without consideration for age

Sleep Disturbances Evaluation

The three-way mixed ANOVA analysis revealed a significant main effect (*at the $p < .05$ level*) of time, $F(1,76) = 13.063$, $p = <.001$. There was no significant main effect for the light condition, $F(1,76) = .036$, $p = .85$, but there was a significant age group main effect, $F(1,76) = 6.308$, $p = .014$. There were no significant interactions between light condition and age, $F(1,76) = .822$, $p = .367$, between time and light condition, $F(1,76) = 2.471$, $p = .12$, between time and age, $F(1,76) = .868$, $p = .354$, and no significant 3-way interaction between time x light condition x age, $F(1,76) = 1.94$, $p = .168$. These results indicate that there was a decline in sleep disturbance component scores from pre- to post-treatment, regardless of light color and age. This suggests that there was a reduction in sleep disturbances, thus there were improvements in sleep over the six weeks for each group. There was no significant difference in component scores according to light condition, but there was a significant difference based off age group, showing that the younger population generally had less sleep disturbances than the older population. There were no significant interactions between the independent variables on sleep disturbance improvements over time.



Figures 13-14 Three-way mixed ANOVA graphs for PSQI Sleep Disturbance Component

When controlling for age, the analysis revealed no significant main effect (*at the p < .05 level*) of time, F (1,77) = .134, p = .716. There was no significant main effect for the light

condition, F (1,77) = .006, p = .94, but there was a significant age group main effect, F (1,77) = 6.011, p = .016. There were no significant interactions between time and light condition, F (1,77) = 1.956, p = .166, nor between time and age, F (1,77) = .675, p = .414. These results mean there was not much of a difference in sleep disturbance component scores from pre- to post-treatment. The average component scores did not significantly differ depending on light condition, but there was a significant difference in scores based off age. There were lower scores in the younger participants suggesting they experience less disturbances in sleep than the older participants. There were no significant interactions between the independent variables on sleep disturbance improvements over time.

