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An examination of the moderating role of hormones on the relationship between sleep patterns and mood regulation

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AN EXAMINATION OF THE MODERATING ROLE OF HORMONES ON THE
RELATIONSHIP BETWEEN SLEEP PATTERNS AND MOOD REGULATION

A Thesis Submitted

in Partial Fulfillment

of the Requirements for the Designation

University Honors

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This Study by:

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An examination of the moderating role of hormones on the relationship between sleep patterns

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Abstract

A large body of research focuses on the relationship between sleep and mental health. Poor emotional regulation is costly to the individual's physical and mental health as well as the wellbeing of the community and economy. To further understand emotional regulation, the current study examines the potential interaction of sleep patterns and hormones. The goal of the current study was to isolate the potentially moderating role of hormones, specifically epinephrine and norepinephrine, as moderators in the relationship of sleep patterns and emotional regulation. The hypotheses were that (1) there would be a strong positive relationship between sleep patterns and emotional regulation, (2) there would be a strong positive moderating impact of epinephrine on the relationship between sleep patterns and emotional quality, and (3) there would be a strong negative moderating relationship between sleep patterns and emotional regulation. The sample included 420 participants, ages 24 to 74 years who completed telephone surveys, had a full biological assessment, and wore actigraphy watches to measure their sleep. Analyses were examined using a regression analysis and a moderation model using the PROCESS macro within SPSS. Results revealed a strong positive relationship for all sleep measures except for sleep time in partial support of the first hypothesis. Of the 16 moderation models ran, six were significant. Epinephrine had inconsistent results as a moderator in the relationship between sleep and emotional regulation. Norepinephrine also had inconsistent results in the relationship between sleep and emotional regulation. Hypothesis 2 and 3 were partially supported by the data. Clinical implications of this study are potential hormone-based therapies. Hormone levels may be an indicator of an emotional dysregulation in an individual. Practical implications of this study may be using sleep patterns and epinephrine/norepinephrine levels to assess the potential emotional

dysregulation habits of that individual. Further studies should investigate alternative

relationships of hormones in relation to sleep patterns and emotional regulation.

Keywords: sleep, depression, epinephrine, norepinephrine, mood regulation, depression

Sleep, Hormones and Emotional Regulation

Sleep deprivation and disruptions are a concerning public health problem (NIH, 2016). Poor sleep patterns impair our ability to function cognitively, physically and emotionally (Holdaway, Luebbe & Becker, 2018; Lang & Bradley, 2009; Walker, 2017). This worldwide lack of quality sleep has been researched more extensively in the past couple of decades due to its growing consequences. With sleep deprivation well over 50% in the US, drowsy driving is the cause of more traffic accidents than alcohol and drug related accidents combined (CDC, 2018; Walker, 2017). In adolescents, 24 consecutive hours of sleep loss results in a reaction time that is three times longer in comparison to when well rested (Czeisler, 2009). Specifically, cognitive abilities such as attention, cognitive performance, and quick reaction times are dramatically impaired when sleep is restricted to seven or fewer hours per night for a week or longer. This sleep debt leads to the accumulation of adenosine, a hormone that gradually increases the need to sleep (Walker, 2017). If enough adenosine accumulates, a person's ability to remain awake is nearly impossible (Walker, 2017). The amount of sleep time helps to determine how rested a person is, but is not the only factor.

Even with adequate sleep quantity, growing levels of sleep disorders are disruptive to sleep (Czeisler, 2009; NHBLI, 2003). Chronic insomnia, the inability to fall asleep or stay asleep, was observed in at least 10% of the United States population in the mid-nineties (NIH, 2018). This percentage continues to grow, revealing an estimated 50-70 million Americans' health and daily functions which are hampered by chronic sleep disorders (NIH, 2018). Today, there are more than 100 different diagnosable sleep disorders (Walker, 2017). Those disorders are determined by assessing both a person's sleep quality and quantity.

The primary focus of the following literature review is the relationship of sleep, both quantity and quality on emotional and mood regulation. An additional focus of the literature review is the role of hormones, specifically epinephrine and norepinephrine, in the linkages between sleep patterns and emotion regulatory capacities. The goal of the current study was to examine existing data on the relationship between sleep quality and quantity, depression and anxious arousal, and epinephrine and norepinephrine. The existing literature suggested that there may be a moderating role of epinephrine and norepinephrine on the interplay of sleep quantity and quality on emotional regulation.

Significance

As previously determined as an international issue, sleep deprivation and poor sleep patterns are negatively impacting economic, emotional and personal health. Moreover, the mental health crisis is also a major concern to a nation's well being. As countries are working to resolve both of these, a practical solution may be within each individual's genetic code. If this study supports a moderating relationship on the impact of sleep and emotional regulation, hormone-based intervention may reduce the suffering of millions. This study is relevant because its findings hold the potential to edify our current mental health prevention and treatment patterns.

Purpose

The goal of this project was to further qualify the relationship between sleep and emotional regulation with a specific emphasis on the body's physiological status. Because the impact of epinephrine and norepinephrine has not been frequently analyzed on the relationship between sleep and emotional regulation, this study's purpose was to provide additional insight

for further analysis of epinephrine and norepinephrine on the relationship between sleep quantity and emotional regulation.

Literature Review

The anticipated relationship, based on the literature present, was that the impact of sleep on emotional regulation would be moderated by epinephrine and norepinephrine. For this reason, the literature review will begin by assessing sleep patterns, emotional regulation and their relationship. After this, the role of epinephrine and norepinephrine on the body will introduced. The interactions of those hormones on both sleep and emotional wellbeing will then be integrated. Lastly, the current gaps in the literature are addressed and the section will be concluded with the hypotheses.

Indicators of Sleep Patterns

Sleep patterns are categorized by the nature and amount of sleep. The most widely used indicators of sleep quality in the current literature are sleep latency (the time it takes one to fall asleep after laying down), number of awakenings, and sleep efficiency (Ohayon et al., 2017). Sleep quantity is most commonly assessed as the amount of time spent sleeping. Some studies use polysomnography to include sleep spent in each of the two main portions of sleep: Rapid Eye Movement (REM) and Non-Rapid Eye Movement (NREM). REM sleep is an advanced sleep that occurs usually at the end of the sleep period and is when dreams occur (Ceisler, 2009; Sun et al., 2013). NREM sleep's primary functions are memory consolidation and the upkeep of basic bodily functions (Moreas, Miranda, Loures, Mainieri, & Marmora 2017; Walker, 2017). Because the body switches between NREM and REM sleep frequently and both offer many similar benefits, numerous researchers simply use time asleep to indicate sleep quantity. As noted

earlier, sleep disruptions are associated with negative physical health and behavioral outcomes, including the capacity to regulate emotions.

Emotional Regulation

Because sleep patterns and emotional wellbeing have such a significant relationship, emotions and their regulation are also integral to an individual's wellbeing (Holdaway et al., 2018; Lang & Bradley, 2009). In the United States, more young adults die by suicide than by any other cause (Holdaway et al., 2018). Emotional regulation is negatively correlated with suicidal thoughts and attempts, which argues for the immediacy in understanding how emotional regulation works. Emotional regulation is the body's intrinsic and extrinsic ways of assessing and moderating emotional reactions (Lang & Bradley, 2009; Thompson, 1991). Another way of defining emotional regulation is endocrine system's patterns for releasing hormones in response to what one is feeling and how they need to be feeling to appropriately fit the situation (Sutton, 2004). Indicators of emotional wellbeing have been widely explored and can be assessed by poor emotional wellbeing through scales such as the Mood And Symptom Questionnaire (MASQ) (Lang & Bradley, 2009; Watson et al., 1995). An additional indicator of poor emotional wellbeing is its economic impact. For example, in 2000, it was estimated that 110 million working days were lost in England due to poor mental health, stacking up to 9 billion pounds (over 11.88 billion dollars) annually (Walker, 2017). In America, the estimated loss of money due to depression-related factors was $210.5 billion, a 21% increase in five years (Greenberg et al., 2015). This financial loss is evidence of the cost of poor emotional regulation to society. With all of these factors indicating poor emotional regulation, it is important to also clearly understand emotions themselves.

A popular belief about emotions is that emotions are the brain's persuasive tool in gaining the body's needs (Greenspan et al., 1969; Lang & Bradley, 2009). The exact definition of an emotion is the "complex reaction pattern that involves experiential, behavioral and physiological elements." indicating the interaction of the external situation with the body's hormones (physiological; Nugent, 2013, p.233). Reliable evidence suggests that emotions are linked to the motivation to survive (NIH, 2010). These hormone circuits, the physiological element, were designed to reward the body of survival-encouraging behavior and punish it in reaction to risky behavior. Stated simply, the body used emotions to encourage survival and avoid danger. Additionally, research on genes and mental health has revealed that a gene involved in serotonin distribution, 5-HTT, moderates the relationship between stressful environments and mental health (Caspi et al., 2003). Emotional regulation is thus influenced by both genetic and cognitive reactions to situations. Patterns of rising depression and anxiety rates are seen in the United States (Nogueira & Paços, 2009).

