The Impact of Sleep Disturbances and Sleep Debt on Emotion Regulation and Reactivity in Obstructive Sleep Apnea Patients

The Impact of Sleep Disturbances and Sleep Debt on Emotion Regulation and Reactivity in Obstructive Sleep Apnea Patients

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Abstract

Sleep debt and disturbances are a growing problem worldwide, and the ramifications are unavoidable and detrimental to one's physical health, mental health, and safety. Individuals diagnosed with Obstructive Sleep Apnea (OSA) are suffering from sleep fragmentation that presents with detriments comparable to those that are sleep deprived. Characterized by its frequent arousals from sleep caused by respiratory flow limitation, OSA is known to have an impact on an individual's emotional wellbeing, however, the mechanism behind this has remained largely unknown. This study analyzed changes in sleep architecture and emotion regulation and reactivity before and after OSA treatment. Sleep architecture was analyzed to determine change in OSA severity (defined by the apnea-hypopnea-index), duration of time spent in Stage 3 sleep, duration of time spent in Rapid-Eye-Movement stage sleep, and Total Sleep Duration. Emotion regulation and reactivity was analyzed to determine change in three prominent emotion questionnaires: The Hospital Anxiety and Depression Scale, The Cognitive Emotion Regulation Questionnaire, and the Profile of Mood States Questionnaire. Paired-sample t-tests, calculated in R-Studio, determined a relationship between change in AHI and Stage 3 sleep with improvement in emotion regulation and reactivity scores. The linear regression analysis showed no significant correlation in the amount of change in sleep architecture with the amount of change in emotion scores. This investigation provides evidence that there is a significant relationship between improvements in sleep architecture and enhancements in emotion regulation and reactivity.

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Chapter I.

Introduction

Sleep is one of the most critical aspects of human life. Shown to be an essential component of health, the duration and quality of sleep can impact both physical and emotional well-being, along with the quality of life of individuals. The World Health Organization continues to emphasize the importance of sustaining the required amount and sufficient quality of sleep on a nightly basis (Fox, 1999). The American Academy of Sleep Medicine and the Sleep Research Society recommend that individuals maintain at least 7 hours of good-quality sleep each night to promote optimal physical and mental health (Giles, 2016). The CDC, however, has determined that at least one third of the American population are not getting this sleep on a regular basis (Giles, 2016).

Sleep disturbances, and associated debt, are problems that evidently plague a widespread community of people. In adolescence, chronic sleep loss, and especially its associated daytime impairments due to sleepiness, pose a threat to the academic success, health, and safety of our youth. For this population, much of the blame falls on environmental and social factors that can, and should, be addressed by making behavioral changes. Regardless of cause, however, this indication of insufficient sleep has been linked to alarming consequences such as motor vehicle accidents, delinquent behaviors, mental health diagnoses and psychological stress (Owens, 2014). For the adult population, however, it is more often seen that the cause of sleeplessness is a combination of both environmental and physical health factors (Chattu et al., 2018).

It has been made clear that from adolescents to adults, it is not uncommon to come across an individual that suffers from significant detriments due to insufficient sleep duration or quality. The damaging effects of poor sleep can be witnessed from a physical and mental standpoint and undoubtedly have a severe impact on the quality of one's life.

Over the years, the scientific community has begun to understand the emotional impact that sleep disturbances and sleep debt have on a person. With consistently increasing rates of Anxiety, Depression, and other mental health disorders, the understanding of sleep's role in this global problem is a crucial mission to take on. A clear association has been found between reduced sleep and psychosocial factors such as tension, anxiety, and depressive symptoms. In adolescents, it is suggested that chronic sleep restriction can increase the likelihood of consequences as grave as suicide (Chattu et al., 2018). Through vast research experiments, it has become clear that sleep deprivation, whether it be partial or total, lies at the forefront of reasoning behind the increased prevalence of emotion regulation and reactivity problems.

Sleep disorders, such as Obstructive Sleep Apnea (OSA), continue to be a leading cause in restricted sleep and chronic sleep deprivation. Such disorders have now also been demonstrated to closely correlate with mental health diagnoses such as Anxiety and Depression. Understanding the mechanism behind the emotional dysregulation commonly comorbid with OSA could allow us insight into ways that we could improve the quality of life of the many individuals suffering. The information learned from this population can also be generalized to those without this diagnosis.

To acquire a diagnosis of OSA, one must undergo extensive sleep monitoring that observes details such as sleep architecture and other physiological parameters. The measurement of these parameters is currently unique to a sleep disorder diagnosis, however, can be crucial in uncovering the mechanisms behind the disruption in emotion regulation and reactivity.

Obstructive Sleep Apnea is characterized by its frequent arousals from sleep caused by respiratory flow limitation and oxygen desaturation. These arousals cause severe sleep fragmentation and significant loss of time spent in the most restorative stages of sleep, Stage 3, and the Rapid-Eye-Movement (REM) stage. These sleep disruptions, caused by OSA, could be the reasoning behind the common presentation of emotion regulation and reactivity dysfunction in OSA patients. Akberzie, W., et al. showed a high prevalence of co-morbidity between Anxiety and Depression in those diagnosed with OSA. In this study, 62.2% of OSA patients were also positive for Anxiety and 64.4% of OSA patients were also positive for Depression (Akberzie, W., et al., 2020).

The aim of this research project is to understand whether improvement of sleep in OSA patients could improve the presentation of mental health difficulties and disorders. The question remains as to the impact that sleep fragmentation has on emotional regulation and reactivity. This project goes further to address whether poor sleep architecture, more specifically the insufficient duration in Stage 3 and REM stage sleep, also plays a role in these emotional disruptions. The hypothesis is that if an OSA patient is treated so that their severity of OSA, quantified by the Apnea-Hypopnea Index (AHI),

is decreased and their sleep architecture is improved, their overall emotion regulation and reactivity would be improved, as well.

To determine if there is a correlation present, I studied OSA patients before and after treatment that alters their AHI and sleep architecture. All patients underwent polysomnographic (PSG) testing with advanced physiological measures to report sleep stages, flow limitation, oxygen desaturation and respiratory efforts. At the time of PSG testing, patient's emotion regulation and reactivity were recorded using objective and subjective measurements. These measures were taken at a baseline visit, and the subsequent treatment visit. The measures taken at treatment visits were compared back to the baseline visit. Through polysomnographic scoring, the severity of OSA was determined by quantifying the AHI. The amount of time spent in each sleep stage was also reported. These measurements were correlated with the results of the emotion regulation and reactivity measurements to determine if there was a relationship present.

By quantifying these sleep disturbance measurements and associating them with objective and subjective measures of emotion regulation and reactivity, I am able to shed light on how partial sleep deprivation is impacting our emotions in conditions like Obstructive Sleep Apnea. The significance of this data can also be utilized to understand people with other disorders like Periodic Limb Movement Disorder, or even people with generalized sleep deprivation due to environmental or social factors. The results of this project may also provide clinicians with a method to encourage patients to persevere with the often-intolerable treatments for OSA. If patients believe they will better regulate their emotions after treatment, they may be more inclined to put effort into accommodating to the gold-standard OSA treatment, continuous positive air pressure (CPAP).

Improvement of emotional wellbeing and quality of life continues to be a priority for the scientific community as the stress of current lifestyles continues to increase. Any information that can be gathered on the topic is essential. It is evident that sleep plays a critical role in this subject, and there is still an abundance of research that must be done to understand the true mechanism behind this relationship.

Definition of Terms

"Anxiogenic": producing anxiety.

"Apnea-Hypopnea-Index" (AHI): the sum of the number of apneas and hypopneas that occurs, on average, each hour.

"Apnea": complete airway obstruction; a pause in breathing.

"Arousal": interruption of sleep lasting 3 to 15 seconds; being brought to a lighter stage of sleep, often times, without realizing it.

"The Cognitive Emotion Regulation Questionnaire" (CERQ): A multidimensional questionnaire constructed in order to identify the cognitive emotion regulation strategies someone uses after having experienced negative events or situations. The questions are divided into those pertaining to the use of "Less Effective" mechanisms to deal with stressors and those pertaining to the use of "Most Effective" mechanisms to deal with stressors. A person is given a score pertaining to each of the two categories.

"Continuous Positive Air Pressure" (CPAP)Machine: a machine that increases air pressure in one's upper airway to avoid collapse and obstruction during sleep.

"Electroencephalography" (EEG): a monitoring method to record electrical activity of the brain using noninvasive electrodes pasted onto the scalp and face.

"Emotional Reactivity": the tendency to experience frequent and intense emotional arousal.

"Emotional Regulation": the ability to exert control over one's own emotional state.

"Flow Limitation": a decrease in air flow that remains constant despite an increase in respiratory effort.

"The Hospital Anxiety and Depression Scale" (HADS): a questionnaire used to measure anxiety and depression in a general population of patients.

"Hypopnea": abnormally slow or shallow breathing; breathing with flow limitation.

"Negative Affect": one's propensity to experience negative emotions.

"Non-Restorative Sleep": the subjective feeling that sleep has been insufficiently refreshing.

"Obstructive Sleep Apnea": when the muscles that support the soft tissues in the throat, such as the tongue and soft palate, temporarily lose muscle tone. This loss in muscle tone causes the airway to narrow or close obstructing one's breathing.

"Oxygen Desaturation": in sleep monitoring, this refers to a decrease in blood oxygen saturation levels by 3%.

"Partial Sleep Deprivation": the reduction in total sleep time relative to one's usual baseline during a 24-hour period.

"Polysomnography" (PSG): the technique or process of using a polygraph to make a continuous record during sleep of multiple physiological variables such as breathing, heart rate, muscle activity, or brain activity.

“The Profile of Mood States Questionnaire” (POMS):. a questionnaire containing statements that describe the feelings people have within a certain time period. Used to analyze total mood disturbance.

“Positive Affect”: one’s propensity to experience positive emotions.

“Sleep Architecture”: the basic structural organization of normal sleep into non-rapid-eye-movement (NREM) and rapid-eye-movement (REM) sleep. NREM sleep is divided into stages 1, 2, and 3.

“Sleep Debt”: the cumulative effect of a person not having sufficient sleep; sleep deficit.

“Sleep Disturbance”: disorders of initiating and maintaining sleep and dysfunctions associated with sleep, sleep stages, or partial arousals.

“Sleep Duration”: the amount of time clinically scored as a sleep stage out of the total time spent in bed after lights out.

“Sleep Fragmentation”: repetitive short interruptions of sleep.

“Sleep Stage, Stage 3”: the deepest period of NREM sleep, thought to be responsible for the restorative functions of sleep.

“Sleep Stage, Rapid-Eye-Movement (REM)”: stage of sleep associated with dreaming and brain restoration; eyes move rapidly in a range of directions, but all other muscles are paralyzed.

“Total Sleep Duration”: The sum of the number of minutes spent in each sleep stage during the overnight visit.

“Total Sleep Deprivation”: the avoidance of sleep for a period of at least one night.

Background of the Problem

Sleep continues to be a crucial component of development, overall physical health, and mental wellbeing. Sufficient sleep is determined by several factors including duration, quality, and the absence of disorders or external factors that may disrupt any of the aforementioned categories. The American Academy of Sleep Medicine along with the Sleep Research Society provide us with guidelines for the amount of sleep needed based on age groups. Under said guidelines, sufficient sleep duration is between 8 and 10 hours for adolescents and greater than 7 hours for adults (Chaput et al., 2018). By adhering to these guidelines, individuals are preserving their physical and emotional well-being, as long as the quality of sleep is sufficient. Over the past decade, it has become more common for individuals to deprive themselves of the sleep that research demonstrates is so critical. This sleep deprivation comes in many forms and at the fault of many factors, however, the consequence of the deprivation is just as grave.

Causes of Insufficient Sleep

There are a variety of factors that contribute to the prevalence of insufficient sleep that is widespread globally in the current times. The cause of insufficient sleep and subsequent sleep debt build up are often a combination of extrinsic and intrinsic factors and may differ based on age group. In both adults and adolescents, insufficient sleep has been linked to extrinsic factors such as increased artificial light from electronic use, increase in caffeine consumption, lack of physical activity, and increase in responsibilities both socially and at school or work (Chaput et al., 2018). In adults, the increased needs in a family setting also contribute to the propensity of insufficient sleep.