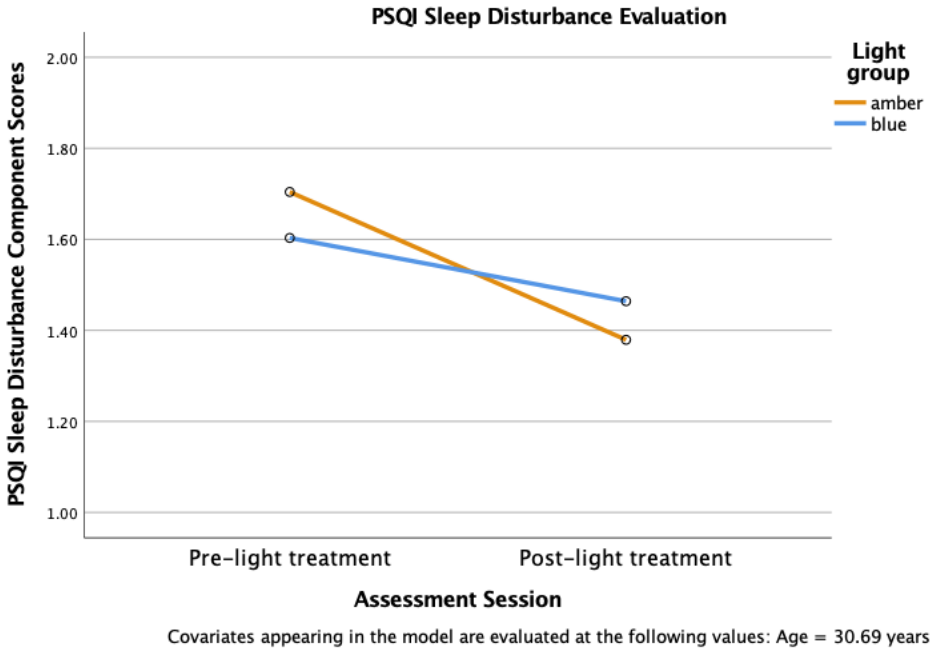
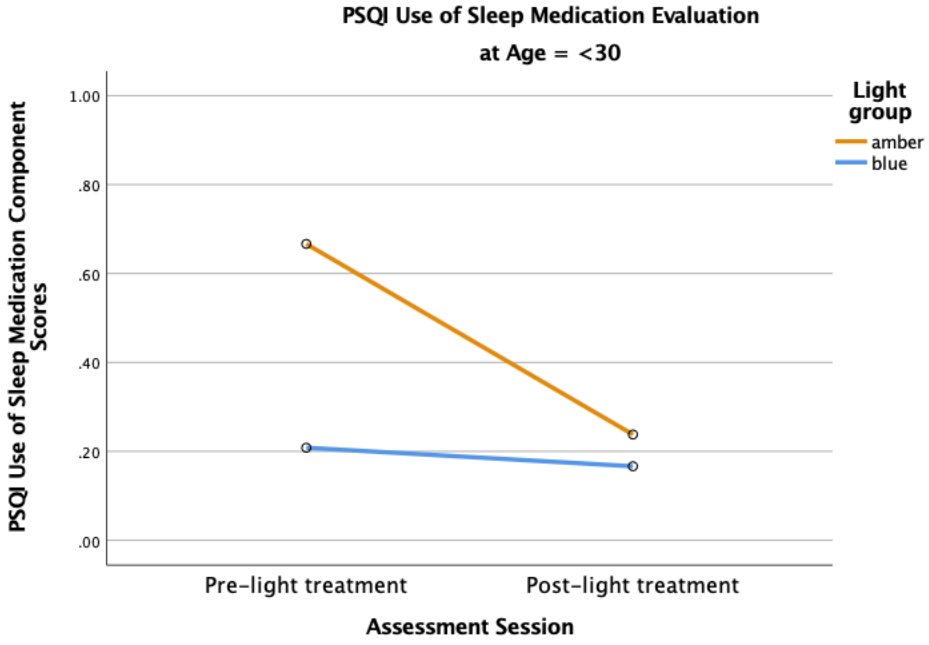
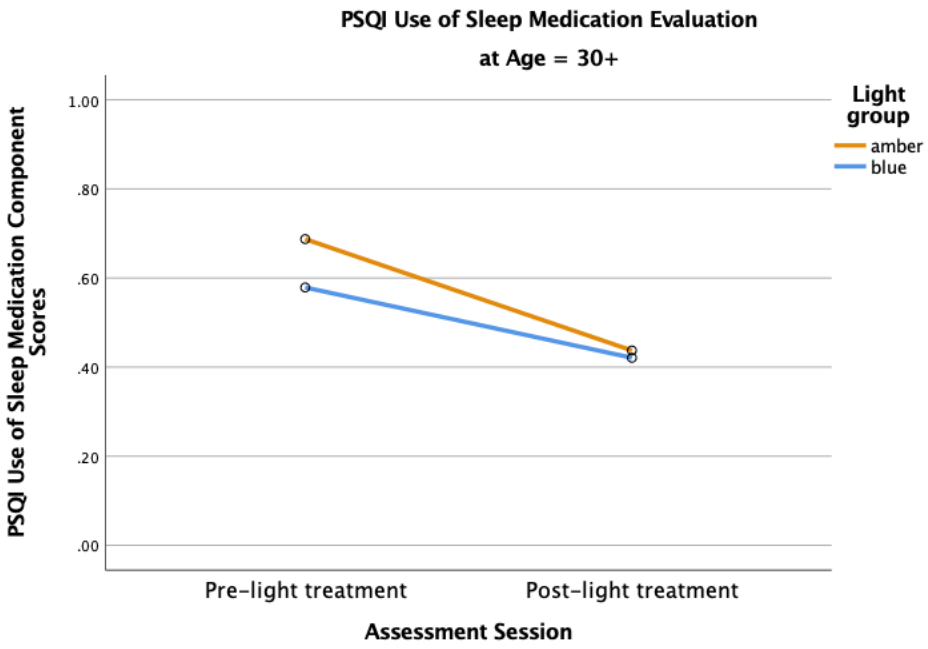


Figure 15 ANCOVA graph for PSQI Sleep Disturbance Component without consideration for age

Use of Sleep Medications Evaluation

The three-way mixed ANOVA analysis revealed a significant main effect (*at the p < .05 level*) of time, F (1,76) = 5.77, p = .019., but there were no significant main effects for the light condition, F (1,76) = .834, p = .364, nor age group, F (1,76) = 1.389, p = .242. There were no significant interactions between light condition and age, F (1,76) = .319, p = .574, between time and light condition, F (1,76) = 1.719, p = .194, between time and age, F (1,76) = .029, p = .865, and there was no significant 3-way interaction between time x light condition x age, F (1,76) = .651, p = .422. These results indicate that there was a decline in the PSQI use of sleep medication component scores from pre- to post-treatment, regardless of light color and age. This suggests that there was a reduction in the need for sleep medications, thus there were improvements in sleep over the six weeks for every group. The average component scores did not differ depending on age group or light condition. There were no significant interactions between the independent variables on sleep medication usage over time.





Figures 16-17 Three-way mixed ANOVA graphs for PSQI Use of Sleep Medication Component

When controlling for age, the analysis revealed no significant main effect (*at the $p < .05$ level*) of time, F (1,77) = .822, p = .367. There was no significant main effect for the light condition, F (1,77) = .994, p = .322, and no significant age group main effect, F (1,77) = 1.517, p = .222. There were no significant interactions between time and light condition, F (1,77) = 2.039, p = .157, nor between time and age, F (1,77) = .012, p = .914. These results mean there was not much difference in PSQI use of sleep medication component scores from pre- to post-treatment when age was used as a continuous variable. The average component scores did not significantly differ depending on age group or light condition. There were no significant interactions between the independent variables on sleep medication usage over time.