One prominent pattern of emotional regulation is the tendency towards fear and anger. Researchers agree that this was evolutionarily vital in primitive years (Holdaway et al., 2018; Lang & Bradley, 2009; Sutton, 2004). Now, however, this stress reaction is often caused by social factors (Lang & Bradley, 2009). Numerous experiments have revealed that a person's fear (negative) response begins high and gradually dissipates whereas a desire (positive) response slowly rises (Lang & Bradley, 2009; Sutton, 2004). In people with poor emotional regulation, rumination often occurs. Rumination is the obsessive and destructive inability to stop thinking about certain concepts. When this stress response is activated, it is added to the pre-existing stress level, leading one to feel helpless. Several other patterns such as these create roadblocks

for healthily processing one's emotions (Holdaway et al., 2018; Sutton, 2004). The

categorization of these patterns is discussed below.

Emotional regulation disorders are often quantified by the following: general distress,

depression, anxiety, loss of interest, and high positive affect. Depression is characterized by

lowered mood which often results in fatigue and lack of interest in activities previously found

enjoyable. Anxiety is noted as an increase in the release of stress hormones, heart rate and blood

pressure. High positive affect is identified as feelings of intense joy, whereas positive affect

includes fatigue (Buckby et al., 2008). Each of these elements are in the Mood and Anxiety

Symptom Questionnaire (MASQ), a popular mental health survey (Watson et al., 1995). The

National Institutes of Health (2010) determined the MASQ as a reliable predictor of poor

emotional regulation. Poor sleep habits also play a heavy role in identifying emotional

regulation, although the exact relationship is not completely clear yet.

Sleep and Emotion Regulation

Ample evidence supports that sleep and emotion regulation are closely intertwined

(Ceisler, 2009; Holdaway, 2018; Ohayon et al., 2015; Walker, 2017). Sleep strips away the

emotional tone of a memory so that the content can be remembered (Walker, 2017). When a

stressful situation occurs and an individual is sleep deprived, the emotional sting of the memory

is not reduced. Sleep is responsible for assessing recent memories, tagging them as 'to be

remembered' or 'to be forgotten,' and placing the selected ones in the hippocampus (main

memory center located in the brain). In a repeated sleep-memory study, a quality afternoon nap

resulted in a 20% increase in memory compared to the group that stayed awake (Walker, 2017).

The electricity of sleep spindles that occur in NREM frees up memory space, increasing the

short-term memory threshold (Walker, 2017). Sleep rightly enhances confidence in memory, a

trait uncommon in people with poor emotional regulation patterns (Xie et al., 2010). The inability to remember as a consequence of sleep deprivation is one part of the tie between sleep and emotional regulation.

The second part of this relationship between sleep and emotional regulation is the brain's ability to selectively remember. Several studies in the past decade have unanimously supported that intention to remember astoundingly impacts memory (Lang & Bradley, 2009). That is, when the brain tags a memory as being 'of high importance', it will add that memory to the hippocampus for easy access. How does this relate to mental health? When a person has a strong stress response to a situation, it is an indicator that they need to store the memory. This frequently leads to rumination (Holdaway et al., 2018; Tsuno, Besset & Ritchie, 2005). This similarity is likely why psychiatric orders such as depression are the most prevalent of all sleep-related health conditions (Weich et al., 2010). Numerous studies have gathered large amounts of data to attempt to detangle the relationship between sleep and emotional regulation. The following are several examples and their results.

In the past three decades, a large body of research has explored the linkages between sleep disturbances and mental health issues (Holdaway et al., 2018; Tsuno et al., 2005; Walker, 2017). There is debate as to whether these are independent, comorbid issues, or whether they are both manifestations of a common underlying pathophysiology. Another contribution to the muddiness of deciphering the two disorders is the diagnostic element of sleep disturbances in depression. Sleep deprivation also contributes to inability to focus, increased daytime sleepiness and frustration, all symptoms of depression. Thus, wakefulness is often spent in guilty thought patterns and anxiety, increasing mental fatigue and continuing the pattern. A study found that 90% of its participants diagnosed with depression also reported having poor sleep quality (Tsuno

et al., 2005). A cross-sectional survey of over 750 US adults aged 20-90 revealed a strong association between number and severity of sleep problems and an increase in depression and anxiety, respectively (Xie et al., 2010). Given the bidirectional linkages between the two in reported literature, it is difficult to discern which contribution precedes the other.

The Relationship between Sleep and Emotional Regulation

In an effort to detangle the temporal relationship between emotional regulatory illnesses such as depression and sleep disturbances, numerous longitudinal studies have been conducted, most showing strong connections between these factors. Most commonly, a study of 1,271 male Johns Hopkins students were annually surveyed for indicators of poor sleep and depression. After 15 years, students who had reported poor sleep quality at the baseline showed a marked increase in depression compared to their well-resting counterparts. After the 15-year survey, this trend sharply increased every year (Weish, 2010). Studies such as these have been conducted all around the world.

A similar study was compiled in Sweden, with 1,870 adults aged 45-65 (Mallon, Broman & Hetta, 2000). After adjusting for symptoms of depression at baseline, a study conducted by the Health Maintenance Organization found the predictive period to be 3.5 years. This means that symptoms of depression were revealed after an average of three and a half years of poor sleep. Another noteworthy study in Switzerland was structured as an interview based 2-year, 6-wave follow up study of participants aged 19 and 20. In participants who reported insomnia for at least 2 weeks, depression symptoms emerged for almost every participant (Weich, 2010). Insomnia was not a precursor to anxiety. Closely related to insomnia is hypersomnia, defined as sleeping longer than is healthy (Montplaisir, Poirier & DeMontigny, 1990). Ample evidence supports that those who sleep for longer than ~9.5 hours per evening also have increased risk of depression

and anxiety (Montplaisir et al., 1990; Lopez, Barateu, Evangelista & Dauvilliers, 2017; Soehner,

Kaplan & Harvey, 2014).

Regardless of the temporal relationship between sleep and poor emotional regulation,

depression is the most prevalent of health conditions related to sleep. With suicide as the second

highest cause of death, understanding the biological indicators is increasingly relevant (NIH,

2016). The biological indicators of poor emotional regulation are seen in hormones such as

epinephrine and norepinephrine.

The Role of Hormones

Epinephrine. Hormone releases are widely understood to directly impact mental health

and thus emotional wellbeing (Nishihara & Mori, 1988; Thompson, 1991). Epinephrine, also

called adrenaline, is a hormone released when one is highly stimulated. Examples of this include

fear and anger. It is located in the Adrenal Medulla and brainstem (Callaham, Madsen, Barton,

Saunders & Pointer, 1992). Epinephrine increases blood pressure and heart rate. It is released

directly into the bloodstream and can be measured in blood, urine and saliva. Artificial

administration is used to stimulate the heart during allergic reactions (Callaham et al., 2003).

Extensive evidence suggests that epinephrine is crucial in the regulation of memory formation by

emotions and arousal (Korol, 2008). Epinephrine has a significant correlation with anxiety and

many other emotional regulation disorders (Callaham et al., 1992). Normally functioning

epinephrine levels gradually increase as one wakes up and significantly decreases as one

prepares for bed. Thus, it is related to sleep patterns and is disrupted when sleep is disrupted

(Walker, 2017). Further disruptions occur when epinephrine levels are abnormal.

Overproduction of epinephrine has been consistently linked to tenseness, palpitation, and

heightened aggression (Callaham et al., 1992). Increased epinephrine levels during sleep have

been shown to result in a higher frequency of waking up in the middle of the night and sleep

disturbances (Nishihara & Mori, 1986). A surplus of epinephrine is also observed in adults with

Alzheimer's, a disease that is believed to be closely linked to sleep wellness (Peskind et al.,

1999). However, another hormone similar to epinephrine is norepinephrine. This hormone plays

a less understood role with sleep and emotional regulation.

       Norepinephrine. Epinephrine's complement hormone, norepinephrine is involved in the

emotional links to memory and perception. During sleep, it is responsible for preserving

temporal order of emotional events. Increased norepinephrine is associated with attention and is

located in the same region of the brain that controls long-term memory (Callaham et al., 1992;

Lang & Bradley, 2009). Because of its energy, general activity and fear regulation qualities,

norepinephrine is suggested to be related to emotional regulation (Nutt et al, 2006). As REM

sleep disorders such as sleep paralysis and hallucinations are growing, medical professionals are

diagnosing serotonin and norepinephrine reuptake inhibitors (Lang & Bradley, 2009, Tsuno et

al., 2005; Walker, 2017). There is growing research about the role of norepinephrine in

interpersonal functioning, especially in anxiety and mood disorders (Nogueira & Paços, 2009).