As responsibilities increase in adulthood, the pressure to maintain one's lifestyle at the sacrifice of their sleep increases, as well.

In adolescents, intrinsic factors such as puberty can be a contributing factor to sleep insufficiency. Puberty is associated with a shift toward an evening chronotype which counteracts with the early awakenings due to school start time. This conflict between going to sleep later (due to biological drive after puberty) and waking up early (due to academic commitment) is a major factor contributing to sleep debt accumulation (Chaput et al., 2018).

In adults, intrinsic factors tend to be more associated with sleep disorders and changes in sleep patterns due to aging. As individuals age, it is more common to see difficulties in falling asleep and staying asleep causing a rise in insomnia and sleep maintenance difficulties. There is also an increase in other sleep disorders such as Obstructive Sleep Apnea and Periodic Limb Movement Disorder due to physical health changes that are commonly associated with aging (Chaput et al., 2018). Particularly for women, the post-menopausal hormonal changes experienced also greatly impact sleeping patterns and tendencies.

Though many feel that they can survive sleeping less than the recommended duration of time, there is inevitable sleep debt that is building beneath the surface and there will come a time when caffeine or adrenaline cannot compensate for this. The accumulation of sleep debt, despite causation of extrinsic or intrinsic factors, can detrimentally impact a person's physical health, mental health, and safety.

Sleep Debt

Sleep debt, the result of total or partial sleep deprivation, is a pervasive and amplifying problem in both adults and adolescents across the world. An average of 20% of people between the ages of 25 and 45 are getting at least 90 minutes less than what is deemed necessary by healthcare providers (Hanson, J., 2020). Unfortunately, research has gone further to show that returning to proper function after accumulating sleep debt takes more work than one may think. It takes several nights of appropriate sleep duration (i.e. 7 or more hours) to return to baseline cognitive function after just one night of poor sleep (Sallinen, M., et. al., 2008). According to the American Sleep Society, researchers have yet to agree on whether or not sleep debt is a measurable phenomenon; what has been confidently concluded, however, is that sleep debt, brought on by both chronic and acute sleep deprivation, has been known to lead to an array of physical and mental health problems.

The role of sleep deprivation in metabolic malfunctions has been a heavily researched topic for many years. The accumulation of sleep debt caused by chronic partial sleep deprivation has been shown to increase the risk of obesity and diabetes. Though the exact mechanism has not yet been fully elucidated, sleep deprivation has shown to detrimentally alter glucose regulation and insulin resistance, cause a dysregulation of neuroendocrine appetite control leading to increased food intake, and subsequently decrease energy expenditure. These findings have been further researched from an epidemiological approach, which largely supports the mechanisms uncovered in the laboratory (Knutson, K.L., et.al., 2007).

Over the past decade, researchers and the scientific community have identified places in which sleep deprivation is widespread. The Intensive Care Unit (ICU) of hospitals has represented a more severe display of how sleep deprivation can impact bodily functions, and, more specifically in this case, the healing of those suffering from critical illnesses. Sleep disruptions are frequent in hospital units as busy as the ICU, and the damages, both physical and mental, are under heavy examination with hopes that if sleep is improved in these units, the patient's health will follow (Kamdar, B.B, et.al., 2012). However, these detriments of sleep disruption can be witnessed beyond just the critically ill. The physical and mental malfunctions that are witnessed after accruing sleep debt can comparatively be seen in school children, students, shift-work employees, and several other populations. Across all age groups, the physical and mental problems associated with sleep debt are present at comparative levels. Furthermore, the detriments of fatigue and mood have been demonstrated to correlate directly with sleep duration (Oginska, H., & Pokorski, J., 2006).

Contemporary research has continued to place focus on the evidence of sleep deprivation leading to fluctuations in mood and emotion regulation and reactivity. Evidence suggests that there is an increase of negative emotional affect and decrease of positive emotional affect associated with sleep deprivation (Babson, K., et.al., 2010). The most predominant example of an increase in this negative affect, and a heavily correlated health problem associated with sleep deprivation, is anxiety and related behaviors. Impacting approximately 29% of the US adult population yearly, anxiety disorders are the most common mental illness country wide (Bostwick, W., et.al., 2010). Sleep deprivation has been proven to have an anxiogenic effect on humans, worsening those

already diagnosed with an anxiety disorder, and manifesting a disorder in those with no prior diagnosis.

Research involving the amygdala, due to its commanding presence in the anxiety-response circuit, has been fundamental to understanding the effects of sleep deprivation. Contemporary research is starting to understand how the amygdala and other brain regions play a role in brain activity after sleep deprivation with compelling signs of disrupted regulation of this major emotion circuit.

Emotion Regulation and Reactivity

Sleep deprivation and disturbances have clear implications in emotional regulation and emotional reactivity. Since researchers began understanding the effects of sleep deprivation, anxiety-like behaviors have become widely accepted as a common and detrimental side effect. This side effect of insufficient sleep has been demonstrated in both rodents and human models.

In mouse studies, evidence indicated that sleep deprivation had an anxiogenic effect after 72 hours. This anxiety-like behavior manifested itself in tasks such as the elevated plus-maze task and the open-field arena task. After 72 hours of sleep deprivation, these mice spent a lesser amount of time in the open arms of the elevated plus-maze task and had less locomotion into the center of the open field task, as compared to controls. When these tasks were performed after 24 hours of sleep deprivation, the sleep deprived mice showed no significant difference in movement when comparing to controls (Silva, R., et.al., 2004). These results suggest that the anxiety-like behavior is dependent on the severity of sleep deprivation, however, it is clear that it is directly correlated.

The first human studies that suggested sleep deprivation may be linked with anxiety-like behavior was not a study of total sleep deprivation, rather it was deprivation of Rapid Eye Movement (REM) sleep. Human subjects were deprived of the REM stage of sleep for a range of 2 to 10 nights consecutively. The subjects involved in this study reported a range of psychological disturbances that developed during the period of REM-sleep deprivation such as anxiety, irritability, and difficulty concentrating. The more severe side effects, causing subjects to withdraw from the study, were prominently anxiety related. One subject withdrew due to panic attacks, two subjects withdrew because the stress was too great and a fourth subject withdrew due to severe anxiety and agitation (Dement, W., 1960). This study was the first indicator that sleep debt, even due to partial sleep deprivation, can cause significant increase in anxiety-like behaviors.

When researchers started to investigate total sleep deprivation in humans, the correlation of sleep debt to induction of abnormal emotional behaviors was only further demonstrated. After a 36-hour period of wakefulness, both self-reported and task-related anxiety levels were increased. The subjective anxiety level was measured using the State-Trait Anxiety Inventory (STAI) questionnaire. The Stoop Task, in three forms – Color-Word, Emotional and Specific, was also used to measure task-related anxiety levels. There was a correlated increase in the scores of the STAI questionnaire, and the Emotional Stroop Task, focused around threatening versus neutral words, showed an increased reactivity (Sagaspe, P., et.al., 2005).

Another study on human subjects showed anxiety behavior from a physiological measurement after acute sleep deprivation. The anxiety behavior was monitored using the Mood and Anxiety Symptom Questionnaire (MASQ) indexes which focused on

physiological arousal such as: racing heart, easily startled, shaky, and twitching muscles. These indexes increased in subjects that were sleep deprived for one night and decreased in control subjects (Babson, K., et.al., 2010).

From mouse models to human, it is clear that sleep deprivation commonly and reliably induces irregular emotional reactions, though research has yet to understand the true mechanisms of impact of sleep-related disorders in this paradigm. With continued studies of populations with partial and total sleep deprivation, it is inevitable that the mechanisms at play will come to light, and the cause of the detriments will be fully revealed.

Obstructive Sleep Apnea

Obstructive Sleep Apnea (OSA) is a disorder characterized by obstruction, or collapse, of the upper airway during sleep. This obstruction can be partial, a hypopnea, or complete, an apnea. Both types of obstruction cause a limitation in airflow which often subsequently causes a decrease in oxygen saturation in the blood. The lack of airflow, and in some cases the oxygen desaturation, causes an arousal or awakening in someone suffering from this disorder. These respiratory arousals are quantified per hour and referred to as the patient's AHI or, apnea-hypopnea-index. The AHI can range anywhere from 5 events to over 100 events per hour, and this value is what determines the severity of the diagnosis. Any AHI that is below 15 is considered a mild diagnosis of OSA. An AHI that falls within the range of 15 and 30 is considered a moderate diagnosis of OSA. Any AHI greater than 30 is considered a severe diagnosis of OSA. Though advantageous to our survival, these arousals associated with this disorder cause significant sleep fragmentation and often non-restorative sleep.

Sleep fragmentation seen in OSA patients is often discussed as a source of partial sleep deprivation. It is also common to see a deficit in Stage 3 and Rapid-Eye-Movement (REM) sleep in those suffering with OSA. These disturbances of specific sleep stages are commonly referred to as poor or disrupted sleep architecture. For several years, research has demonstrated that it is Stage 3 and REM stage sleep that is responsible for much of the restorative functions that occur during sleep. Being deprived of these crucial stages could be at the root of emotional dysregulation and disrupted reactivity.

Due to increasing rates of obesity and improvements in diagnosis and public knowledge, the prevalence of OSA is on the rise. In eleven epidemiological studies published between 1993 and 2013, there was evidence of diagnosed OSA in a mean of 22% of men and 17% of women (Franklin, K. A., & Lindberg, E., 2015). Due to the ambiguity of the disorder, it is likely that there is a large majority of people that remain undiagnosed. Often times people overlook the subjective symptomology such as daytime sleepiness and will only visit a physician for diagnosis after a bedpartner witnesses the more significant night-time symptoms such as pauses in breathing or disruptive snoring. Unfortunately, despite a formal diagnosis, some still choose to remain untreated. Approximately 50% of OSA patients cannot tolerate the gold-standard treatment of CPAP and, thus, would prefer to remain untreated.

The majority of OSA research to date has been focused on the cardiovascular and neurological detriments that are often thought to be caused by this disorder. Stroke, hypertension, and coronary artery disease are all highly associated with OSA as seen in several longitudinal studies (Franklin, K. A., & Lindberg, E., 2015). Though this diagnosis is much more prevalent in men, there are more complicated risk factors that

play into the prevalence of OSA in women. Women diagnosed with OSA are typically of the post-menopausal age or suffering from hormonal imbalances such as androgen excess in Polycystic Ovary Syndrome (PCOS) (Fogel, R.B., et.al., 2001). Obesity, however, remains to be the greatest risk factor in developing OSA at any age and of any gender (Franklin, K. A., & Lindberg, E., 2015). It is also seen that physical structure (i.e., a narrow upper airway or facial structure) and inherited genes play a role in developing this disorder.

The significant sleep fragmentation caused by frequent arousals, especially seen in moderate-to-severe cases of OSA, takes away from the total sleep time of those suffering. It is only natural to look at these sleep disruptions as a form of deprivation, and the consequential deficits have been compared in research with other forms of sleep deprivation. In a 2001 study, researchers from Oxford compared the effects of sleep deprivation (subjects deprived of one night's sleep), alcohol intoxication (subjects with blood alcohol level of 71.6 mg/dl) and untreated Obstructive Sleep Apnea. The participants were asked to complete a 90-minute driving simulation and, not surprisingly, there was clear evidence of cognitive impairment in all three subcategories of subjects. What proved interesting was that subjects with untreated, moderate-to-severe OSA were found to have greater cognitive difficulties than those sleep deprived, but not quite as bad as subjects under the influence of alcohol. The subjects with untreated OSA had a larger standard deviation, meaning they scored with a greater range of difficulty, compared to those sleep deprived; however, the mean value came closer to those with sleep deprivation (Hack, M.A., et. al, 2001). With this information, along with other research

studies that have been executed over the past decade, it is within scientific reason to compare the untreated OSA subcategory to those suffering from sleep deprivation.