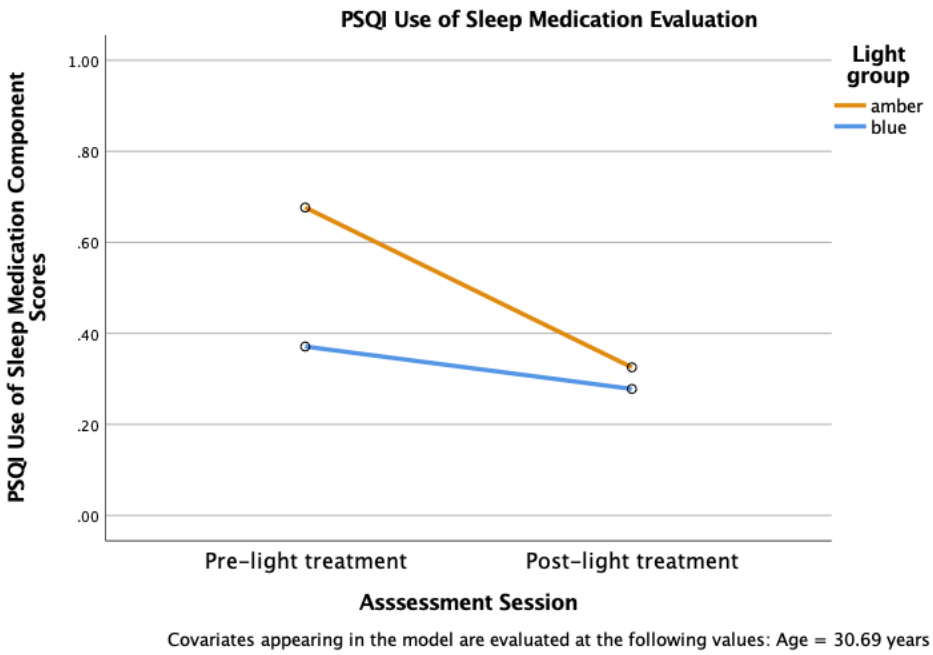
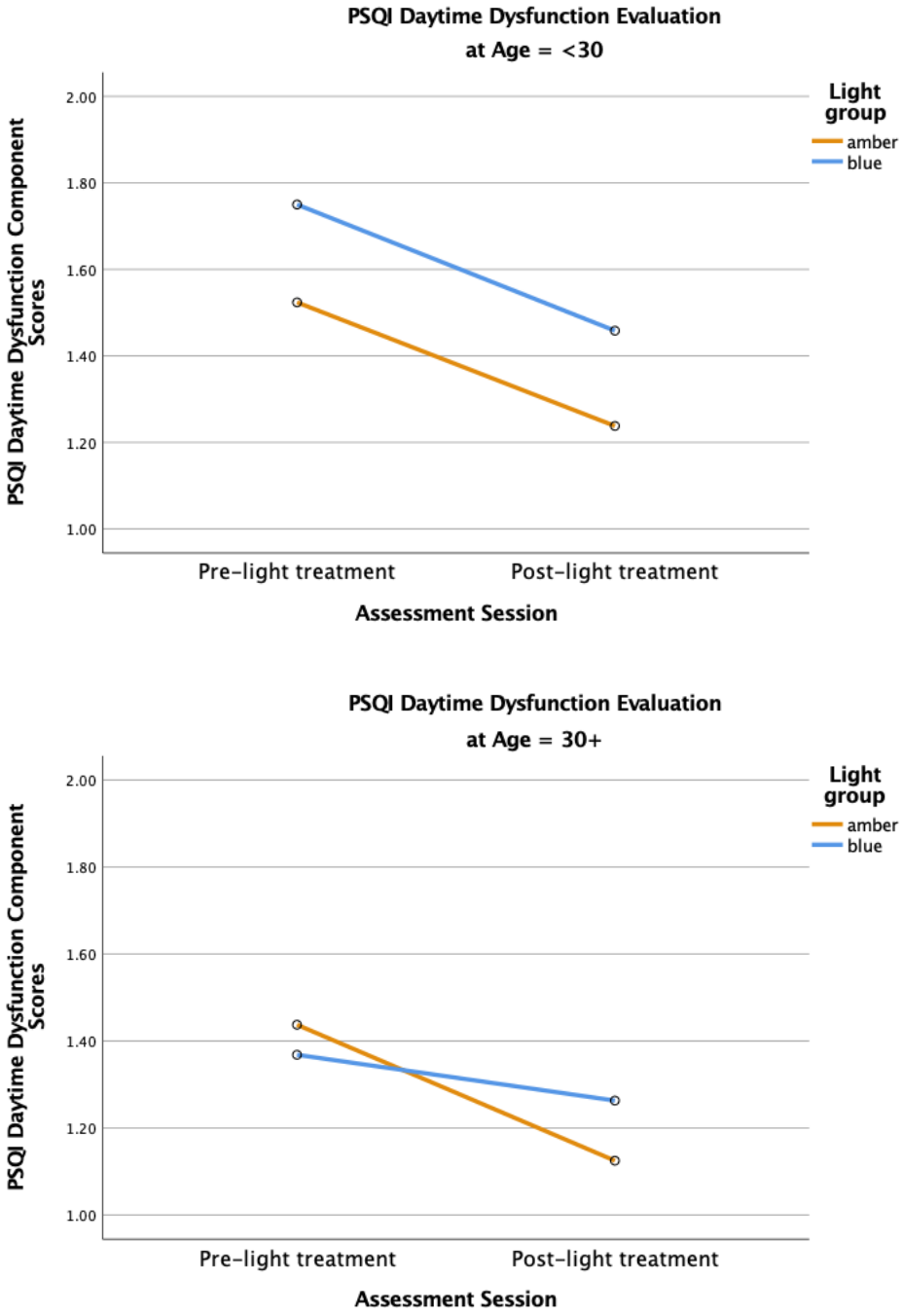