       A decreased amount of norepinephrine may lead to emotional dysregulation, as it is a key

player in perception, modulation, and response to pain (Nogueira & Paços, 2009; Silvo, 2009).

Insufficient norepinephrine has been found to be linked to the inability to forget painful events

(Nogueira & Paços, 2009). A handful of studies have observed that upon re-exposure to

traumatic events, PTSD patients have a spiked increase of norepinephrine (Newport & Nemeroff,

2000). Similar studies suggest that increased (injected) norepinephrine is involved in panic

attacks and acute anxiety (Newport & Nemeroff, 2000). This directly contradicts other research

that argues norepinephrine's role in reducing depression and emotional regulation (Honzak,

1986; Nutt et al., 2006). One study revealed that alcohol abuse has been observed in African

Americans with elevated norepinephrine levels more than in Caucasian men (Ransome, Slopen

Karisson & Williams, 2017). A reduced amount of norepinephrine levels was observed in

alcohol abusers for Caucasian men. This study implies that norepinephrine may have a larger

impact on the African American population than the caucasian population. This study has not

been replicated many times and thus may not be conclusive. There is strong evidence in

numerous studies, including the aforementioned one, that norepinephrine is related to emotional

regulation and may indicate overall wellbeing.

Current Study and Gaps in the Literature

There is a need for research on the effects of epinephrine and norepinephrine on stress

reducing activities. One study found a reduction in the spread of HIV, cancer, depression and

sleep disorders as the direct result of epinephrine and norepinephrine (Moraes et al., 2017). This

study suggests hormones may serve as a potential moderator impacting all aspects of mental and

physical well-being. Although there was a significant relationship between mental wellbeing and

physical health as moderated by norepinephrine and epinephrine, ample replications of this have

yet to be conducted (Moraes et al., 2017). Although sleep is not the only indicator of physical

well-being, a handful of studies have shown a significant reduction in suicide ideation replicated.

Research on norepinephrine in relation to cardiovascular effects on emotional regulation is

conflicting. Research on the impact of norepinephrine on emotional regulation over an extended

period of time is also scarce. Emotional regulation is observed as having a close relationship with

sleep (Walker, 2017). The literature shows that hormones such as epinephrine and

norepinephrine predict sleep patterns. The goal of this study is to analyze the interplay of

emotional regulation, hormones, and sleep patterns. The hypothesis is that there will be a

moderating role of hormones on the relationship between sleep patterns and emotional regulation.

Current Research

The primary purpose of this research was to examine the relationship of sleep patterns (quality, quantity) and mood regulation (general distress, depression, anxiety, loss of interest, positive affect) as moderated by hormones (i.e., epinephrine and norepinephrine). Specifically, the current study focused on the effects of sleep patterns on mood disorders in people with regular hormonal levels versus those with elevated and decreased hormone levels. The goal of this research was to understand the potential factors, both biological and social, that contribute to disrupted mood regulation. The findings of the study would have implications for the general population to better understand the effects of their poor sleep patterns, and to encourage a governmentally involved solution to the national sleep deprivation issue.

Hypotheses

H1: It was expected that there will be a positive association between sleep patterns (i.e., overall sleep quality and sleep quantity) and emotional regulation (i.e., general distress, depression, anxiety, loss of interest, positive affect).

H2: It was expected that there would be a moderating role of epinephrine in the relationship between sleep patterns (sleep quality and sleep quantity) and emotional regulation (i.e., general distress, depression, anxiety, loss of interest, positive affect).

H3: It was expected that there would be a moderating role of norepinephrine in the relationship between sleep patterns (sleep quality and sleep quantity) and emotional regulation (i.e., general distress, depression, anxiety, loss of interest, positive affect).

Method

Participants

The sample that the data were obtained from is the second wave of the Midlife in the

United States (MIDUS) longitudinal study (2011). This study consisted of 1054 Americans,

though only a smaller subsample (420) included actigraphy recordings. Actigraphy was recorded

by placing a watch on the participant that measures blood pressure and sleep waves. Only those

with actigraphy data were used for the current study. The participants were derived from three

study pools, all of whom had completed the preceding Likert-type survey (1932). The first pool

(n=794) was the participants who completed the Daily Stress Project at the first time interval.

The Daily Stress Project is a national survey style questionnaire assessing the daily stress of

individuals. The second pool (n=1,048) consisted of sets of twins (for the sake of noting the

impact of genetics) and randomly dialed digits subjects who had participated in the Daily Stress

project at the second time interval and did not participate in the first time interval. Lastly, 180

Milwaukee residents comprised the third group who had completed the baseline MIDUS study

that began in 2005. The 420 participants used for the current project were between the ages of 25

and 74 (60% female) at baseline and were all living in the United States. Participants were

located through random digit dialed responses and were of varied ethnicities, gender, and

demographic backgrounds. None of the participants were institutionalized.

Procedure

Data were collected from 2004-2009 as the second wave of the MIDUS Biomarker study

(Ryff, Seeman & Weinstein, 2017). This study was longitudinal. It consisted of a telephone

survey for eight nights and a comprehensive biological assessment. The response rate for the

entirety of the study was 39.3%. Those who participated in the first study were invited to

participate in the second (MIDUS 2). The MIDUS 2 consisted of two sub-studies. With support

from the National Institute on Aging, there was the longitudinal survey sample ($n = 1,054$) and

the Milwaukee sample ($n = 201$). The Milwaukee sample included a comprehensive biological

assessment for further psychological analysis. This included urine and blood samples, as well as

actigraphy to measure sleep.

The procedure for data analysis was to run a regression analysis for each sleep and

emotional regulation measure to determine associations. Additionally, moderated regression was

examined using the PROCESS macro within SPSS (model 1). Specifically, moderated

regressions were examined to find moderations of hormones on the links between sleep and

emotional regulation. The R-squared was measured to assess the magnitude of the relationship.

Measures

Sleep quantity. Participants were asked to wear an Actiwatch-64 which began collecting

data at 7:00 am on the Tuesday after the initial visit until the day the participant returns the

watch. Time spent asleep was assessed using motion and heart rate. Weekday and weekend sleep

patterns were recorded. The current study only used weekday data.

Sleep quality. As previously stated, participants wore watches during their sleep. These

watches recorded the sleep onset latency, wake after sleep onset (WASO), total wake time, total

activity counts, average activity counts, wake bouts, wake percentage, and cumulative sleep

efficiency. For this study, sleep quality was analysed using onset latency, WASO and sleep

efficiency. The sleep onset latency is defined as the time between the 'Start Time' of a given rest

interval and the following 'Sleep Start Time,' measured in minutes. It was used to measure the

number of minutes between an individual's first attempt to sleep and the beginning of their first

sleep stage. WASO is measured by totaling the number of waking epochs between the beginning

and end of each sleep interval and multiplying that by the minutes of each epoch. It is used as an

indicator of poor sleep quality, as it is the time an individual is awake during their sleep time.

The overall sleep efficiency is the percentage of scored total sleep time to the minutes of the

sleep intervals minus invalid sleep. Invalid sleep is when a person's sleep status cannot be

confidently recorded in a specific stage. It is also assessed by dividing the Interval Duration

minus Total Invalid time from the Scored Total Sleep time multiplied by 100. Onset Latency,

WASO, and sleep efficiency were all used to measure sleep quality.

Hormones. Epinephrine and norepinephrine were collected through the urine using

High-Pressure Liquid Chromatography (Jiang & Machacek, 1987). Urine samples were taken

overnight, starting at 7:00 pm and ending at 7:00 am. Samples were recorded at the baseline and

again after 12 hours. This study included only the baseline epinephrine and norepinephrine

levels.

Emotional regulation. Emotional Regulation is measured by the Mood and Symptom

Questionnaire (MASQ). The MASQ has questions that assess general distress/depressive

symptoms and anxious arousal symptoms. An example of the anxious arousal items is "(how

much have you felt or experienced things this way during the past week, including today) had

trouble swallowing." All items were assessed on a 1-5 scale. The mean of the depressive and

anxious arousal scales was used as emotional regulation indicators for this study.

Results

All models were analyzed using SPSS version 23 and the PROCESS macro version 3.