Utilizing the aforementioned comparative research as a foundation, it is logical to look for the detriments of sleep deprived subjects in those with untreated OSA. Research has yet to link the concept of sleep debt from untreated OSA, and its associated sleep disturbances, to emotion regulation and reactivity dysfunction. Already it has been demonstrated that Depression and Anxiety have a high comorbidity rate with OSA, however, the mechanism behind this has yet to be determined. It is crucial to not only look at the number of arousals, but also at the lack of time spent in particular, restorative sleep stages due to OSA. In this research, the amount of time spent in each sleep stage will be quantified and compared with and without treatment, therefore, a correlation, if present, can be uncovered. It is important to look into the impact of improved sleep, defined by improved sleep architecture, and less sleep disruption, on the emotion regulation and reactivity of those diagnosed with Obstructive Sleep Apnea.

Question and Hypothesis

The aim of this research project is to understand whether the improvement of sleep in Obstructive Sleep Apnea patients could improve the emotion regulation and reactivity of patients manifesting as symptoms of mental health disorders. The question remains as to the impact that sleep fragmentation has on emotion regulation and reactivity, and the mechanism behind this. This project also goes further to address whether poor sleep architecture, more specifically the insufficient duration in Stage 3 and

REM stage sleep, also plays a role in these disruptions in emotion regulation and reactivity.

The hypothesis is that if an OSA patient is treated so that their severity of OSA is decreased, and their sleep architecture is also improved, their overall emotion regulation and reactivity would be improved, as well. This would be demonstrated as an initial improvement in apnea-hypopnea-index (AHI) and an increase in duration of time in Stage 3 and REM stage sleep, along with a subsequent improvement in emotion regulation and reactivity questionnaires.

Implication of Research

By quantifying sleep disturbance measurements and associating them with measures of emotion regulation and reactivity, I am able to shed light on how partial sleep deprivation is impacting our emotions in conditions like Obstructive Sleep Apnea. The significance of this data can further be utilized to understand people with other disorders such as Periodic Limb Movement Disorder.

The results of this project may also provide clinicians with a way to encourage persevering with the often-intolerable treatments for OSA. If patients believe they will better regulate their emotions after treatment, they may be more inclined to put more effort into accommodating to the gold standard OSA treatment, continuous positive air pressure (CPAP). With a recent pull away from pharmaceutical treatments for emotional wellbeing, treating one's sleep disturbances to improve mental health may be a welcomed alternative.

Furthermore, this research can be generalized to individuals with sleep deprivation due to social or environmental factors. Research has clearly demonstrated

that too many individuals are not allowing themselves to get sufficient sleep on a nightly basis. Research like this may cause individuals to consider their emotional wellbeing before depriving themselves of proper sleep. It is also common that people do not place an emphasis on creating a quiet sleep environment, however, this can be crucial to maintaining proper sleep quality. This research may bring awareness to the importance of eliminating sleep disruptions as seemingly innocent as sleeping with a pet in the bed or with the television on. By encouraging individuals to consciously control their sleep environment, they may eliminate disruptions and allow for improved emotion regulation and reactivity.

Enhancement of emotional wellbeing and quality of life continues to be a priority for the scientific community as the stress of current lifestyles continues to increase. Any information that can be gathered on the topic is essential. It is evident that sleep plays a critical role in this issue, and there is still an abundance of research that must be done to understand the true mechanism behind this relationship.

Chapter II

Materials and Methods

In this research project, I utilized measurement techniques to uncover the relationship between Obstructive Sleep Apnea (OSA), more specifically its consequential sleep disturbances, and emotion regulation and reactivity. OSA is a prevalent disorder that directly impacts the quality of one's sleep and additionally impacts the quality of one's life. Mental health disorders like Depression and Anxiety are known to be major comorbidities with OSA, however, the mechanism behind this remains unclear. OSA is primarily known to disrupt one's sleep with frequent arousals causing substantial sleep fragmentation. These arousals are often due to respiratory flow limitation and subsequent oxygen desaturation. These respiratory-related arousals are measured per hour to determine the severity of one's OSA diagnosis. The Apnea-Hypopnea-Index, or AHI, is the quantification of those respiratory arousals. Sleep apnea patients' AHI can range anywhere from 5 events per hour to over 100 events per hour. OSA is also posited to disrupt sleep architecture by decreasing the amount of time that one spends in the most restorative sleep stages: Stage 3 and Rapid-Eye-Movement (REM) sleep. These disturbances that are attributed to OSA are proven to have major emotional ramifications. This study began to expose the mechanism behind this relationship.

Research Subjects

In this study, I investigated the correlation between OSA-related sleep disruptions and challenges in emotion regulation and reactivity in 20 research subjects volunteering at the Harvard-Affiliated Sleep Disordered Breathing Lab based in Brigham and Women's Hospital. All procedures were approved and monitored by the Partners Institutional Review Board with an IRB Authorization Agreement with the Harvard Institutional Review Board. Informed consent was obtained for all subjects. Subjects that enrolled in the research studies at this lab were entering clinical research studies for improvement in Obstructive Sleep Apnea events by experimental treatments, mainly pharmaceutical treatments. These subjects were otherwise healthy and did not suffer from any other sleep disorders. The subjects were not taking medications that would interfere with their sleep or any psychiatric medications, which could interfere with their emotion regulation and reactivity. No subjects had formal diagnoses of mental health disorders or severe chronic illnesses. In the study population there were 5 females and 15 males. Patients ranged in age from 28 to 61. There were patients represented in all three OSA severities: Mild, Moderate, and Severe (Table 2).

Sleep Stage Measurements

Sleep was recorded using advanced polysomnography recording devices that measure sleep stages using electroencephalography (EEG) on the scalp, eyes, and chin. This is critical information because it allows for the quantification of time spent in each of the sleep stages. This study more specifically focused on determining the amount of time spent in Stage 3 and Rapid-Eye-Movement (REM) sleep stages. This was a unique measurement within this research project because many current studies rely on clinical

diagnoses of OSA which only quantify the AHI of the patient. In clinical diagnostic sleep studies, the duration of time spent in specific sleep stages is not collected. Known for their responsibility in restorative functions during sleep, Stage 3 and REM sleep may play a large part in emotion regulation and reactivity. The amount of time spent in Stage 3 and REM sleep, along with the Total Sleep Duration, was determined by a registered polysomnography technician. These sleep architecture values were used in comparison with the emotion regulation and reactivity measurements to determine if a correlation exists.

Respiratory Measurements

Subjects' breathing and flow limitation was recorded using a nasal canula or a full-face mask. Though the full-face mask remains more precise, both means of measurement are satisfactory in determining the amount of flow limitation a patient experiences throughout the night. The full-face mask measurement is also unique to this research project, as many clinical polysomnographic tests solely rely on the nasal canula. Saturated oxygen levels were also measured throughout the night using a finger-probe pulse oximeter. Blood pressure and heart rate were recorded, as these too can be elevated after sleep deprivation in conditions like Obstructive Sleep Apnea and have also been correlated with a decrease in emotion regulation and reactivity. The apnea-hypopnea index (AHI) was determined by a registered polysomnography technician utilizing criteria determined by the American Academy of Sleep Medicine. According to these criteria, an apnea is defined as a complete cessation of airflow for greater than 10 seconds. Per these criteria, a hypopnea is considered a 30% decrease in airflow that is followed by a 3% decrease in oxygen saturation or an arousal visible in the

polysomnography sleep stage recordings. The AHI is the number of events (apneas and hypopneas) recorded per hour. The AHI was used in comparison with the emotion regulation and reactivity measurements to determine if a correlation exists.

Emotion Regulation and Reactivity Measurements

Emotion regulation and reactivity was quantified using several measurements. One measure of emotion regulation used was the Hospital Anxiety and Depression Scale (HADS). The HADS is well supported in research as a valid assessment of the symptom severity in those suffering from Anxiety and Depression (Bjelland, I., et.al., 2002). As discussed earlier, Anxiety and Depression are commonly seen in subjects that suffer from sleep deprivation and Obstructive Sleep Apnea; thus, this measure was used to confirm this commonly evident emotion regulation problem in this population of people. With an improved quality of sleep, it was expected that the score of this questionnaire would decrease indicating a decrease in anxiety- and depression-related behaviors.

The HADS questionnaire (Fig. 11) contains 14 questions probing how an individual may have felt in the previous week. Seven of the questions pertain to depressive symptoms and seven of the questions pertain to anxiety symptoms. The participant is asked to score each statement based on the following scale: 0 – Not at all, 1 – Occasionally, 2 – A lot of the time, and 3 – Most of the time. A total score of 0-7 in either the Depression or Anxiety categories is considered “Normal”. A score between 8 and 10 is “Borderline Abnormal” in either of the categories. Any score between 11 and 21 is considered “Abnormal” in either of the categories (Fig. 12). The mean score between the two categories was determined for each participant on the two visits being

studied. These means were used to correlate with improvement in AHI and Sleep Architecture over the two nights.

Subjects were also asked to complete the Cognitive Emotion Regulation Questionnaire (CERQ). The Cognitive Emotion Regulation Questionnaire allowed me to assess the tendencies toward negative affect and away from positive affect commonly seen in those suffering from emotional dysregulation. The CERQ was shown to be a valuable tool in understanding emotion regulation problems in the general population (Garnefski, N. & Kraaij, V., 2007).

In this questionnaire (Fig. 13), participants answered questions by ranking the probability of using a particular stress coping mechanism. The range of answers was 1 – Almost Never, 2 – Rarely, 3 – Occasionally, 4 – Frequently, and 5 – Almost Always. An average greater than 3 for any mechanism in question was considered elevated compared to the general population. All questions fell into either the category of "Less Effective" strategies (Self- Blame, Rumination, Catastrophizing, Blaming Others) or the category of "More Effective" strategies (Acceptance, Positive Refocusing, Positive Reappraisal, Putting into Perspective). Participants with difficulty in emotion regulation had an average greater than 12 when taking the sum of the "Less Effective" mechanism category and an average less than 12 when taking the sum of the "More Effective" mechanism category (Fig. 14). The values from both the "Less Effective" and the "More Effective" categories were used to correlated with improvements in AHI and Sleep Architecture over the two nights.

Finally, subjects were asked to complete the Profile of Mood States questionnaire (POMS). The Profile of Mood States Questionnaire (Fig. 15) focuses more on emotional

reactivity and measures a vast range of emotions (Grove, J. R., & Prapavessis, H., 1992). After an improved night of sleep, I expected to see a decrease in the Total Mood Disturbance determined by the Profile of Mood States Questionnaire displaying more controlled emotional reactivity.

In the POMS questionnaire, the participants responded to 40 words or statements that describe the feelings people may have. The individuals were instructed to answer each statement based on how they had been feeling for the previous week. Each of the statements is scored based on a scale of 0 – Not at All, 1 – A little, 2 – Moderately, 3 – Quite a Lot, or 4 – Extremely. The Total Mood Disturbance (TMD) score is calculated by adding the scores for the following categories: "Tension", "Depression", "Anger", "Fatigue", and "Confusion", and subtracting the score for "Vigor" (Fig. 16). Each category is made up of the statements described by Grove, J. R., & Prapavessis, H., 1992. The TMD score was used to correlate with improvements in AHI and Sleep Architecture over the two nights.

Correlating Sleep and Emotion Regulation Measurements

All subjects entered into these studies completed a baseline polysomnography to determine the severity of their sleep apnea (defined by their AHI). This baseline study also determined their untreated sleep architecture (i.e. how much time is spent in each stage of sleep) and their baseline heart rate and blood pressure. During this baseline visit, subjects also underwent the initial testing of emotion regulation and reactivity. These measurements determined their emotion regulation and reactivity while considered sleep deprived. Only subjects with clear signs of Obstructive Sleep Apnea (AHI > 10 events per hour) were considered to continue in the research study, as they were the only

subjects with above normal sleep fragmentation. After this baseline study, the polysomnography study was scored to measure a baseline quantification of AHI and sleep duration in each stage. The subjects were followed throughout their secondary visit at the lab when they received an experimental treatment. The experimental treatment varied based on the visit, however, I was focusing solely on correlating changes in AHI and Sleep Architecture with the changes in Emotion Regulation and Reactivity, not on the treatments. The same physiological and emotional procedures were performed at treatment visits and later compared to the baseline night. There were no other significant changes between baseline and treatment that could have impacted the study results.