Figure 18 ANCOVA graph for PSQI Use of Sleep Medication Component without consideration for age

Daytime Dysfunction Evaluation

The three-way mixed ANOVA analysis revealed a significant main effect (*at the p < .05 level*) of time, F (1,76) = 10.095, p = .002. There were no significant main effects for the light condition, F (1,76) = 1.007, p = .319, nor for age group, F (1,76) = 2.283, p = .135. There were no significant interactions between light condition and age, F (1,76) = .54, p = .465, between time and light condition, F (1,76) = .413, p = .522, between time and age, F (1,76) = .26, p = .612, and no significant 3-way interaction between time x light condition x age, F (1,76) = .463, p = .498. These results indicate that there was a decline in daytime dysfunction component scores from pre- to post-treatment, regardless of light color and age. This suggests there was a reduction in daytime dysfunction meaning there were improvements in sleep over the six weeks for each group. The average component scores did not differ depending on age group or light

condition. There were no significant interactions between the independent variables on improvements in daytime dysfunction over time.



Figures 19-20 Three-way mixed ANOVA graphs for PSQI Daytime Dysfunction Component

When controlling for age, the analysis revealed no significant main effects (*at the p < .05 level*) of time, F (1,77) = 2.577, p = .113, light condition, F (1,77) = 1.229, p = .271, nor age group, F (1,77) = 2.487, p = .119. There were no significant interactions between time and light condition, F (1,77) = .317, p = .575, nor between time and age, F (1,77) = .32, p = .573. These results mean there was not much difference in daytime dysfunction component scores from pre- to post-treatment. The average component scores did not significantly differ depending on age group or light condition. There were no significant interactions between the independent variables on improvements in daytime dysfunction over time.