For all models, actigraphy-reported sleep were the predictors, anxiety and depression were the

outcomes, and norepinephrine and epinephrine were the moderators. Of the 16 regression models

analysed, six showed a significant moderating role of hormones in the relationship between sleep

and emotional regulation. Descriptive statistics and bivariate correlation was examined among all

study measures.

The regression analysis for all emotional regulation and sleep patterns are found inTable

1. In partial support of hypothesis 1, all measurements of emotional regulation were significantly

associated with the measures of sleep quality and quantity except for sleep time ($p < .05$). The

association between sleep time and and depression was negative but not significant ($\beta = -.05$, *R*

squared $= .002$). Similarly, the association between onset latency and sleep time was negative

and not significant ($\beta = -.01$, *R squared* $= 0$). Sleep time was not a reliable indicator or result of

emotional regulation. Onset latency was significantly positively associated to both depression (β

$= .11$, *R squared* $= .01$, $p < .05$) and anxious arousal ($\beta = .07$, *R squared* $= .15$, $p < .01$). Sleep

efficiency was negatively associated with depression ($\beta = -.14$, *R squared* $= .02$, $p < .01$) but

positively associated with anxious arousal ($\beta = .2$, *R squared* $= .04$, $p < .001$). According to

these results, the longer it took a person to fall asleep, the lower were their depression was and

the higher was their anxious arousal symptoms. The WASO was positively associated with both

depression ($\beta = .12$, *R squared* $= .01$, $p < .05$) and anxious arousal ($\beta = .21$, *R squared* $= .04$, *p*

$< .001$). The more frequently an individual awoke during the sleep time was associated with

higher levels of both depression and anxious arousal. Overall, anxious arousal had more

significant correlations than depression did with sleep measurements such as wake-sleep onset,

onset latency and sleep efficiency ($p < .01$).

As noted in table 2, there was a statistically significant interaction between sleep time and

norepinephrine in the prediction of depression ($B = -.002$, $p < .05$, *ULCI* $= .003$, *LLCI* $= .0005$).

The total model R-squared was .021, ($p < .05$) and the interaction R-squared was .018

($p < .05$). As seen in figure 1, findings indicated a moderation of norepinephrine in the relation

between sleep time and depression. At the 10th and 25th percentiles i.e., at lower levels of

epinephrine, higher sleep time was associated with higher levels of depression. There was no

association at moderate and high levels of norepinephrine. At low levels of epinephrine, the more

minutes an individual sleeps, the more depression they may experience. This supported

hypothesis two, that norepinephrine would play a moderating role in the sleep quality and

depression, though not in the anticipated direction.

As indicated on table 2, there was a statistically significant interaction between sleep time

and epinephrine in the prediction of depression ($B = .01$, $p<.05$, $ULCI = .02$, $LLCI = 0.001$). The

total model R-squared was .02, ($p < .05$) and the interaction R-squared was .01 ($p<.05$) (see

Table 2). Findings did not indicate a moderating role of epinephrine at the 10th, 25th, 50th, 75th,

or 90th percentile. The Johnson-Neyman technique revealed that the moderating role of

epinephrine was only significant at the 98.6th percentile level of epinephrine ($B = 3.19$, $p<.05$).

Specifically, at extremely high levels of epinephrine, greater sleep time was linked to greater

levels of depressive symptoms (see Table 2). For normal levels, this data did not support

hypothesis two.

There was a statistically significant interaction of the sleep efficiency and epinephrine in

the prediction of anxiety ($B = .049$, $p < .01$, $ULCI = .086$, $LLCI = .013$) (see Table 2). The total

model R-squared was .04 ($p < .001$) and interaction R-squared was .009 ($p < .01$). Findings

indicated a moderation of epinephrine in the relation between sleep efficiency and anxiety, as

seen in figure 3. At low, moderate, and fairly high levels of epinephrine, lower sleep efficiency

was associated with increased anxiety. The only level of epinephrine that did not have an

association was at the 90th percentile (see Figure 3). For people with low levels of epinephrine, poor sleep efficiency was associated with increased levels of anxious arousal.

As indicated in table 2, there was a statistically significant interaction between sleep time and norepinephrine in the prediction of anxiety ($B = .001$, $p < .001$, $ULCI = .002$, $LLCI = .0003$). The total model R-squared was .02 ($p < .01$) and the interaction R-squared was .01 ($p < .001$). Findings did not indicate a moderation of norepinephrine at 10th, 25th, 50th, 75th or 90th percentiles. The Johnson-Neyman Technique revealed that the moderating role was significant at extremely high and extremely low levels of norepinephrine (see Figure 4). For only extreme levels of norepinephrine, when an individual has longer sleep time, they will have higher levels of anxious arousal. At standard levels of norepinephrine, norepinephrine did not change the relationship between sleep time and anxious arousal.

As indicated in table 2, there was a statistically significant interaction between onset latency and epinephrine as predictors of depression ($B = .03$, $p<.01$, $ULCI = -.01$, $LLCI = -.05$). The total model R-squared was .03 ($p < .01$) and the interaction R-squared was .02 ($p<.01$). Findings indicated a moderation of epinephrine in relation to onset latency and depression at all levels below the 90th percentile. As seen on figure 5, at high levels of epinephrine, as onset latency increases, depression also increases. With increased epinephrine, the longer it takes one to fall asleep, the more depression they have. This is in support of hypothesis one.

As noted on table 2, there was a statistically significant interaction between onset latency and epinephrine in the prediction of anxiety ($B = -.02$, $p<.05$, $ULCI = -.009$, $LLCI = .04$). The total model R-squared was .04 ($p < .01$) and the interaction R-squared was .02 ($p < .01$). Findings indicated a moderating effect of epinephrine in the links of onset latency and anxiety, as seen in figure 6. At all levels of epinephrine under the 90th percentile, the longer it takes one to

fall asleep, the higher anxiety they will have. There is no association at extremely high levels of epinephrine.

Discussion

Hypothesis 1, that emotional regulation measures would be associated with sleep measures, was supported. All levels of sleep quality were highly associated with depression and anxious arousal. Neither of the emotional regulation measures were associated with sleep time. This is supported by previous research that too much sleep can also be maladaptive (Montplaisir et al., 1990; Lopez et al., 2017; Soehner et al., 2014).

The goal of the study was to explore the moderation of hormones (epinephrine and norepinephrine) in the sleep to emotion regulation links. The findings support a moderating role of epinephrine on the relationship between sleep and emotional regulation. When epinephrine was analyzed as a moderator between sleep onset latency and depression, it was significant for all levels except for extremely large amounts of epinephrine. In Callaham et al.'s study, (1992), it was also discovered that large amounts of epinephrine increase anxiety. Other models, such as sleep time and depression revealed significant amounts for only extreme cases of epinephrine. This supports Walker's (2017) meta-analysis of abnormal levels of epinephrine disrupting sleep time. While the direction in Walker's meta-analysis was different than the current study, it does provide evidence of a relationship between sleep time and epinephrine. Further, at normal levels of epinephrine, only onset latency had a significant negative relationship with depression and anxiety. The literature provides clarity for this by acknowledging rumination as a maladaptive thought pattern that is strongest before bed (Holdaway et al., 2018; Sutton, 2004). Thus rumination may be both why onset latency is increased and is a symptom of poor emotional

regulation. This supports hypothesis two. The results suggest that epinephrine has a moderating role of sleep time and onset latency in increasing both depression and anxious arousal, consistent with Callaham et al.'s (1992) argument that anxiety increases anxiety and stress. The variations in levels at which it is significant for each variable may be due to a further complexity of the relationship between epinephrine and sleep time. It may also be due to a confounding variable such as insomnia. Nonetheless, the data suggest partial support for hypothesis two.

At extremely high levels of epinephrine, sleep time had an increased relationship with anxiety, but not with depression. While it is not strong enough to be conclusive, the data suggest that at extremely low amounts of norepinephrine, high levels of sleep time is associated with low levels of anxious arousal. In contrast, high levels of norepinephrine are associated with high levels of sleep time and high levels of anxious arousal. This is contrary to both Nogueira & Paços' (2009) study and Silvo's (2009) studies that found heightened acute anxiety was associated with elevated norepinephrine levels. However, in Newport & Nemeroff's study assessing PTSD responses, spikes in stress and anxiety were associated with spikes in norepinephrine. The only significant model that suggests a moderating role of norepinephrine at standard levels is on the relationship between sleep efficiency and anxiety. This model reveals that in individuals with low, moderate, and fairly high levels of norepinephrine, higher sleep efficiency is related to lower levels of anxious arousal. The literature may support this finding by observing the relationship between norepinephrine as fear-regulation (Nutt et al., 2006). This supports the previous literature and hypothesis three, though caution should be exercised with interpreting extremes of sleep.