The emotion regulation and reactivity data was correlated with the changes in AHI, the changes in sleep architecture, and the changes in total sleep duration. When analyzing the sleep architecture, the focus was placed on Stage 3 and REM sleep, as these are known to have more restorative functions. “Sleep duration” was measured as the amount of time clinically scored as a sleep stage out of the total time spent in bed after lights out. The sleep duration allowed us to estimate how much sleep deprivation a subject was experiencing, and whether this change impacted emotion regulation and reactivity.

Statistical Analysis

The changes in sleep (quantified by a change in AHI, an change in duration of time spent in Stage 3 or REM sleep, and a change in Total Sleep Duration) and variations in emotional regulation measurements between the baseline and the treatment night were compared using paired-sample t-tests (Table 1). Paired-sample t-tests, calculated in R Studio, were used to determine the correlation between the change in AHI and all

emotional regulation and reactivity scores including HADS Score, CERQ “LE” Score, CERQ “ME” Score and the POMS TMD score. Paired-sample t-test were also used to determine any correlation between sleep architecture measures including changes in duration of Stage 3, changes in duration of REM sleep, and changes in Total Sleep Duration with all emotional regulation and reactivity scores. The standard p threshold of .05 was used to determine significance and the 95% confidence interval was calculated.

Table 1. Changes in Sleep Architecture and Emotion Regulation and Reactivity.

	Changes in Sleep				Changes in Emotion Regulation and Reactivity			
Subject	ΔAHI	ΔStage 3 Duration	ΔREM Sleep Duration	ΔTotal Sleep Duration	ΔHADS	ΔCERQ "LE"	ΔCERQ "ME"	ΔTMD
1	-65.6	19	-4	8	-2	-1.75	2.5	-6
2	-41	-9	-49	-71	-3	-5	0.75	-6
3	-12.9	13	-10	71	1	0	1.75	-11
4	-20.7	13	3	-45	-5	-1.75	-0.5	-14
5	-48.3	-4	2	-76	0	0.25	0.75	-1
6	-10.6	19	6	13	-2	-4.5	1.25	-4
7	-8.1	-20	42	46	3	-0.5	-1.25	-11
8	-18.1	-3	-43	-19	1	-1	-1.25	-10
9	-5.5	17	0	27	-1	-2.5	0.25	1
10	-38.4	40	-25	4	1	-0.5	2.5	-1
11	-30.9	129	45	63	-5	-3.5	2.25	-13
12	-13.9	30	2	75	-9	-5.5	0.25	3
13	-38.1	27	3	26	0	1.75	2.25	-6
14	-40.8	43	17	36	2	0.5	0.75	1
15	-13.3	11	-53	-98	-1	-4.5	-1	-3
16	-9.4	20	1	11	-3	-4.5	1.25	-5
17	-47.3	34	19	30	-9	-5.5	0.75	-5
18	-7.8	11	26	77	-1	-2.5	-0.5	-2
19	-24.1	1	6	33	-1	-0.75	1.25	-6
20	-20.4	24	5	42	-6	-6.25	-1.25	-10

The data found in Table 1 describes the changes in sleep architecture and emotion regulation and reactivity measurements from the baseline to treatment visit. This data was used to perform paired-sample t-tests.

Research Limitations

As with all research projects, there are several limitations that are unavoidable. The greatest limitation to this investigation was the impact of the global pandemic happening at the time of the study. This marginally impacted the study due to the delay in sleep studies taking place, however, this could have greatly impacted the values determined for emotion regulation and reactivity. Individuals were experiencing a

heightened sense of stress due to the external circumstances, and this should be noted when evaluating the data. The psychological stress of the pandemic also made entering a hospital setting even more stressful, therefore, this could also greatly impact one's responses to emotional regulation questionnaires and tasks. Furthermore, due to the stress individuals were experiencing during this time, it is more than likely that their sleep habits have been modified, and this could also contribute to a potential skew in the data.

Another limitation is that the treatments were only given to the patient for up to 7 nights prior to measurement. While some individuals may see immediate improvement in mood after one week of sufficient sleep, many of those with chronic sleep deprivation for several years may need a much longer duration to witness any significant improvement in mood. Research has proven that it can take several nights of quality sleep to make up for a single night of insufficient sleep (Sallinen, M., et. al., 2008). With that said, the amount of sleep debt incurred by most individuals will likely take much longer than one week to diminish and thus, their emotion regulation and reactivity may also take just as long.

Whenever experimental treatment options are being used, there is always the potential that they will not work sufficiently, and also the potential that they will have side effects of their own. The dynamics of using an experimental treatment option was another limitation to this study, as everyone reacts differently to treatments they have never used before. There may be an exaggerated response due to the novelty of the treatment, or there may be an adverse response due to a poor reaction to the treatment. This is something to take into consideration when analyzing the results of this study.

Another common treatment-related limitation of this study was the possibility for placebo effect. Individuals that are anticipating that a treatment will help them feel better

will often report that it is helping. In some cases, there could be no quantitative evidence of sleep improvement, however, patients will report to subjectively feel as though their emotional state has improved. In our research study we attempt to control for this by keeping the treatment blinded to the patient, however, sometimes that is not sufficient. It was also clear, due to the nature of the emotion regulation and reactivity questionnaires, that we were looking for a change in emotional state. This may have also impacted the results calculated in this study.

A final limitation to this study was the size of the study population. With only 20 participants studied, there were not enough individuals to create subgroups by AHI severity. I feel that creating subgroups by OSA severity could have led to interesting results that differed from investigating the group as a whole. I feel that OSA severity plays a major role in this issue, and likely contributes to the amount of sleep debt a person accumulates. By creating subgroups based on OSA severity, this contributing factor could have been ruled out and there would have been greater variety in the individuals studied.

Chapter III.

Results

Baseline Characteristics

Participants involved in this project were volunteers for research studies testing experimental treatment options for Obstructive Sleep Apnea (OSA). All participants had prior diagnoses of OSA. These subjects were otherwise healthy and did not suffer from any other sleep disorders. The subjects were not taking medications that would interfere with their sleep or any psychiatric medications, which could interfere with their emotion regulation and reactivity. No subjects had formal diagnoses of mental health disorders or severe chronic illnesses. The minimum age of participants studied was 28 and maximum age was 61 (Mean Age: 45.45). Of the participants studied, 1 had Mild OSA, 6 had Moderate OSA and 13 had Severe OSA. In the study population there were 5 females studied and 15 males studied. Baseline characteristics of the participants studied are shown in Table 2.

Table 2. Baseline Characteristics of 20 Participants Studied.

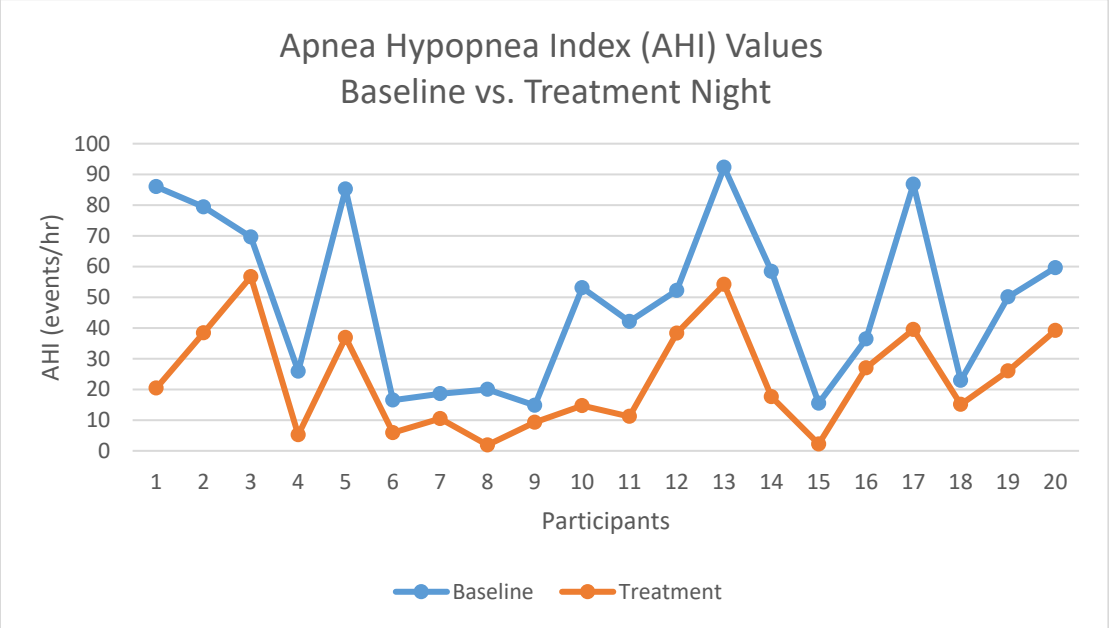
Baseline Characteristics				
Subject	Age	Baseline AHI	Severity of OSA	Sex
1	56	86	Severe	F
2	49	79.4	Severe	F
3	32	69.6	Severe	M
4	55	25.9	Moderate	M
5	51	85.2	Severe	M
6	42	16.5	Moderate	M
7	28	18.6	Moderate	F
8	32	20	Moderate	M
9	35	14.8	Mild	M
10	61	53.1	Severe	M
11	58	42.1	Severe	F
12	44	52.2	Severe	M
13	45	92.3	Severe	M
14	54	58.4	Severe	M
15	39	15.5	Moderate	M
16	41	36.4	Severe	M
17	57	86.8	Severe	M
18	38	22.9	Moderate	F
19	43	50.1	Severe	M
20	49	59.6	Severe	M

Sleep Architecture Improvement

Apnea Hypopnea Index

The calculation of the Apnea Hypopnea Index (AHI) improvement was determined by comparing scored AHI values from Baseline and Treatment visits across all 20 participants. The data was used to calculate and graph the change witnessed in each participant across the two nights. Raw data showed that the mean change in AHI was -25.76 with a Standard Deviation of 16.98. All participants decreased in AHI from Baseline to Treatment Visit, however, not all participants AHI was decreased to a normal value (AHI <10). Of the 20 participants, on 25% were brought down to normal AHI values. These reported changes can be seen in Figure 1.

Figure 1: Apnea Hypopnea Index Scores Recorded During the Baseline and Treatment Visits.



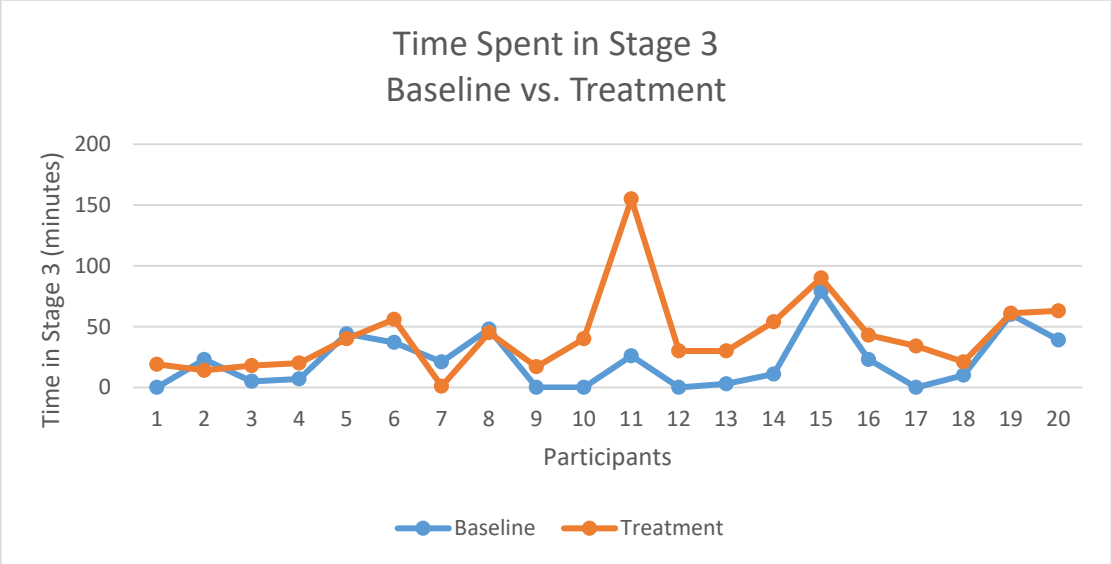
All AHI values are reported in events per hour.