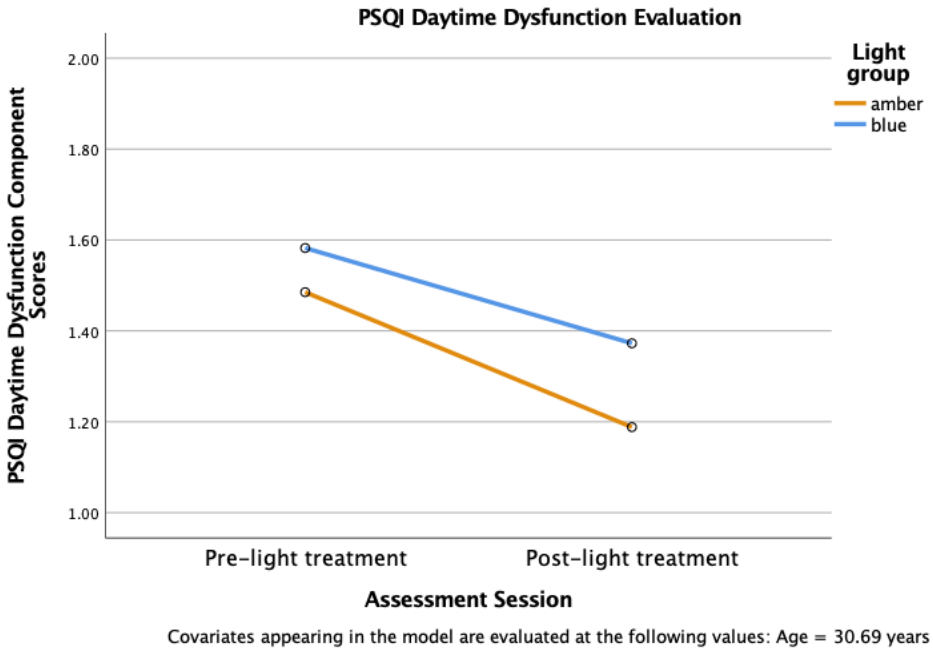
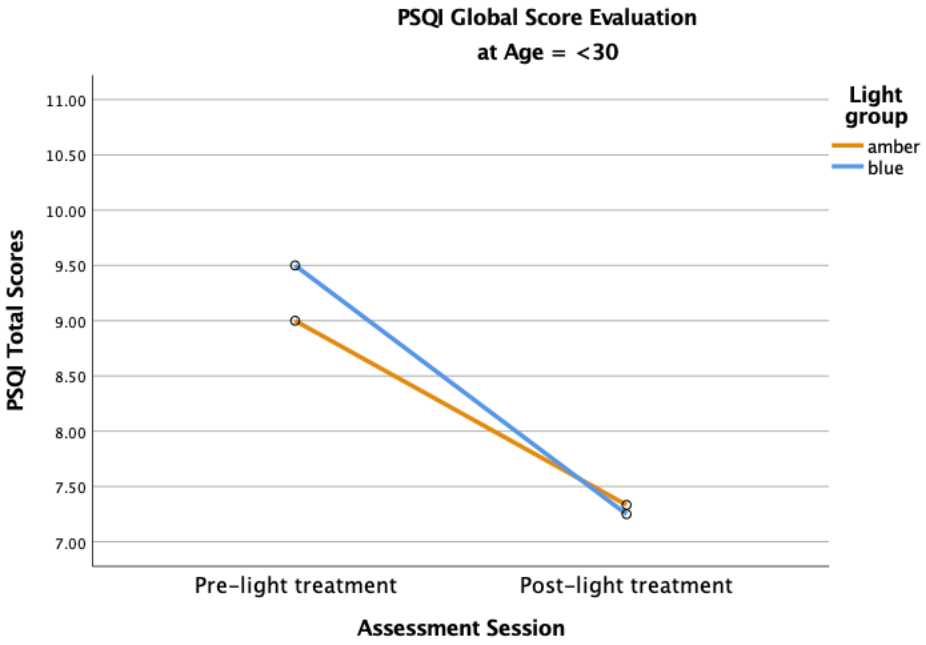
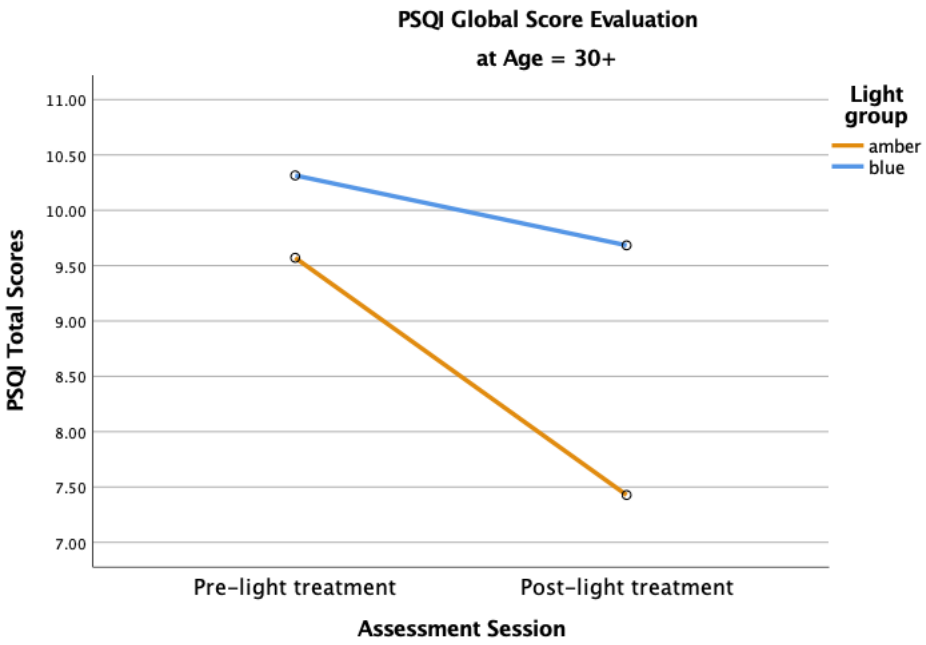


Figure 18 ANCOVA graph for PSQI Daytime Dysfunction Component without consideration for age

Global Evaluation

The three-way mixed ANOVA analysis revealed a significant main effect (*at the p < .05 level*) of time, F (1,74) = 20.78, p = <.001. There were no significant main effects for the light condition, F (1,74) = 1.856, p = .177, nor the age group, F (1,74) = 2.439, p = .123. There were no significant interactions between light condition and age, F (1,74) = 1.061, p = .306, between time and light condition, F (1,74) = .4, p = .529, between time and age, F (1,74) = .606, p = .439, and no significant 3-way interaction between time x light condition x age, F (1,74) = 2.036, p = .158. These results indicate that there was a decline in PSQI total scores from pre- to post-treatment, regardless of light color and age. This suggests that sleep improved over the six weeks for each group. The average PSQI total scores did not differ depending on age group or light condition. There were no significant interactions between the independent variables on sleep improvements over time.