Strengths

This study examined a novel exploration of the role of hormones in the links of sleep and mental health. The study identified a gap in the current literature and used appropriate measurements to reduce that gap. The study explored physiological conditions under which sleep patterns were linked to mental health. The ability to use both objective measures for sleep and hormone levels and subjective measures for emotional regulation reduces the common method bias (Jakobsen & Jensen, 2015). Another strength in this study was the use of actigraphy recorded measures. This allowed for a non-invasive and inexpensive way to gather information on sleep patterns. Since this is an objective sleep measure, it has a high validity. Multiple objective measures such as the sleep watch and urine sampling were used for this study. Additionally, this study had a large sample size and represented a wide range of demographics in the United States. The sample was diverse and one of the three groups of participants was randomly sampled, which made conclusions more generalizable and informative.

Limitations

While using actigraphy is an advanced means of measuring data, a stronger method would be polysomnography (NIH, 2018). This would reduce the invalid sleep markings and provide a more thorough understanding of the sleep patterns and sleep stages. Further, using both urine and blood samples would allow the hormone levels to be confirmed at baseline more so than only using one. The hormone levels were also only taken once per person as their baseline levels. If any individuals had circumstances that altered their stress levels and thus epinephrine or norepinephrine levels, it may have impacted the data. Another limitation to this study was only having one baseline measurement for epinephrine and norepinephrine levels.

This study did also not take into account confounding variables that may impact the relationship between sleep and mental health such as sleep disorders. Especially with hypersomnia, increased levels of sleep time or decreased levels of sleep efficiency may be indicators of a different issue which may mediate the relationship between sleep and mental health (Walker, 2017).

Implications and Future Directions

Practical implications for this study would be to use hormones as an intervention for sleep disruptions. Clinical implications for this study would be to use hormone-based interventions for individuals with emotional regulation disorders. Hormones could be used as a screening for checking the likelihood an individual may develop an emotional regulation disorder. Further attention should be paid to repeating the study with polysomnography and multiple measures of epinephrine and norepinephrine. Different hormones should be assessed and compared as both moderators and mediators in the relationship between sleep and emotional regulation. Another future direction of this study may be to isolate potentially confounding factors such as sleep disorders. A practical purpose of further studies can focus on a specific at-risk demographic such as third-shift workers, doctors, and astronauts.

<div align="center">

Conclusion

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A goal of the current study was to examine the risk enhancing or buffering roles of hormones epinephrine and norepinephrine in the relationship between sleep quantity/quality and emotional regulation. The big picture purpose of this direction is to identify a physiological solution in addition or replacement to the behavioral interventions of poor emotional regulation in the United States. With both norepinephrine and epinephrine as statistically significant moderators in the role of sleep patterns and emotional regulation, further research may unlock an

intervention that resolves emotional dysregulation more than the current interventions. Likewise,

understanding the interactions of sleep and emotional regulation may enhance the quality of life

for every individual. This study began to close the gap in understanding the moderating role of

hormones on links with sleep and emotional regulation and has opened the door for happier,

healthier Americans.

References

Anxiety. (2003) Miller-Keane Encyclopedia and Dictionary of Medicine, *Nursing, and Allied Health, 7*.

Retrieved from https://medical-dictionary.thefreedictionary.com/anxiety.

Any Mental Illness (AMI) Among Adults. (n.d.). Retrieved October 23, 2015, from

http://www.nimh.nih.gov/health/statistics/prevalence/any-mental-illness-ami-among-adults.shtml

Baekeland, F., Schenker, V. J., Schenker, A. C., & Lasky, R. (1969). Urinary excretion of epinephrine,

norepinephrine, dopamine, and tryptamine during sleep and wakefulness. *Psychopharmacologia*,

14(5), 359-370. DOI:10.1007/BF00403576

Callaham, M., Madsen, C.D., Barton, C.W., Saunders, C.E., Pointer, J. (1992). A randomized clinical

trial of high-dose epinephrine and norepinephrine vs standard-dost epinephrine in prehospital

cardiac arrest. *Jama,* 268(19): 2667-72.

Caspi, A., Sugden, K, Moffitt, T., Taylor, A., Craig, I., Harrington, H., McClay, J., Mill, J., Martin, J.,

Braithwaite, A., Poulton, R. (2003). Influence of Life Stress on Depression: Moderation by a

Polymorphism in the 5-HTT Gene. *Science, 301*(5631) 386-389.

Czeisler, C. A. (2009). Medical and Genetic Differences in the Adverse Impact of Sleep Loss on

Performance: Ethical Considerations for the Medical Profession. *Transactions of the American*

Clinical and Climatological Association, 120, 249–285.

Czeisler, C. A., Wickwire, E., Barger, L., Dement, W., Gamble, K., Hartenbaum, N., Ohayon, M.,

Pelayo, R., Phillips, B., Strohl, K., Tefft, B., Rajaratnam, S., Malhotra, R., Whinton, K.,

Hirshkowitz, M. (2016). Sleep-deprived motor vehicle operators are unfit to drive: a

multidisciplinary expert consensus statement on drowsy driving. *Sleep Health, 2*(2), 94-99. DOI:

10.1016/j.sleh.2016.04.003

Depression Anxiety Stress Scale. (2013, December 11). Retrieved September 15, 2018, from

https://www.sralab.org/rehabilitation-measures/depression-anxiety-stress-scale

Dodt, C., Breckling, U., Derad, I., Fehm, H., Born, J. (1997). Plasma Epinephrine and Norepinephrine

Concentrations of Healthy Humans Associated With Nighttime Sleep and Morning Arousal.

American Heart Association Journals, 30, 71-76

DOI: 10.1161/01.HYP.30.1.71

Epinephrine. (2003) Miller-Keane Encyclopedia and Dictionary of Medicine, *Nursing, and Allied*

Health, 7, (2003). Retrieved on September 15, 2018, from https://medical-

dictionary.thefreedictionary.com/epinephrine

Greenberg, P., Fournier, A.A., Sisitsky, T, Pike, C., Kessler, R. (2015) The Economic Burden of Adults

With Major Depressive Disorder in the United States (2005 and 2010). *J Clin Psychiatry,*

76(2):155-162 DOI:10.4088/JCP.14m09298

Greenspan, K., Schildkraut, J. J., Gordon, E. K., Levy, B., & Durell, J. (1969). Catecholamine

metabolism in affective disorders: II. Norepinephrine, normetanephrine, epinephrine,

metanephrine, and VMA excretion in hypomanic patients. *Archives of General Psychiatry,*

21(6), 710-716. DOI:10.1001/archpsyc.1969.01740240070009

Gross, J.J., & John, O.P. (2003). Individual differences in two emotion regulation processes:

Implications for affect, relationships, and well-being. *Journal of Personality and Social*

Psychology, 85, 348-362.

Hayes, A. F. (2012). PROCESS: A versatile computational tool for observed variable mediation,

moderation, and conditional process modeling [White paper]. Retrieved from

http://www.afhayes.com/ public/process2012.pdf

Holdaway, A., Luebbe, A., Becker, S. (2018). Rumination in relation to suicide risk, ideation, and

   attempts: Exacerbation by poor sleep quality?. *Journal of Affective Disorders, 236*, 6-13. DOI:

   10.1016.j.jad.2018.04.087

Honzak, R., Veselkova, A., Prat, V., & Brodan, V. (1986). 'The norepinephrine-to-epinephrine ratio and

   suicidal ideation in depression': Comment. *The American Journal of Psychiatry*, *143(5)*, 683-

   684. doi:10.1176/ajp.143.5.683-a

Jakobsen, M. & Jensen, R. (2015) Common Method Bias inPublic Management Studies, International

   Public Management Journal, 18:1, 3-30, DOI:10.1080/10967494.2014.997906

Jiang NS, Machacek D. (1987) Measurement of catecholamines in blood and urine by liquid

   chromatography with amperometric detection. *In Progress in HPLC, 2,* 397-426

Korol, D. L., & Gold, P. E. (2008). Epinephrine converts long-term potentiation from transient to

   durable form in awake rats. *Hippocampus, 18(1),* 81-91. DOI:10.1002/hipo.20372

Likert, R. (1932). A Technique for the Measurement of Attitudes. *Archives of Psychology*, *140*, 1–55.

Lucam J. Moraes, Márcia B. Miranda, Liliany F. Loures, Alessandra G. Mainieri & Cláudia Helena C.

   Mármora (2018) A systematic review of psychoneuroimmunology-based interventions,

   Psychology, Health & Medicine, 23(6), 635-652.

   DOI: 10.1080/13548506.2017.1417607

Lang, P. & Bradley, M. (2009). Emotion and the motivational brain. *Biological Psychology*, *84(3),* 437-

   450. DOI: 10.1016/2009.10.007

Likert, R. (1932). A Technique for the Measurement of Attitudes. *Archives of Psychology, 140*, 1–55.