The sample paired t-test supported the evidence found in the raw data calculations and provided significance behind the decrease in AHI values. The test reported a p value < .001 and 95% confidence intervals of [17.81, 33.71]. This shows that after treatment, the Obstructive Sleep Apnea of all patients had significantly improved as proven by a decrease in their AHI.

Stage 3 Sleep

The number of minutes spent in the Stage 3 of sleep was measured in each of the 20 participants during both the Baseline and Treatment night. These measurements were used to determine a change in duration after treatment. Raw data provided evidence that there was a mean increase of 20.75 minutes in Stage 3 sleep after treatment with a standard deviation of 30.21 minutes. Of the 20 participants, 80% showed an increase in Stage 3 Duration. The changes in Stage 3 Duration can be seen in Figure 2.

Figure 2: Time Spent in Stage 3 Sleep During the Baseline and Treatment Visits.



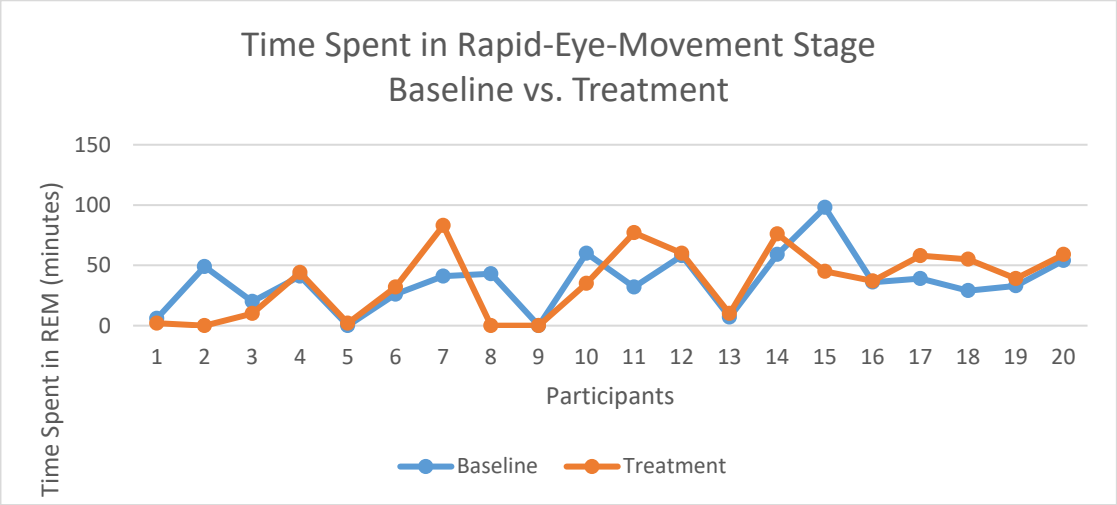
Duration of time spent in Stage 3 sleep was recorded in minutes.

The sample paired t-test supported the significance of an increase in minutes spent in Stage 3 sleep with a p-value of .003 and 95% confidence intervals of [-34.89, -6.61]. This shows that after treatment, participants spent more time in stage 3 sleep than during their baseline visit.

Rapid-Eye-Movement Sleep

The number of minutes spent in the Rapid-Eye-Movement (REM) stage of sleep was measured in each of the 20 participants during both the Baseline and Treatment night. These measurements were used to determine a change in duration after treatment. Raw data determined that there was a mean change of -0.35 minutes in REM sleep with a standard deviation of 26.19 minutes. Of the 20 participants, 60% showed an increase in duration of time spent in the REM stage. The changes in duration of REM stage can be seen in Figure 3.

Figure 3: Time Spent in the Rapid-Eye-Movement Stage of Sleep During the Baseline and Treatment Visits.



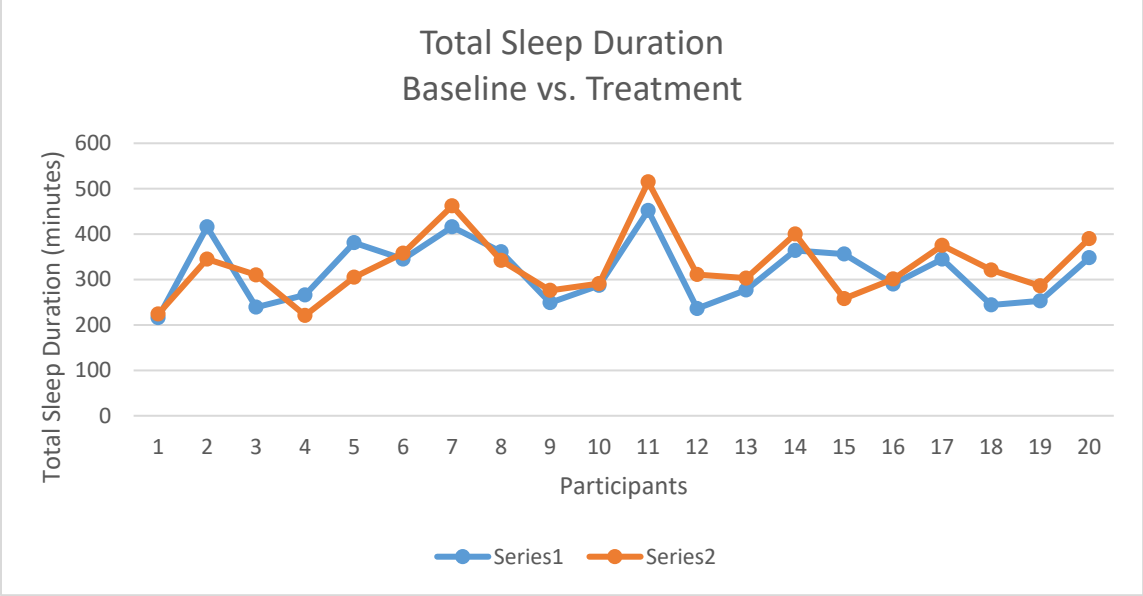
Duration of time spent in REM stage sleep was recorded in minutes.

The sample paired t-test determined that there was no significant change in minutes spent in the REM stage of sleep with a p-value of 0.9. This suggests that the duration of time in this stage did not significantly increase with treatment.

Total Sleep Duration

The total sleep duration (TSD) was measured in each of the 20 participants during both the Baseline and Treatment night. This was calculated by adding the number of minutes spent in each sleep stage during the overnight visit. These measurements were used to determine a change in duration after treatment. Raw data determined there was a mean change of 12.65 minutes of Total Sleep Duration with a standard deviation of 50.84 minutes. Of the 20 participants, 75% showed an increase in Total Sleep Duration. The changes in Total Sleep Duration can be seen in Figure 4.

Figure 4: Total Sleep Duration During the Baseline and Treatment Visits.



Total sleep duration was recorded in minutes.

The sample paired t-test determined that there was no significant change in minutes of total sleep duration when comparing the baseline and treatment visit. This provided a calculated p-value of 0.3. This suggests that the total sleep duration did not significantly increase after treatment.

All calculated changes and values in sleep architecture subgroups have been summarized in Table 3.

Table 3. Summary of analyses for sleep architecture subgroups.

Sleep Architecture Analysis						
					Confidence Interval	
Subgroup	**Mean**	**Standard Deviation**	**Improvement**	**p-value**	**Lower Limit**	**Upper Limit**
AHI	-25.76	16.98	20/20 (1.0)	1.3E-06	17.8	33.7
Stage 3	20.75	30.21	16/20 (.8)	.006	-34.9	-6.61
REM Stage	-0.35	26.19	12/20 (.6)	0.952	-11.9	12.6
Total Sleep Duration	12.65	50.84	15/20 (.75)	.280	-36.4	11.1

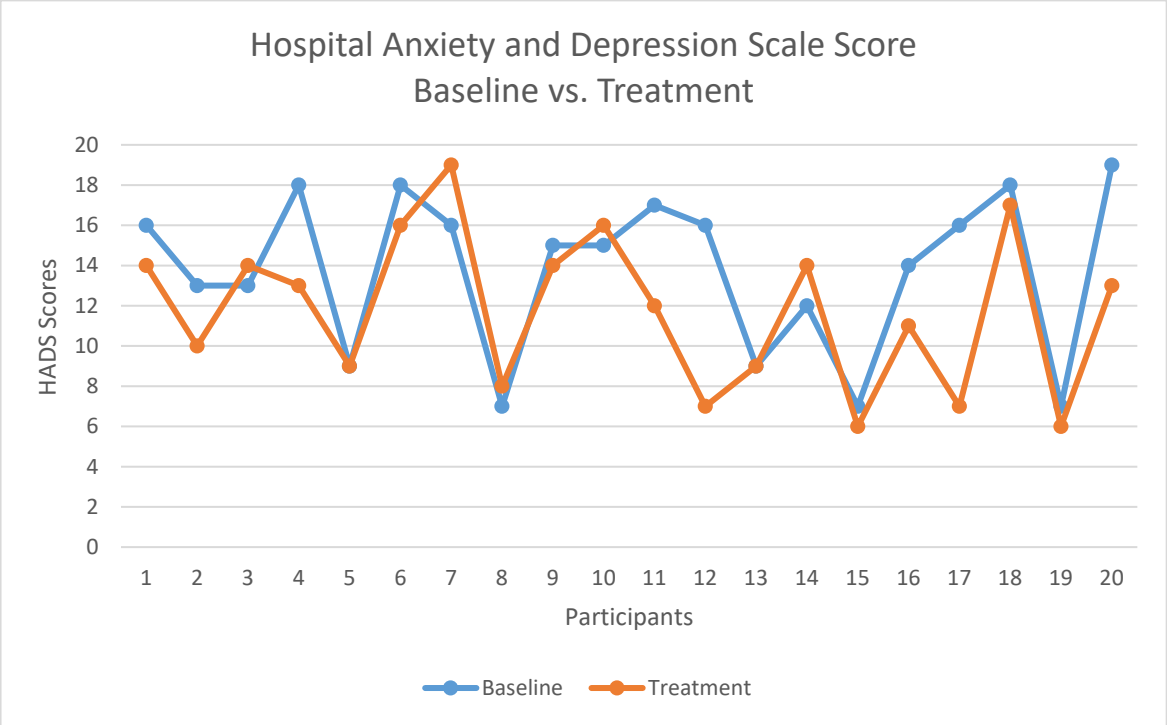
Emotional Regulation and Reactivity Improvement

Hospital Anxiety and Depression Scale

Participants were asked to complete the Hospital Anxiety and Depression Scale (HADS) Questionnaire on their Baseline and Treatment visits. The data recorded was used to calculate and graph the change witnessed in each participant across the two nights. Raw data showed that after treatment, participants reported HADS scores that decreased by a mean of -2.0 points with a standard deviation of 3.36. Of the 20 participants, 65% showed a decrease in reported HADS score, however, only 20% of participants were

brought down to a normal value (HADS < 8). The changes in HADS scores can be seen in Figure 5.

Figure 5: Hospital Anxiety and Depression Scale scores reported during the Baseline and Treatment Visits.



The sample paired t-test determined that there was a significance in the change in HADS scores with a p-value of .02 and a 95% confidence interval of [.429, 3.57]. This suggests that participants reported lower Anxiety and Depression scores after treatment.

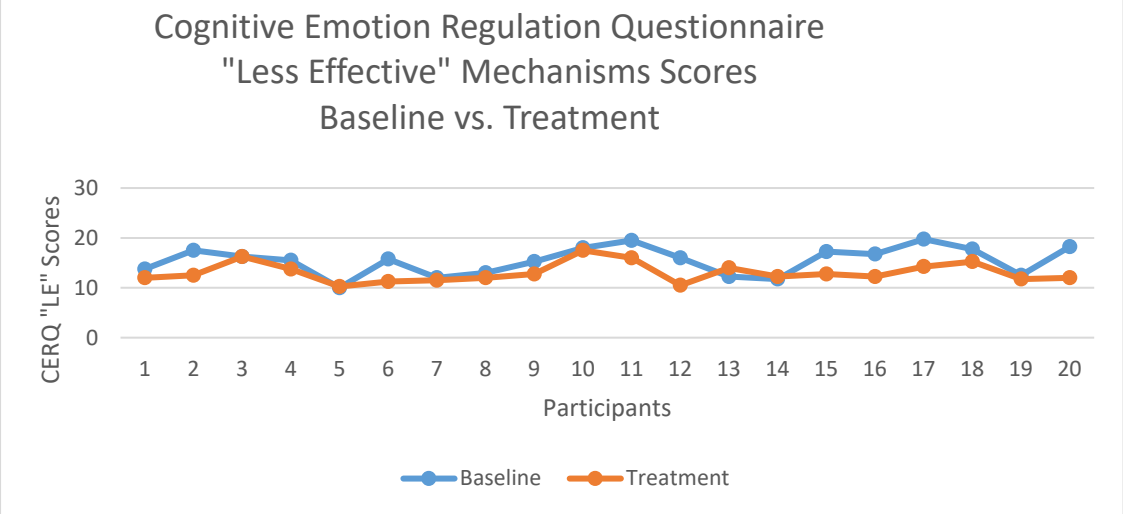
Cognitive Emotion Regulation Questionnaire

The scores for the Cognitive Emotion Regulation Questionnaire (CERQ) were analyzed by separating the reported answers into two categories: those pertaining to the "Less Effective" coping mechanisms questions and those pertaining to the "Most Effective" coping mechanisms questions.