Figures 22-23 Three-way mixed ANOVA graphs for PSQI Global Evaluation

When using age as a covariate, the analysis revealed a significant main effect (*at the p < .05 level*) of time, F (1,75) = 5.886, p = .018. There were no significant main effects for the light condition, F (1,75) = 1.465, p = .23, nor age group, F (1,75) = 2.863, p = .095. There were no significant interactions between time and light condition, F (1,75) = .162, p = .689, nor time and age, F (1,75) = .89, p = .348. These results suggest there was a decline in PSQI total scores from pre- to post-treatment, regardless of light color and age. There were improvements in sleep for every group over the six weeks. The average PSQI total scores did not significantly differ depending on age group or light condition, but there was a trend (p = .095) toward better sleep in the younger participants. This suggests that younger individuals may gain more improvement from light treatment over time than older individuals. There were no significant interactions between the independent variables on sleep improvements over time

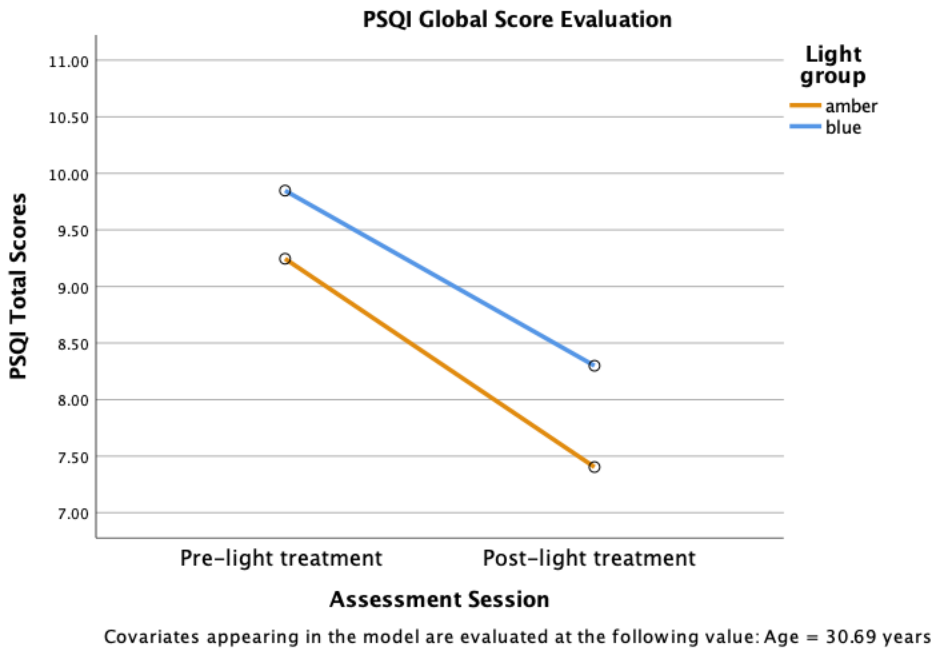


Figure 24 ANCOVA graph for PSQI Global Evaluation without consideration for age

Discussion

Overall, sleep in PTSD patients got better after six weeks of treatment, regardless of light condition and/or age. The results failed to support the hypothesis that blue light would improve overall sleep quality better than amber light and that the younger participants would experience greater improvements from light treatment than the older participants. The findings suggest a significant difference in PSQI total scores from pre- to post-treatment, but type of light treatment and age did not significantly impact this relationship. There was only one component of the PSQI that showed significant differences in scores over time based off light condition, such that blue light significantly improved subjective sleep latency over six weeks of treatment. This is surprising because past research has shown that treatment with blue light was significantly more effective at improving sleep quality in mTBI populations than treatment with amber light (Killgore et al., 2020). However, the present study examined sleep quality according to participants' subjective ratings of sleep, while the findings in previous research obtained

objective measures of sleep quality through actigraphy and other methods. There are a few possible explanations for the findings which will be discussed in turn below.

Perhaps both light treatments were effective and/or the study's scheduled administration of the light therapy improved sleep in PTSD patients. Sleep operates through biological and societal systems that influence critical components of sleep, including the duration and quality of sleep. The circadian rhythm endogenously stimulates sleep, while the structure of our society and behaviors exogenously drive sleep (Foster et al., 2013). Problems in sleep may arise when these systems conflict with each other, and individuals with mental health conditions, such as PTSD, may be more vulnerable to dysregulations in these systems. As previously mentioned, the PTSD population often adopt abnormal sleep behaviors, such as frequent daytime naps and/or excessive sleeping in, which leads to circadian misalignment and leaves them in a vicious cycle of nighttime wakefulness and daytime sleepiness (Richards er al., 2020). There is compelling evidence that suggests that structuring behaviors in individuals with mental illnesses and abnormal sleep/wake cycles improved alignment of sleep patterns with biological and solar time (Foster et., 2013). Maybe administration of the exogenous light source, during a certain time frame, each morning allowed individuals with PTSD set a daily rhythm which ultimately helped realign their circadian rhythms. It could also be that the light therapy unintentionally stimulated another unknown variable which may have caused improvements in sleep. Further research could be conducted to analyze the potential cause for these findings.