Lopez, R., Barateu, L., Evangelista, E., Dauvilliers, Y. (2017) Depression and Hypersomnia: A Complex

   Association. *Sleep Medicine Clinics. 12*(3), 395. DOI: 10.1016.2017.03.016

Mallon, L. Broman, J.E. & Hetta, J. (2000). Relationship Between Insomnia, Depression, and Mortality: A 12-Year Follow-Up of Older Adults in the Community. *International Psychogeriatric Association,* 12(3), 295-306. DOI: 10.1017.S104161002000006414

Montplaisir, J., Poirier, G., & DeMontigny, C. (1990). HLA antigens in depression and hypersomnia. *Biological Psychiatry, 27*(6), 664–666. DOI: 10.1016/0006-3223(90)90536

Moraes, L., Miranda, M., Loures, L., Mainieri, Al., Marmora, C. (2017). A systematic review of psychoneuroimmunology-based interventions. *Psychology, Health & Medicine, 23(6)*, 235-252 DOI:10.1080/13548506.2017.1417607

Nishihara, K., & Mori, K. (1986). High urinary excretion of epinephrine during day sleep associated with sleep disturbance. *Journal of Human Ergology, 15(2),* 155-162.

Nishihara, K., & Mori, K. (1988). The Levels of Urinary Epinephrine During REM Sleep Deprivation At Night. *National Institute of Industrial Health, 17*, 81-86

Nogueira, S., & Paços, A. (2009). Neurochemistry : Molecular Aspects, Cellular Aspects and Clinical Applications. New York: Nova Science Publishers, Inc.

Newport, D. & Nemeroff, J. (2000) Neurobiology of Post-Traumatic Stress Disorders. *Current Opinion in Neurobiology, 10*, 211-218

Nugent, Pam M.S., "Emotion," in PsychologyDictionary.org. (April 7, 2013). Retrieved from https://psychologydictionary.org/emotion

Nutt, D. J., Baldwin, D. S., Clayton, A. H., Elgie, R., Lecrubier, Y., Montejo, A. L., Tylee, A. (2006). The role of dopamine and norepinephrine in depression and antidepressant treatment. *Journal of Clinical Psychiatry, 67*(6), 46-49.

Ohayon, M., Wickwire, E., Hirshkowitx, M, Altert, S., Avidan, A., Daly, F., Dauvilliers, Y., Ferri, R., Fung, C., Gonzal, D., Hazen, N., Kyrstal, A., Lichstein, K., Mallampalli, M., Plazzi, G.,

Rawding, R., Scheer, F., Somers, V., Vitello, M. (2017). National Sleep Foundation's sleep

   quality recommendations: first report. *Sleep Health, 3(*1), 6-19. DOI: 10.1016/j.sleh.2016.11.006

Paine, N., Watkins, L., Blumenthal, J., Kuhn, C., Sherwood, A. (2015) Association of Depressive and

   Anxiety Symptoms With 24-Hour Urinary Catecholamines in Individuals With Untreated High

   Blood Pressure. *Psychosomatic Medicine, 77(*2), 136-144 DOI:

   10.1097/PSY.0000000000000144

Peskind, E., Elrod, R., Dobie, D., Pascualy, M., Petrie, E., Jensen, C., Brodkin, K., Murray, S., Veith, R.,

   Raskind, M. (1999) Cerebrospinal Fluid Epinephrine in Alzheimer's Disease and Normal Aging.

   Neuropsychopharmacology, 19(6), 465-471

   DOI: 10.1016/S0893-133X(98)00054-2.

Ransome, Y., Slopen, N., Karisson, O., Williams, D. (2017). The Association Between Alcohol Abuse

   and Neuroendocrine System Dysregulation: Race Differences in a National Sample. *Brain,

   Behavior, and Immunity, 66,* 313-321. DOI: 10.1016.2017.07.154

Ryff, C.D., Seeman, T. & Weinstein, M. (2017) Midlife in the United States (MIDUS 2): Biomarker

   Project, 2004-2009. Ann Arbor, MI: *Inter-university Consortium for Political and Social

   Research* DOI:10.3886/ICPSR29282.v7

Sun, X., Song, H.,, Yang, T., Zhang, L., Zhao, L., Yang, J., Bai, J. (2013). Effects of sleep deprivation

   on neuroendocrine hormones in servicemen. *Sleep and Biological Rhythms*, *11*(4*)*

   DOI: 10.1111/sbr.12027

Soehner, A. M., Kaplan, K. A., & Harvey, A. G. (2014). Prevalence and clinical correlates of co-

   occurring insomnia and hypersomnia symptoms in depression. *Journal of Affective Disorders*,

   167, 93–97. https://doi-org.proxy.lib.uni.edu/10.1016/j.jad.2014.05.060

Sutton, R. (2004) Emotional Regulation Goals and Strategies of Teachers. *Social Psychology of Education, 7,* 379-398

Thompson, R.A. (1991). Emotional Regulation and Emotional Development. *Educational Psychology Review 3(4)* 269. DOI: 10.1007/BF01319934

Tsuno, N., Besset, A. & Ritchie, K. (2005). Sleep and depression. *J Clin Psychiatry, 66*

Watson, D., Clark, L.A., Weber, K., Assenheimer, J.S., Strauss, M.E., and McCormick, R.A. (1995). Testing a Tripartite Model: II. Exploring the Symptom Structure of Anxiety and Depression in Student, Adult, and Patient Samples. *Journal of Abnormal Psychology, 104(1)*, 15-25.

Weich, S. (2010). Sleep, Health and Society: From Aetiology to Public Health. The Epidemiology of sleep and depression. (140-162) *Oxford, NY: Oxford University Press.* DOI: 10.1093/acprof:oso/9780199566594.003.0008

Wong, D. L., Tai, T. C., Wong-Faull, D. C., Claycomb, R., Meloni, E. G., Myers, K. M., Kvetnansky, R. (2012). Epinephrine: A short- and long-term regulator of stress and development of illness: A potential new role for epinephrine in stress. *Cellular and Molecular Neurobiology, 32(5),* 737-748. DOI:10.1007/s10571-011-9768-0

Xin, A., Kakkar, R., Teodorescu, M.C., Herpel, L., Krishnan, V., Teodorescu, M. (2010). Sleep and Respiratory Diseases. *Sleep, Health and Society: From Aetiology to Public Health. Sleep and respiratory diseases.* (182-194) Oxford, NY: Oxford University Press.

Table 1

Regression of Sleep, Hormones and Emotional Regulation

Depression	B(se)	β	R^2	Anxious Arousal	B(se)	β	R^2
Sleep Time	-.005(.005)	-.047	.002	Sleep Time	.001(.004)	-.013	0
Onset Latency	.02(.01)*	.11	.01	Onset Latency	.07(.01)	.15	.02
Sleep Efficiency	-.09(.03)**	-.14	.02	Sleep Efficiency	.1(.03)***	.2	.04
WASO	.03(.01)*	.12	.01	WASO	.05(.01)***	.21	.04

Note. * is significant at the 0.05 level, ** is significant at the .01 level, *** is significant at the .001 level.

Table 2

Model of Epinephrine and Norepinephrine as Moderators in the Relationship between Sleep and Depression.

Variables		B(se)	t	LLCI	ULCI	R-Squared
Depression	Constant	19.114(.375)***	50.9973	18.377	19.85	.021*
	Sleep Time	-.0057(.006)	-9.518	-.0175	.006	
	Norepinephrine	0.0345(.0375)	.9185	-.04	0.108	
	Interaction	0.0015(.0005)*	2.8743	.0005	.0025	.018*
Depression	Constant	19.027(.356)	53.443	18.327	19.727	.02*
	Sleep Time	.004(.006)	-.651	-.016	.008	
	Epinephrine	.658(.475)	1.385	-.276	1.591	
	Interaction	.0118(.005)*	2.204	.001	.022	.01*

		B(se)	t	LLCI	ULCI	R-Squared
Depression	Constant	18.956(.352)***	53.896	18.264	19.647	.04**
	Onset Latency	.034(.01)***	3.337	.014	.055	
	Epinephrine	.39(.448)	.87	-.491	1.27	
	Interaction	-.0288(.009)**	-3.113	-.047	-.011	.02**

Note. * is significant at the 0.05 level, ** is significant at the .01 level, *** is significant at the .001 level.

Table 3

Model of Epinephrine and Norepinephrine as Moderators in the Relationship between Sleep and Anxious Arousal.