Change in "Less Effective" Coping Mechanisms

The scores from the questions pertaining to the frequency of using the "Less Effective" coping mechanisms were analyzed and used to calculate and graph changes between the two nights. Raw data showed that after treatment, participants reported the use of the "Less Effective" coping mechanisms at a mean decrease of -2.4 with a standard deviation of 2.37. Of the 20 participants, 80% of them showed a decrease in the CERQ "Less Effective" score. Only 40% of participants showed a decrease in the CERQ "LE" down to normal values (score < 12). The changes in this score can be seen in Figure 6.

Figure 6: Cognitive Emotion Regulation Questionnaire, "Less Effective" Mechanisms scores reported during the Baseline and Treatment visits

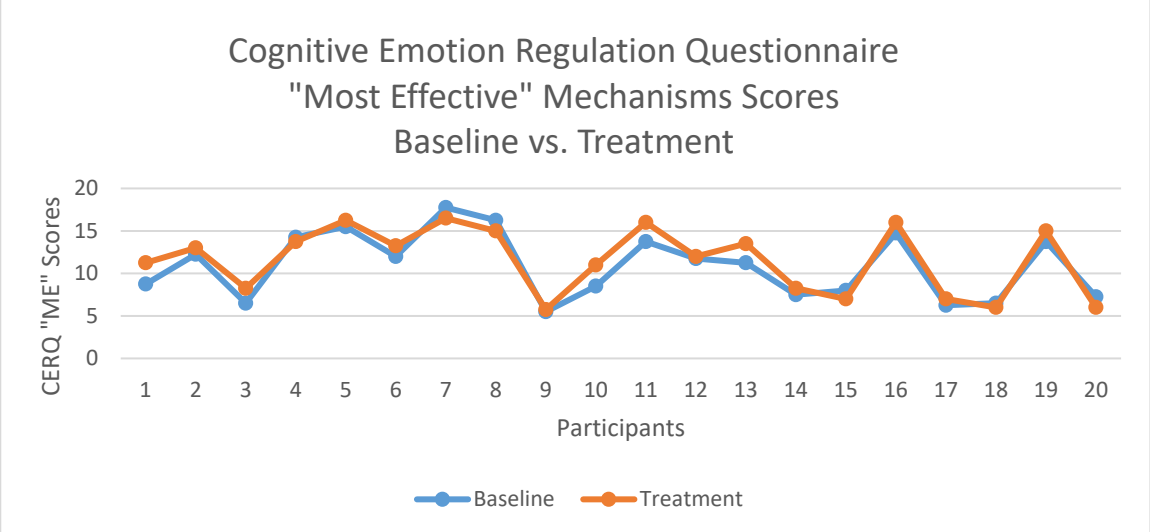


The sample paired t-test showed significance in the decrease of this score with a p-value < .001 and a 95% confidence interval of [1.29, 3.51]. This suggests that participants reported using the “Less Effective” coping mechanisms at a significantly lower frequency after treatment.

Change in “Most Effective” Coping Mechanisms

The scores from the questions pertaining to the frequency of using the “Most Effective” coping mechanisms were analyzed and used to calculate and graph changes between the two nights. Raw data showed that after treatment, participants reported the use of the “Most Effective” coping mechanisms at a mean increase of .64 with a standard deviation of 1.27. Of the 20 participants, 70% of them showed an increase in the CERQ “Most Effective” score. Only 50% of participants showed an increase in the CERQ “ME” to normal values (score > 12) The changes in this score can be seen in Figure 7.

Figure 7: Cognitive Emotion Regulation Questionnaire, “Most Effective” Mechanisms scores reported during the Baseline and Treatment visits

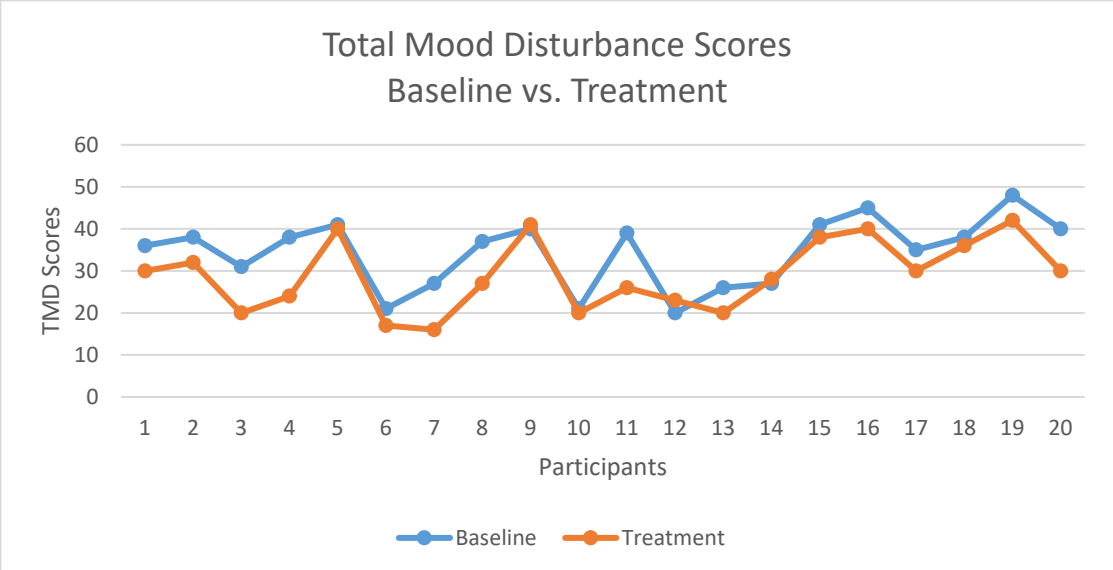


The sample paired t-test showed significance in the increase of this score with a p-value of .04 and a 95% confidence interval of [-1.23, -0.04]. This suggests that participants reported using the "Most Effective" coping mechanisms at a higher frequency after treatment.

Total Mood Disturbance

The Total Mood Disturbance (TMD) scores were reported using the abbreviated Profile of Mood States questionnaire that participants completed on the baseline and treatment visit. The data recorded were used to calculate and graph the change witnessed in each participant across the two nights. Raw data showed that after treatment, participants reported a decrease in TMD with a mean of -5.45 and standard deviation of 4.86. Of the 20 participants, 85% of them showed a decrease in the Total Mood Disturbance score. The changes in the TMD score can be seen in Figure 8.

Figure 8: Total Mood Disturbance scores reported using the abbreviated Profile of Mood States questionnaire during the Baseline and Treatment visits



The sample paired t-test showed significance in the decrease of this score with a p-value < .001 and a 95% confidence interval of [3.18, 7.73]. This suggests that participants total mood disturbance was decreased after treatment.

All calculated changes and values in emotional regulation and reactivity subgroups have been summarized in Table 4.

Table 4. Summary of analyses for emotional regulation and reactivity subgroups.

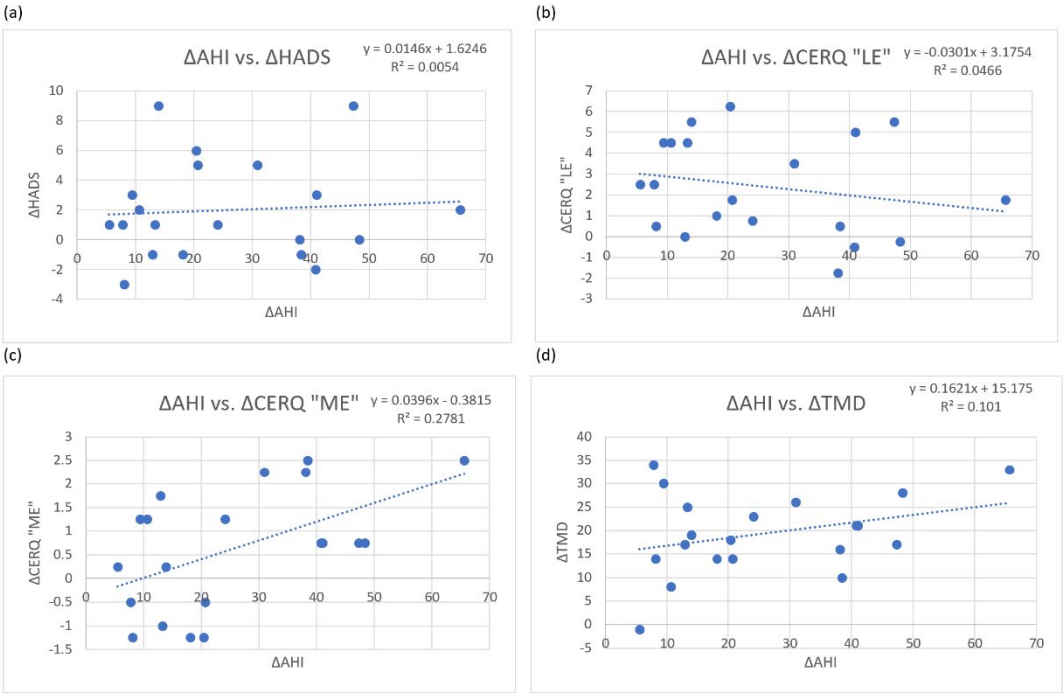
Emotion Regulation and Reactivity Analysis						
					Confidence Interval	
Subgroup	**Mean**	**Standard Deviation**	**Improvement**	**p-value**	**Lower Limit**	**Upper Limit**
HAD	-2.0	3.36	13/20 (.65)	.02	0.43	3.57
CERQ "LE"	-2.4	2.37	16/20 (.80)	.0002	1.29	3.51
CERQ "ME"	.64	1.27	14/20 (.70)	.04	-1.23	-0.04
TMD	-5.45	4.86	17/20 (.85)	7.7E-05	3.18	7.73

Linear Regression Analysis

In the sleep architecture analysis, it was determined that only the AHI and the duration of Stage 3 sleep changed significantly from baseline to treatment measurements. With this information, a linear regression analysis was performed to determine whether the amount of change seen in the AHI or Stage 3 sleep duration correlated with the amount of change seen in any of the emotional regulation and reactivity questionnaires.

When performing a linear regression analysis on the data, it was determined that the amount of change witnessed in the AHI values from Baseline to Treatment did not significantly correlate with the amount of change witnessed in any of the emotion regulation and reactivity questionnaires (i.e. decrease in HADS score, decrease in CERQ "LE" score, increase in CERQ "ME" score, or decrease in TMD Score). These linear regression models can be seen in Figure 9.