There was partial support for the notion that age impacts the extent of artificial light therapy at improving sleep quality over time because there was evidence for interactions between time and age, specifically for sleep quality and sleep efficiency. These findings suggest that the younger individuals experienced greater improvements in these components of sleep over the six

weeks of treatment than the older individuals. The younger population may experience benefits of light therapy to a greater extent than older population. These findings continued to be supported when age was used as a continuous variable in statistical analysis. Furthermore, this suggests that younger individuals may gain more improvement in sleep from light treatment than older individuals. It is possible that the more youthful ocular health permitted light to penetrate the ipRGCs more effectively, thus allowing for more favorable circadian shifts which resulted in better nighttime sleep. It is also possible that the total number of ipRGCs progressively diminishes with age (Mure, 2021). Perhaps the greater improvements in sleep found among the younger individuals is due in part to the higher volume of ipRGCs they possess. Additionally, there was evidence for main effects of age group in some components of the PSQI included sleep duration and sleep disturbances. These findings suggest that the younger individuals experienced more satisfactory durations of sleep and less sleep disturbances than older participants. Therefore, the younger population may have better sleep in general than the older population.

There are a few limitations that should be mentioned. First, the age range of the population under study was chosen to be representative of the ages of military personnel and the typical onset of PTSD. Age-related cataracts often present after 45-55 years of age. The population that comprised the older individuals were primarily in their 30s and likely did not have cataracts that would impede ipRGC stimulation, which could account for the limited age effects seen here. Another element to consider involves participant compliance to light treatment. Since the light treatment phase was completed at home, there is limited control and knowledge of whether participants were following the administration directions that were provided. Participants may not have completed the light treatment as intended. Future research would benefit from the inclusion of some metrics of participants compliance (i.e., treatment fidelity).

Additionally, sleep disruption and sleep quality were self-reported, and these ratings are likely multifactorial in nature, potentially independent complaints that may manifest in multiple ways. It is difficult to clearly define and measure these variables as they differ for everyone. One person's definition of a good night's sleep may not be for another. Future research should aim to fix some of these problems through the use of objective measurements, such as actigraphy or polysomnography.

Conclusions & Future Directions

The data used for this thesis is from the first study that examined the impact of BLT on PTSD symptoms. There is preliminary evidence that suggests light therapy may be a promising intervention for treating sleep problems in the PTSD population, especially for certain sleep complaints including sleep disturbances. The present findings show that six weeks of light treatment improved sleep, regardless of light condition or age. Maybe light therapy itself and/or the daily regularity of light therapy administration are the key factors that led to better sleep. It was also found that younger participants were experiencing more improvements in the overall quality of sleep with light treatment than the older population. This suggests that the younger population may benefit more from light therapy than older populations. Further research should examine the possible reasoning(s) for why this was observed and include participants of a wider age range which consist of people in their 50s, 60s, and possible 70s, who are more likely to have cataracts that would impede light transmission to the retina. Future research could also analyze the role of other variables, such as different wavelengths of light treatment, and assess their abilities at improving sleep.

References

Bisson, J. I., Cosgrove, S., Lewis, C., & Robert, N. P. (2015). Post-traumatic stress disorder. *BMJ (Clinical research ed.)*, *351*, h6161. https://doi.org/10.1136/bmj.h6161

Buysse, D. J., Reynolds, C. F., 3rd, Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry research*, *28*(2), 193–213. https://doi.org/10.1016/0165-1781(89)90047-4

Foster, R. G., Peirson, S. N., Wulff, K., Winnebeck, E., Vetter, C., & Roenneberg, T. (2013). Sleep and circadian rhythm disruption in social jetlag and mental illness. *Progress in molecular biology and translational science*, *119*, 325–346. https://doi.org/10.1016/B978-0-12-396971-2.00011-7

Grandner M. A. (2017). Sleep, Health, and Society. *Sleep medicine clinics*, *12*(1), 1–22. https://doi.org/10.1016/j.jsmc.2016.10.012

Kida S. (2019). Reconsolidation/destabilization, extinction and forgetting of fear memory as therapeutic targets for PTSD. *Psychopharmacology*, *236*(1), 49–57. https://doi.org/10.1007/s00213-018-5086-2

Killgore, W. D., Vanuk, J. R., Shane, B. R., Weber, M., & Bajaj, S. (2020). A randomized, double-blind, placebo-controlled trial of blue wavelength light exposure on sleep and recovery of brain structure, function, and cognition following mild traumatic brain injury. *Neurobiology of Disease*, 134, 104679. doi: 10.1016/j.nbd.2019.104679