Variables		B(se)	t	LLCI	ULCI	R-Squared
Anxiety	Constant	21.973(.277)***	79.335	21.429	22.518	.04***
	Sleep Efficiency	-.101(.025)	5.26	-4.066	-.052	
	Epinephrine	.09(.338)	.2676	-.5741	.7549	
	Interaction	.049(.019)**	2.663	.013	.086	.009***
Anxiety	Constant	22.121(.287)***	77.066	21.557	22.685	.02**
	Sleep Time	-.0009(.004)	-.214	-.009	.007	
	Norepinephrine	.038(.033)	1.133	-.028	.103	
	Interaction	.0011(.0004)**	2.599	.0003	.002	.01***
Anxiety	Constant	21.977(.278)***	79.195	21.431	22.52	.037*
	Onset Latency	0.3(.01)**	3.147	.011	.049	
	Epinephrine	.0818(.342)	.239	-.591	.755	
	Interaction	.0246(.008)**	-3.057	-.041	-.009	.02**

Note. * is significant at the 0.05 level, ** is significant at the .01 level, *** is significant at the .001 level.
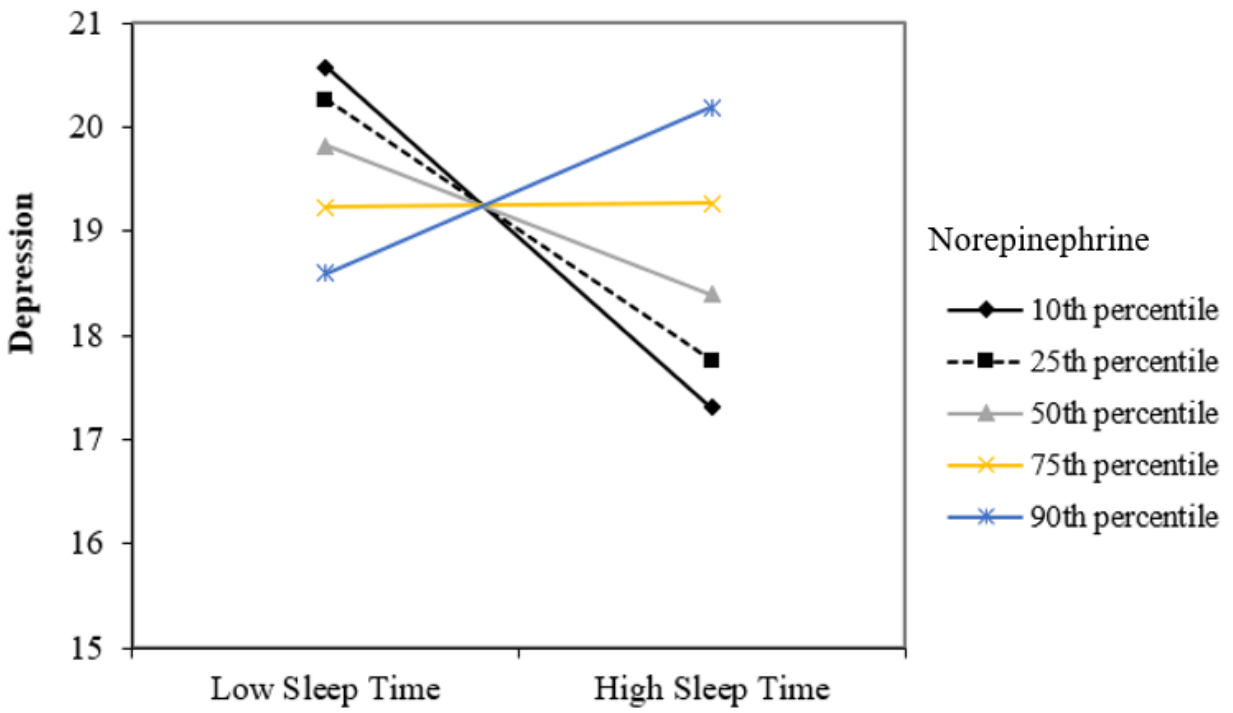
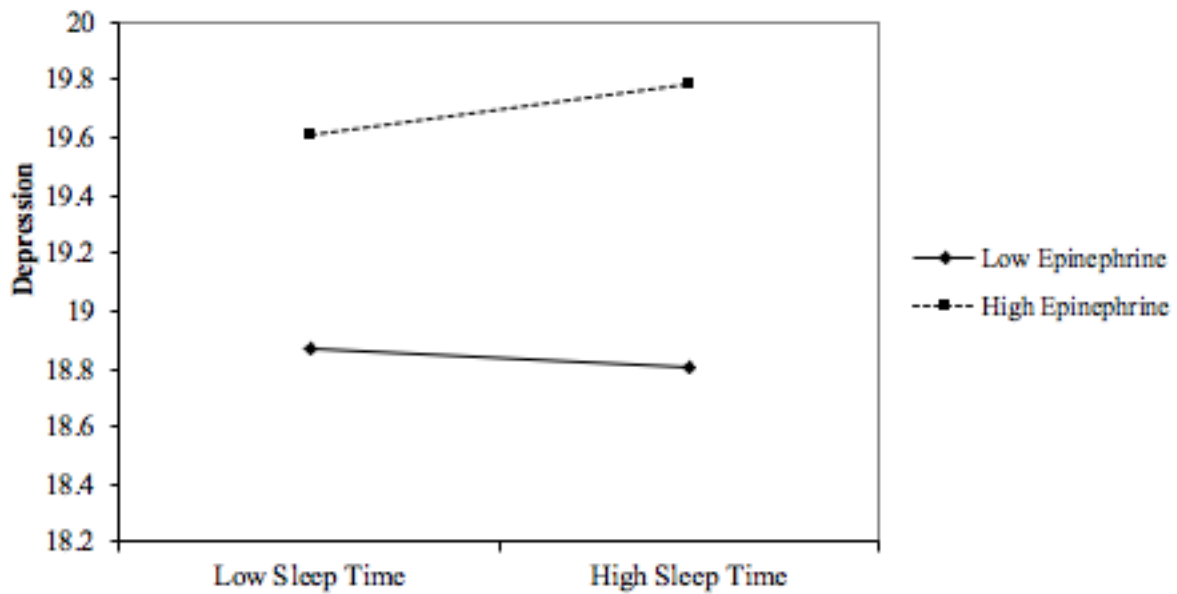*Figure 1*. The moderating effects of Norepinephrine on Sleep Time and Depression.

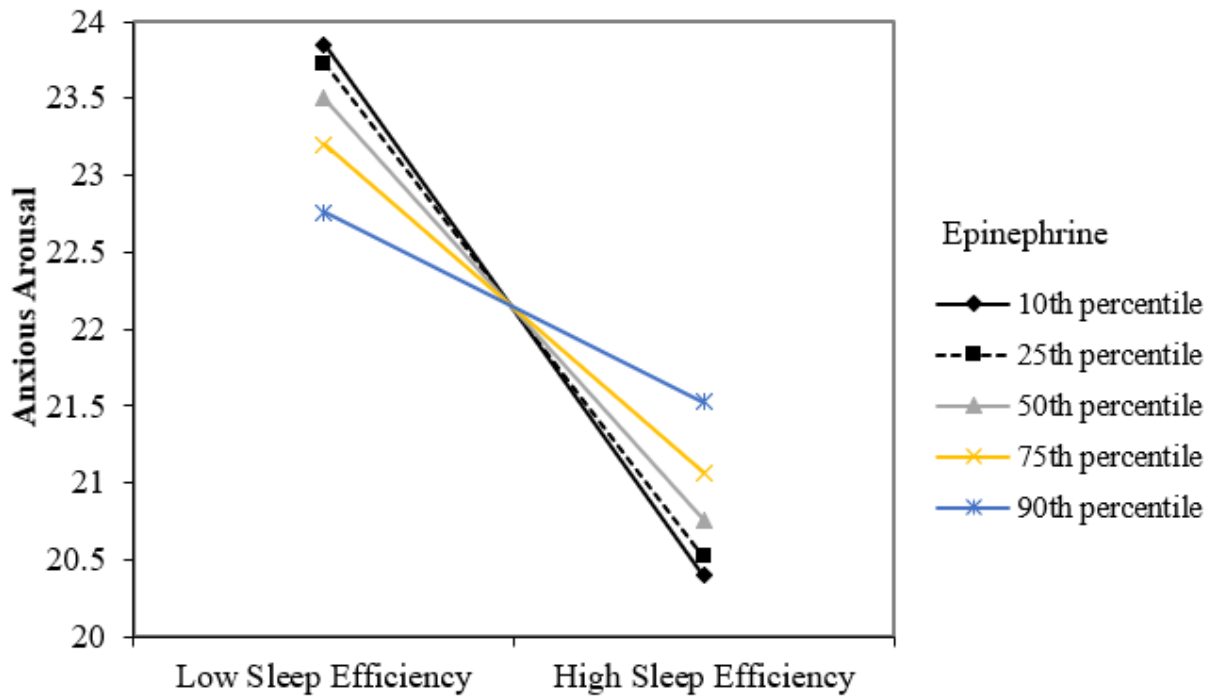*Figure 2*. The moderating effects of Epinephrine on Sleep Time and Depression.