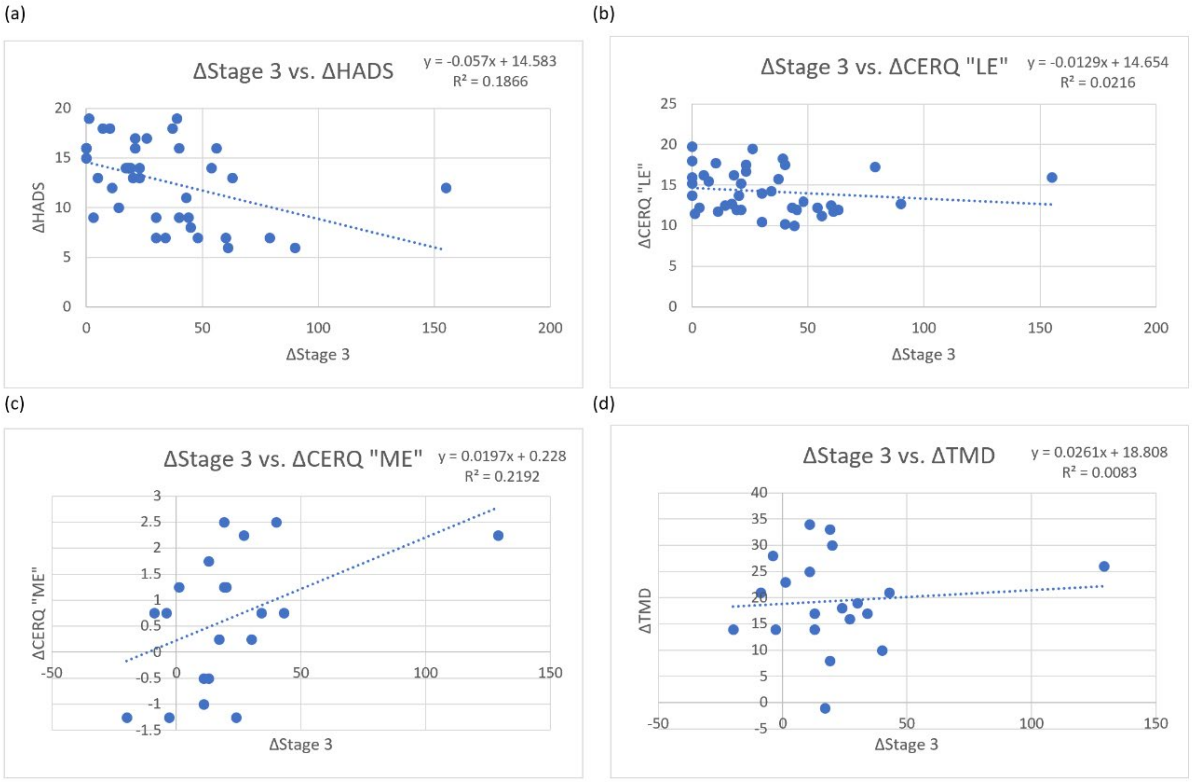
Figure 9. Linear Regression Models for Change in AHI vs. Change in Emotion Regulation and Reactivity Questionnaires.



The linear regression models in Figure 9 demonstrate (a) AHI vs. HADS, (b) AHI vs. CERQ "LE", (c) AHI vs. CERQ "ME", (d) AHI vs. TMD.

The linear regression analysis also determine that the amount of change witnessed in the duration of time spent in Stage 3 sleep also did not significantly correlate with any of the emotion regulation and reactivity questionnaires. These linear regression models can be seen in Figure 10.

Figure 10. Linear Regression Models for Change in Stage 3 Sleep Duration vs. Change in Emotion Regulation and Reactivity Questionnaires.



The linear regression models in Figure 10 demonstrate (a) Stage 3 Sleep vs. HADS, (b) Stage 3 Sleep vs. CERQ "LE", (c) Stage 3 Sleep vs. CERQ "ME", (d) Stage 3 Sleep vs. TMD.

The results of the linear regression analysis suggest that though there is a relationship present, one cannot predict the amount of change that will be seen in any emotion regulation and reactivity parameter based on the amount of change that can be seen in AHI values or stage 3 sleep durations. With that said, the sample-paired t-tests suggest that one would be able to predict whether or not a significant change would be present.

Chapter IV.

Discussion

Improvements in emotion regulation and reactivity continue to be a top priority as the stress of current lifestyles continue to increase. It is evident that sleep plays a critical role in this issue, however, it remains unclear what those implications are. In Obstructive Sleep Apnea (OSA), sleep is continuously disrupted, and it is clear that this impacts the emotional state of people suffering from this disorder (Akberzie, W., et al., 2020). This study uncovered several aspects of Obstructive Sleep Apnea that could be contributing to the emotional state of sufferers by measuring the change in emotion regulation and reactivity after significant treatment.

The data analyzed suggests that with improvement of general sleep architecture parameters, there is subsequent and significant improvement in emotion regulation and reactivity measures. Of the four sleep architecture subgroups measured, both the Apnea-Hypopnea-Index (AHI) and the duration in Stage 3 sleep improved significantly. The change in AHI demonstrated was significant, however, did not bring all participants down to values that would be considered normal. Only 5 out of the 20 participants were brought down to an AHI value that would be considered normal in the general population. Neither the duration of REM sleep nor the Total Sleep Duration improved to a significant extent. What this data suggests is that any subsequent improvement seen in emotional regulation and reactivity would likely be correlated to the change in AHI or duration of Stage 3 sleep, however, the same was not proven for duration of REM sleep nor Total Sleep Duration.

The emotion regulation and reactivity scores were determined using three prominent questionnaires that are widely accepted in the scientific community. The first questionnaire utilized was the Hospital Anxiety and Depression Scale Questionnaire (HADS). While the changes calculated between the baseline and the treatment night were significant, further investigation showed that only 4 out of the 20 participants were brought down to a HADS score that was considered normal in the general population. This data suggested that while an improvement in AHI and duration of Stage 3 sleep likely correlated with an improvement in the HADS score, the improvement was not significant enough to normalize emotion regulation and reactivity levels determined by this questionnaire in many of the participants.

The second questionnaire utilized to determine changes in emotion regulation and reactivity was the Cognitive Emotion Regulation Questionnaire (CERQ). In this questionnaire, data was analyzed in two subgroups, as these groups were expected to change inversely. The first subgroup was analyzing the responses to questions pertaining to the frequency of using the "Less Effective" coping mechanisms (CERQ "LE") for stress, as determined by Garnefski, N. & Kraaij, V., 2007. There was a significant change in the reported values that showed 80% of participants decreased their use of the "Less Effective" coping mechanisms after treatment. However, only 8 out of 20 participants were brough down to normal scores. This data suggests that an improvement in AHI and Stage 3 sleep correlated with a decrease of utilization of "Less Effective" coping mechanisms as defined by the Cognitive Emotion Regulation Questionnaire.

In the analysis of the Cognitive Emotion Regulation Questionnaire, the second subgroup being analyzed were the responses to questions pertaining to the frequency of

using the “Most Effective” coping mechanisms (CERQ “ME”), as determined by Garnefski, N. & Kraaij, V., 2007. In this subgroup, as well, there was a significant change in the reported values. 70% of the group showed a significant increase in the frequency of using the “Most Effective” coping mechanisms for stress after treatment. However, only 10 out of the 20 participants were increased to a normal score. This data suggests that an improvement in AHI and Stage 3 sleep correlated with an increase of utilization of “Most Effective” coping mechanisms as defined by the Cognitive Emotion Regulation Questionnaire.

The final questionnaire used to measure emotion regulation and reactivity after treatment was the abbreviated Profile of Mood States questionnaire and the calculated Total Mood Disturbance score. There was a significant improvement in Total Mood Disturbance in participants after treatment. After treatment, 15 of the 20 participants reported less total mood disturbance. This improvement suggests that an improvement in the AHI and Stage 3 sleep duration correlates with a decrease in total mood disturbance.

When evaluating the post-treatment emotion regulation and reactivity scores, it is evident that there is significant improvement in the responses of all three questionnaires. It is demonstrated, however, that while there was improvement present, many of the participants were not improved to normal values.

While a distinct and significant correlation with an improvement in AHI and Stage 3 sleep with emotion regulation and reactivity measures is present, it does not guarantee causation. What is clear, however, is that there is a significant relationship between sleep architecture and emotion regulation and reactivity that should continue to be studied.

It was determined, through linear regression analysis, that the amount of change calculated in AHI and Stage 3 sleep did not strongly correlate with the amount of change calculated in emotional regulation and reactivity questionnaires. What this suggests is that one could not use the amount of change in AHI and Stage 3 to predict the amount of change in any of the emotional regulation and reactivity questionnaires. Using the data calculated by the paired-sample t-tests, however, one could predict whether a significant change would be expected.

While improvements were significant and evident, it is of importance to note that several of the parameters did not improve to normal values and thus there were still issues at large. For example, though all participants improved in their AHI values, only 5 out of the 20 participants were improved enough to be considered “normal”. With that said, if more participants were improved to normal values, it is possible that the correlation would be stronger between change in AHI and improvement in emotional regulation and reactivity. It is also possible that, with a more significant improvement in AHI, emotional regulation and reactivity scores would improve more significantly than seen in this study.

This same pattern was seen in the emotion regulation and reactivity parameters. In many of the measurements, while significant improvement was demonstrated, the improvements did not bring the large majority of participants to “normal” values when comparing them to the general population. It is posited that with stronger, and longer, improvement of sleep parameters, there would be a more promising improvement of emotion regulation and reactivity measures.

There are other physiological parameters that are disrupted in those diagnosed with OSA that were not discussed in this study that could also contribute to difficulties with emotion regulation and reactivity. The most prominent consequence is the persistent oxygen desaturation that typically occurs in these patients. This desaturation is the cause of several risk factors associated with OSA such as the risk for stroke, heart attack, and cognitive difficulties. Especially due to its link to cognitive difficulties, it is exceedingly possible that this prominent oxygen desaturation also contributes to difficulties in emotion regulation and reactivity, however, this was not analyzed in this study.

The size of the study group is important to recognize, as well. With only 20 participants enrolled, there was a clear lack of variation in OSA severity. There were not enough participants to organize and analyze subgroups by OSA severity and further determine if there was a correlation within Mild, Moderate, or Severe participants alone. This information could provide an important distinction between groups and would be interesting to study in the future. While the evidence presented was promising, a much larger and more diverse group would need to be studied in order to fully determine the implications being suggested.

It is also crucial to recognize the general increase in difficulties with emotion regulation and reactivity due to the worldwide pandemic occurring simultaneous to this study. It is of importance to discuss that much of the general population was experiencing a heightened level of Anxiety and Depression that was likely seen in our study population, as well. Due to the timing of the study, it is possible that emotion regulation and reactivity scores were skewed due to the amount of unrest within the community as a whole. It is also possible that sleep quality was consequentially skewed or even further

disrupted than normal. It is more than likely that both sleep and emotional regulation values would be different if this study took place during different circumstances.

While several research studies have linked the comorbidity of OSA with Anxiety and Depression, the mechanism behind this remained widely unclear (Asghari, A., et.al. 2012). This study went a phase further to break down different sleep parameters and correlate them with changes in emotion regulation and reactivity. It allowed us to get one step closer to understanding the impact that sleep debt, caused by untreated OSA, has on the sufferer's emotional state and how this can translate to the general population. This study clearly demonstrated that there is a link between a decrease in sleep disruption (defined, in this study, by a lower AHI) and an improvement of emotion regulation and reactivity (defined by improved questionnaire scores). This study went further to demonstrate that an increase in Stage 3 sleep duration also correlates with this improvement demonstrated in emotion regulation and reactivity.

Generalized Implications

When evaluating the implications of this study, it is important to realize the impact an AHI has on an individual. As the scored AHI value increases, the amount of sleep disruption a person encounters increases, as well. With that said, the sleep architecture of a person can vary significantly. There is no clear way to determine how much sleep debt a person with OSA accumulates on a nightly basis, however, it is clear that with more disruptions there will be more sleep debt. Research has also made clear that it may take an individual several nights to compensate for one night of poor sleep (Sallinen, M., et. al., 2008). By analyzing different sleep parameters, this study provided

some clarity in the sleep disruptions caused by OSA and created a way to generalize these suggestions into populations beyond those suffering with Obstructive Sleep Apnea.

The clearest implication of this study, however, remains in the population of people suffering with Obstructive Sleep Apnea. This provides clear evidence that those with OSA could find relief in their emotion regulation and reactivity problems with sufficient treatment. The gold-standard treatment for OSA is currently continuous-positive-air-pressure (CPAP), however, patient tolerability consistently remains low. If clinicians are able to provide evidence that the emotional wellbeing of their patients will improve without the use of medications such as SSRI's, this may be a convincing factor to help patients persevere. While the importance of treating OSA spans much further than just emotional wellbeing, it is important for clinicians to emphasize this factor, as it is a tangible difference that will be noticeable to patients over time. This evidence may cause patients to be more inclined to put enhanced effort into accommodating to CPAP and sufficiently treating their OSA.