Kline, A. C., Cooper, A. A., Rytwinksi, N. K., & Feeny, N. C. (2018). Long-term efficacy of psychotherapy for posttraumatic stress disorder: A meta-analysis of randomized

controlled trials. *Clinical psychology review*, *59*, 30–40. https://doi.org/10.1016/j.cpr.2017.10.009

Koenen, K. C., Ratanatharathorn, A., Ng, L., McLaughlin, K. A., Bromet, E. J., Stein, D. J., Karam, E. G., Meron Ruscio, A., Benjet, C., Scott, K., Atwoli, L., Petukhova, M., Lim, C., Aguilar-Gaxiola, S., Al-Hamzawi, A., Alonso, J., Bunting, B., Ciutan, M., de Girolamo, G., Degenhardt, L., … Kessler, R. C. (2017). Posttraumatic stress disorder in the World Mental Health Surveys. *Psychological medicine*, *47*(13), 2260–2274. https://doi.org/10.1017/S0033291717000708

Lebois, L., Wolff, J. D., & Ressler, K. J. (2016). Neuroimaging genetic approaches to Posttraumatic Stress Disorder. *Experimental neurology*, *284*(Pt B), 141–152. https://doi.org/10.1016/j.expneurol.2016.04.019

Maruani, J., & Geoffroy, P. A. (2019). Bright Light as a Personalized Precision Treatment of Mood Disorders. *Frontiers in Psychiatry*, 10. doi: 10.3389/fpsyt.2019.00085

Miao, X. R., Chen, Q. B., Wei, K., Tao, K. M., & Lu, Z. J. (2018). Posttraumatic stress disorder: from diagnosis to prevention. *Military Medical Research*, *5*(1), 32. https://doi.org/10.1186/s40779-018-0179-0

Miller, K. E., Brownlow, J. A., & Gehrman, P. R. (2020). Sleep in PTSD: treatment approaches and outcomes. *Current opinion in psychology*, *34*, 12–17. https://doi.org/10.1016/j.copsyc.2019.08.017

Mure L. S. (2021). Intrinsically Photosensitive Retinal Ganglion Cells of the Human Retina. *Frontiers in neurology*, *12*, 636330. https://doi.org/10.3389/fneur.2021.636330

Murkar, A., & De Koninck, J. (2018). Consolidative mechanisms of emotional processing in REM sleep and PTSD. *Sleep medicine reviews*, *41*, 173–184. https://doi.org/10.1016/j.smrv.2018.03.001

Neigh, G. N., & Ali, F. F. (2016). Co-morbidity of PTSD and immune system dysfunction: opportunities for treatment. *Current opinion in pharmacology*, *29*, 104–110. https://doi.org/10.1016/j.coph.2016.07.011

Pace-Schott, E. F., Germain, A., & Milad, M. R. (2015). Effects of sleep on memory for conditioned fear and fear extinction. *Psychological bulletin*, *141*(4), 835–857. https://doi.org/10.1037/bul0000014

Richards, A., Kanady, J. C., & Neylan, T. C. (2020). Sleep disturbance in PTSD and other anxiety-related disorders: an updated review of clinical features, physiological characteristics, and psychological and neurobiological mechanisms. *Neuropsychopharmacology: official publication of the American College of Neuropsychopharmacology*, *45*(1), 55–73. https://doi.org/10.1038/s41386-019-0486-5

Shiels, A., & Hejtmancik, J. F. (2019). Biology of Inherited Cataracts and Opportunities for Treatment. *Annual review of vision science*, *5*, 123–149. https://doi.org/10.1146/annurev-vision-091517-034346

Spoormaker, V. I., & Montgomery, P. (2008). Disturbed sleep in post-traumatic stress disorder: secondary symptom or core feature?. *Sleep medicine reviews*, *12*(3), 169–184. https://doi.org/10.1016/j.smrv.2007.08.008

Tempesta, D., Socci, V., De Gennaro, L., & Ferrara, M. (2018). Sleep and emotional processing. *Sleep medicine reviews*, *40*, 183–195. https://doi.org/10.1016/j.smrv.2017.12.005

Tosini, G., Ferguson, I., & Tsubota, K. (2016). Effects of blue light on the circadian system and eye physiology. *Molecular vision*, *22*, 61–72.

Touitou, Y., Reinberg, A., & Touitou, D. (2017). Association between light at night, melatonin secretion, sleep deprivation, and the internal clock: Health impacts and mechanisms of circadian disruption. *Life sciences*, *173*, 94–106. https://doi.org/10.1016/j.lfs.2017.02.008

Wahl, S., Engelhardt, M., Schaupp, P., Lappe, C., & Ivanov, I. V. (2019). The inner clock - Blue light sets the human rhythm. *Journal of biophotonics*, *12*(12), e201900102. https://doi.org/10.1002/jbio.201900102

Wirz-Justice, A., Skene, D. J., & Münch, M. (2021). The relevance of daylight for humans. *Biochemical pharmacology*, *191*, 114304. https://doi.org/10.1016/j.bcp.2020.114304