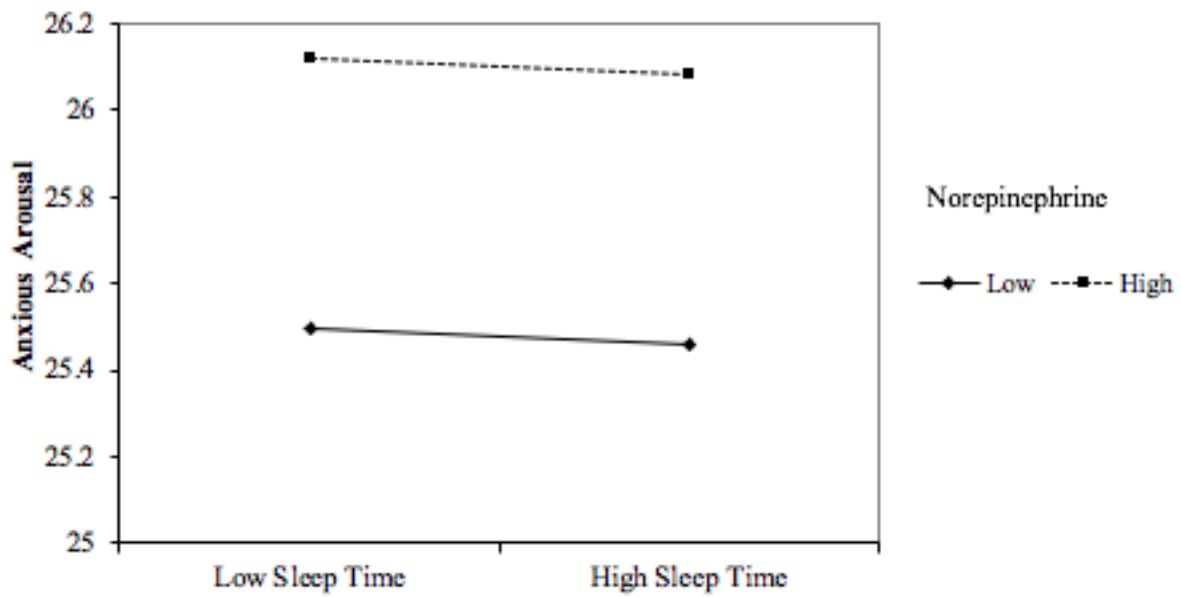*Figure 3*. The Moderating effects of Epinephrine on Sleep Efficiency and Anxious Arousal.

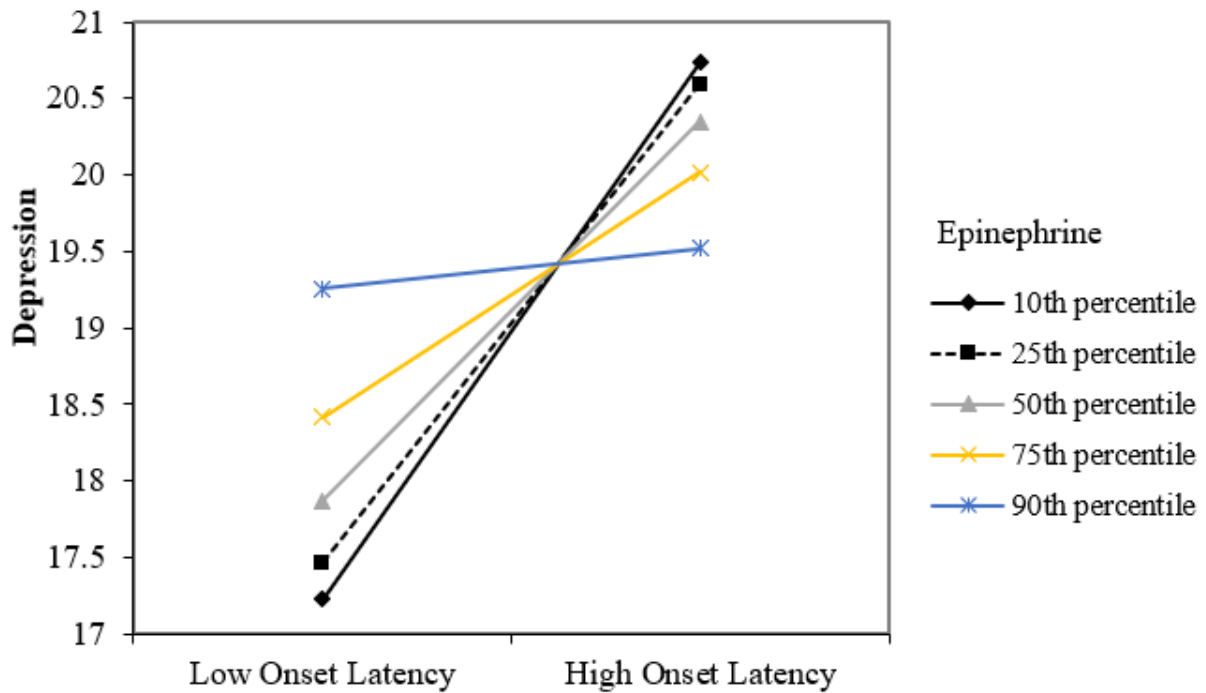Figure 4. The Moderating effects of Norepinephrine on Sleep Time and Anxious Arousal

Figure 5. The Moderating Impacts of Epinephrine on Onset Latency and Depression.
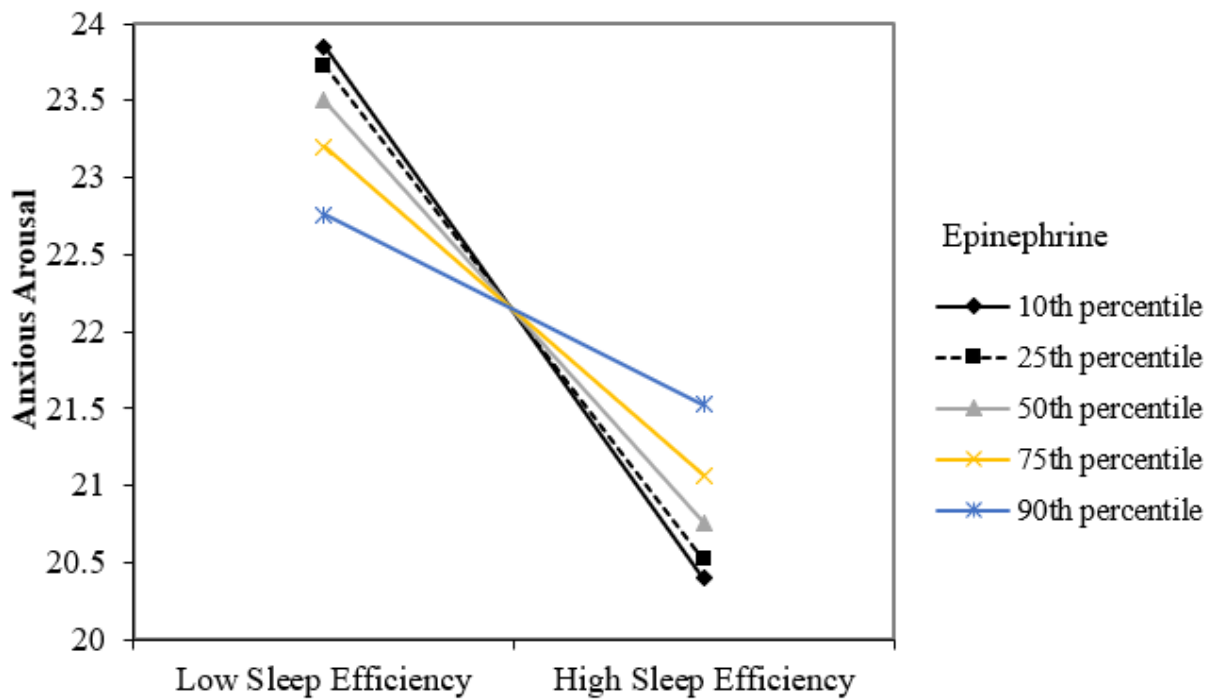

Figure 6. The Moderating Impacts of Epinephrine on Onset Latency and Anxious Arousal.