The evidence provided in this study can also be correlated to those suffering from other disruptive sleep disorders such as Central Sleep Apnea, Periodic Limb Movement Disorder, or Insomnia. All of these alternative sleep disorders also cause significant disruptions during sleep which could contribute to an accumulation of sleep debt. This detriment is now known to severely impact one's emotion regulation and reactivity the way the disruptions caused by Obstructive Sleep Apnea has in this study population.

In a more general population, this evidence can be linked to any factor that is contributing to sleep disruptions such as: sleeping with pets in the bed, sleeping with a snoring or apneic bed partner, or sleeping in an environment with other significant

distractions (i.e., television on, loud train outside, persistent construction or traffic noises). This can also be linked to the large population of individuals that are currently depriving themselves of sufficient duration of sleep on a nightly basis. Anyone who falls victim to sleep disruptions or deprivation over a long period of time are likely to incur the same sleep debt issues that someone with a sleep disorder does, and evidently this could negatively impact their emotional wellbeing. Having this evidence available to individuals may provide an explanation to why a person's emotional wellbeing has diminished over time and encourage them to improve their sleep hygiene and environment.

Finally, though the evidence in this study may be difficult to correlate in a precise manner, it has made clear the implication that sleep debt has on any individual's emotional wellbeing. Sleep debt can accumulate by depriving an individual of proper sleep or by disrupting one's sleep throughout the night. This study examined the latter; however, the results are just the same. Any accumulation of sleep debt will be correlated with a disturbance in one's emotional regulation and reactivity. With that said, this evidence can emphasize to the general population the importance of getting sufficient and high-quality sleep on a nightly basis.

Conclusion

Sleep disruptions, like those witnessed in Obstructive Sleep Apnea patients, pose a significant risk to developing difficulties with emotion regulation and reactivity. It is evident that an improvement in sleep disruptions displayed by decreasing AHI and increasing the duration of time in Stage 3 sleep, correlates with an improvement in

emotion regulation and reactivity scores. This study provides a good foundation for understanding the mechanism behind sleep disruptions and consequential emotion regulation and reactivity problems in Obstructive Sleep Apnea patients.

By analyzing specific sleep architecture parameters, this data can be generalized to the general population as a whole. We can begin to understand the specific parameters being impacted by sleep disruptions and continue to expand our knowledge on the consequences that sleep debt has on our emotional wellbeing and overall quality of life.

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Appendix

Figure 11. The Hospital Anxiety and Depression Scale

Hospital Anxiety and Depression Scale (HADS)

Tick the box beside the reply that is closest to how you have been feeling in the past week.
Don't take too long over you replies: your immediate is best.

D	A		D	A	
		I feel tense or 'wound up':			**I feel as if I am slowed down:**
	3	Most of the time	3		Nearly all the time
	2	A lot of the time	2		Very often
	1	From time to time, occasionally	1		Sometimes
	0	Not at all	0		Not at all
		I still enjoy the things I used to enjoy:			**I get a sort of frightened feeling like 'butterflies' in the stomach:**
0		Definitely as much		0	Not at all
1		Not quite so much		1	Occasionally
2		Only a little		2	Quite Often
3		Hardly at all		3	Very Often
		I get a sort of frightened feeling as if something awful is about to happen:			**I have lost interest in my appearance:**
	3	Very definitely and quite badly	3		Definitely
	2	Yes, but not too badly	2		I don't take as much care as I should
	1	A little, but it doesn't worry me	1		I may not take quite as much care
	0	Not at all	0		I take just as much care as ever
		I can laugh and see the funny side of things:			**I feel restless as I have to be on the move:**
0		As much as I always could		3	Very much indeed
1		Not quite so much now		2	Quite a lot
2		Definitely not so much now		1	Not very much
3		Not at all		0	Not at all
		Worrying thoughts go through my mind:			**I look forward with enjoyment to things:**
	3	A great deal of the time	0		As much as I ever did
	2	A lot of the time	1		Rather less than I used to
	1	From time to time, but not too often	2		Definitely less than I used to
	0	Only occasionally	3		Hardly at all
		I feel cheerful:			**I get sudden feelings of panic:**
3		Not at all		3	Very often indeed
2		Not often		2	Quite often
1		Sometimes		1	Not very often
0		Most of the time		0	Not at all
		I can sit at ease and feel relaxed:			**I can enjoy a good book or radio or TV program:**
	0	Definitely	0		Often
	1	Usually	1		Sometimes
	2	Not Often	2		Not often
	3	Not at all	3		Very seldom

Bjelland, I., Dahl, A. A., Haug, T. T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale. An updated literature review. Journal of psychosomatic research, 52(2), 69–77.

Figure 12. The Hospital Anxiety and Depression Scale Scoring

Scoring:
Total score: Depression (D) _____ Anxiety (A) _____
0-7 = Normal
8-10 = Borderline abnormal (borderline case)
11-21 = Abnormal (case)

Bjelland, I., Dahl, A. A., Haug, T. T., & Neckelmann, D. (2002). The validity of the Hospital Anxiety and Depression Scale. An updated literature review. Journal of psychosomatic research, 52(2), 69–77.

Figure 13. The Cognitive Emotion Regulation Questionnaire

How do You Cognitively Regulate Emotions? (The Cognitive Emotion Regulation Questionnaire, CERQ)

Instructions

Emotion regulation is the ability to modulate – that is, change and alter one's emotional experiences. Emotion regulation is differentiated from suppression, i.e. 'bottling up' one's emotions, and is also different from catharsis, where one expresses or vents their emotions. This questionnaire assesses how well you regulate emotions through a range of cognitive processes such as positive reappraisal (rethinking the emotion-causing event more positively), acceptance, rumination and self-blame. State how often you think in the following manner when experiencing strong threatening or stressful life events.

	Almost never	Rarely	Occasionally	Frequently	Almost always
1. I feel that I am the one to blame for it.					
2. I feel that I am the one who is responsible for what has happened.					
3. I think about the mistakes I have made in this matter.					
4. I think that basically the cause my lie within myself.					
5. I think that I have to accept that this has happened.					
6. I think that I have to accept the situation.					
7. I think that I cannot change anything about it.					
8. I think I must learn to live with it.					
9. I often think about how I feel about what I have experienced.					
10. I am preoccupied with what I think and feel about what I have experienced.					
11. I want to understand why I feel the way I do about what I have experienced.					
12. I dwell upon the feelings the situation has evoked in me.					
13. I think of nicer things that what I have experienced.					
14. I think of pleasant things that have nothing to do with it.					
15. I think of something nice instead of what has happened.					
16. I think about pleasant experiences.					
17. I think about what I can do best.					

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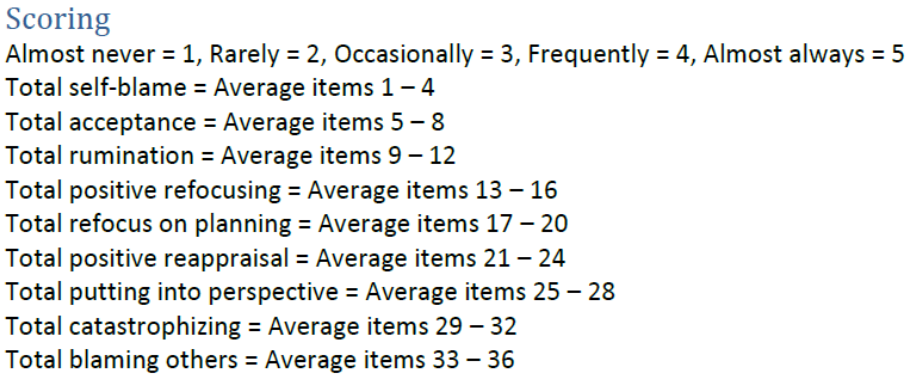
18. I think about how I can best cope with the situation.					
19. I think about how to change the situation.					
20. I think about a plan of what I can do best.					
21. I think I can learn something from the situation.					
22. I think that I can become a stronger person as a result of what has happened.					
23. I think that the situation also has its positive sides.					
24. I look for the positive sides to the matter.					
25. I think that it could have all been much worse.					
26. I think that other people go through much worse experiences.					
27. I think that it hasn't been too bad compared to other things.					
28. I tell myself that there are worse things in life.					
29. I often think that what I have experienced is much worse than what others have experienced.					
30. I keep thinking about how terrible it is what I have experienced.					
31. I often think that what I have experienced is the worst that can happen to a person.					
32. I continually think how horrible the situation has been.					
33. I feel that others are to blame for it.					
34. I feel that others are responsible for what has happened.					
35. I think about the mistakes others have made in this matter.					
36. I feel that basically the cause lies with others.					

Garnefski, N., and Kraaij, V. "The Cognitive Emotion Regulation Questionnaire." European Journal of Psychological Assessment, vol. 23, no. 3, 2007, pp. 141–149., doi:10.1027/1015-5759.23.3.141.

Figure 14. The Cognitive Emotion Regulation Questionnaire Scoring



Scoring

Almost never = 1, Rarely = 2, Occasionally = 3, Frequently = 4, Almost always = 5

Total self-blame = Average items 1 – 4

Total acceptance = Average items 5 – 8

Total rumination = Average items 9 – 12

Total positive refocusing = Average items 13 – 16

Total refocus on planning = Average items 17 – 20

Total positive reappraisal = Average items 21 – 24

Total putting into perspective = Average items 25 – 28

Total catastrophizing = Average items 29 – 32

Total blaming others = Average items 33 – 36

Garnefski, N., and Kraaij, V. "The Cognitive Emotion Regulation Questionnaire." European Journal of Psychological Assessment, vol. 23, no. 3, 2007, pp. 141–149., doi:10.1027/1015-5759.23.3.141.

Figure 15. The Abbreviated Profile of Mood States Questionnaire

Below is a list of words that describe feelings people have. Please **CIRCLE THE NUMBER THAT BEST DESCRIBES HOW YOU FEEL RIGHT NOW**.

	Not At All	A Little	Moderately	Quite a lot	Extremely
Tense	0	1	2	3	4
Angry	0	1	2	3	4
Worn Out	0	1	2	3	4
Unhappy	0	1	2	3	4
Proud	0	1	2	3	4
Lively	0	1	2	3	4
Confused	0	1	2	3	4
Sad	0	1	2	3	4
Active	0	1	2	3	4
On-edge	0	1	2	3	4
Grouchy	0	1	2	3	4
Ashamed	0	1	2	3	4
Energetic	0	1	2	3	4
Hopeless	0	1	2	3	4
Uneasy	0	1	2	3	4
Restless	0	1	2	3	4
Unable to concentrate	0	1	2	3	4
Fatigued	0	1	2	3	4
Competent	0	1	2	3	4
Annoyed	0	1	2	3	4
Discouraged	0	1	2	3	4
Resentful	0	1	2	3	4
Nervous	0	1	2	3	4
Miserable	0	1	2	3	4
Confident	0	1	2	3	4
Bitter	0	1	2	3	4
Exhausted	0	1	2	3	4
Anxious	0	1	2	3	4
Helpless	0	1	2	3	4
Weary	0	1	2	3	4
Satisfied	0	1	2	3	4
Bewildered	0	1	2	3	4
Furious	0	1	2	3	4
Full of Pep	0	1	2	3	4
Worthless	0	1	2	3	4
Forgetful	0	1	2	3	4
Vigorous	0	1	2	3	4
Uncertain about things	0	1	2	3	4
Bushed	0	1	2	3	4
Embarrassed	0	1	2	3	4

Grove, J. R., & Prapavessis, H. (1992). Preliminary evidence for the reliability and validity of an

abbreviated Profile of Mood States. International Journal of Sport Psychology, 23(2), 93–109.

Figure 16. The Profile of Mood States Questionnaire Total Mood Disturbance Score

$$TMD = Tension + Depression + Anger + Fatigue + Confusion - Vigor$$

Grove, J. R., & Prapavessis, H. (1992). Preliminary evidence for the reliability and validity of an abbreviated Profile of Mood States. International Journal of Sport Psychology, 23(2), 93–109.