THE EFFECT OF SLEEP QUALITY ON REWARD MOTIVATION AND DEPRESSIVE SYMPTOMS IN ADOLESCENT GIRLS

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A THESIS

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Title: The Effect of Sleep Quality on Reward Motivation and Depressive Symptoms in Adolescent Girls

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Depression is one of the leading causes of global disease, (Kessler et al., 2005) and adolescents face a higher likelihood of diagnosis (Fleming and Offord, 1990). Because of its prevalence and recurrent nature (Pine et al., 1999), factors related to depression have been researched for many years. Sleep health is one of the many factors established to be a correlate of depressive disorders. While worse sleep is known to increase the risk of depressive symptomatology (Nutt, Wilson & Paterson, 2022), less is understood about the mechanisms that mediate this relationship. Connected to both sleep and depression are reward processes (Nestler & Carlezon, 2006; Shankman et al., 2007; Bress et al., 2012; Heshmati & Russo, 2015). One particular aspect of reward processing is reward motivation, or desire-driven action for a reward. Because depression rates in adolescent women are especially prevalent, finding risk factors and providing adequate intervention for symptom onset is a noble goal. My aim is to find the associations between sleep quality, depressive symptoms, and reward motivation. More specifically, I aim to determine if reward motivation mediates sleep quality and depression symptomatology. After screening criteria were met, data were collected from 120 adolescent girls. Of this sample, 65% were Black, 27% were White, and 8% were multi-racial (Casement et al., 2019). Through a series of linear regressions, I found nonsignificant relationships between the variables of depression, sleep quality, and hypothesized mediating factor of reward motivation. Results from this study aim to provide context for depression prevention and intervention and future research direction.

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Chapter 1: Introduction

Major Depressive Disorder (MDD) is a relatively common mood disorder and is characterized by a persistent, low mood lasting at least two weeks. Symptoms of MDD can include lethargy, appetite changes, weight fluctuation, sleep disruptions, feelings of worthlessness, and sometimes thoughts of suicide (American Psychiatric Association, 2013). People who are diagnosed with MDD will also often experience a loss of interest in activities they used to enjoy (Judd, Akiskal, & Paulus, 1997). Furthermore, Kessler et al. (2005) report that approximately 16.6% of adults are diagnosed with MDD within their lifetime.

Critically, adolescents are at a greater risk for diagnosis of MDD (Saluja et al., 2004). In a school survey of students ages 11-15, 18% of participants self-reported symptoms of MDD. Moreover, a diagnosis of depression during adolescence strongly predicts the presence of depression in adulthood (Pine, Cohen, Cohen, and Brooke 1999). Not only is depression most prevalent amongst adolescents, but it is also disproportionately distributed between the sexes. Of those who report depressive symptomatology, a greater proportion of young women (25%) were affected than young men (10%), and from the beginning of puberty onward, rates of depressive disorders are two to three times more common in women than in men (Saluja et al., 2004; Leibenluft, 1999). One research extension from Kessler et al. (2005) found that the lifetime risk of any mood disorder is 1.5 times higher for women.

Unfortunately, adolescent depression is frequently ignored or misunderstood, with its symptomatology incorrectly explained through teenage angst or other disorders (i.e. conduct, attentional, or substance use disorders). As depression is highly correlated with the risk of suicide, this mischaracterization can have potentially fatal consequences (Birmaher et al., 1996). Alongside the increased risk of suicide, those who experience symptoms of depression at an

early age are far more likely to struggle with the disorder throughout their lives (Lewinsohn et al., 1999). Depression is also associated with psychopathological comorbidities including anxiety, conduct disorders, substance abuse disorders, and risk behaviors such as having unprotected sex (Brooks et al., 2002). Depression also affects interpersonal relationships with correlations found between diagnoses and fights with peers (Ryan et al., 1987) alongside deficits in overall physical health (Beekman et al., 1997).

There are a variety of potential explanations for depressive symptoms, however, one particular correlate is sleep. Within the past twenty years, poor sleep has been reconceptualized as a risk factor for the onset of mental health difficulties and disorders. For example, sleep difficulty has been established as a correlate for negative affect (Finan et al., 2015), increased burnout (Vela-Bueno et al., 2008), social isolation (Yu et al., 2018), increased loneliness (Xu et al., 2012), and deficits in cognitive ability (McCoy & Strecker, 2011). The relationship between poor sleep and worse mental health also translates to adolescent populations. A longitudinal study published in 2009 determined that sleep disruption in adolescence is correlated with later declines in mental health (Kaneita et al, 2009).

Critically, sleep has a profound association with depressive symptoms. In fact, approximately 90% of people diagnosed with MDD have experienced some type of sleep disturbance (Geoffroy, et al., 2018). Furthermore, researchers Breslau et al. (1996) found that complaints of insomnia for two weeks or more were a marker for the onset of MDD in their study with adult participants. Moreover, research from Chang et al. (1997) demonstrates that the effects of poor sleep persist years after it is first recognized. Through a longitudinal study of graduating men, researchers found that complaints of insomnia were associated with an increased risk for MDD and psychological distress 34 years later. Just as there is evidence that

poor sleep worsens depression symptoms, researchers have also found that treatment for poor sleep mitigates depression. For example, in a study by Cheng et al. (2019), researchers found that the treatment of insomnia through digital cognitive behavioral therapy mitigated depression symptoms a year following the treatment.

Ties between sleep and depression exist in populations of mentally healthy patients as well as adolescents. In 2014, researchers Tkachenko et al. found that neurotypical subjects who reported sleep disruption also reported anxiety and depression at increased rates. Working alongside a sample of adolescents, Roberts and Duong (2013) found that insomnia increased the risk of clinical depression 2-3-fold. In correspondence with these findings, researchers have discovered successful sleep interventions that limit the effects of worse sleep on depressive symptoms. Researchers Casement et al. (2020) randomly assigned two groups of depressed female adolescents to a typical sleep or sleep extension group. Through actigraphy, sleep diary, and subjective sleep data, they found that a sleep extension of just one hour decreased symptoms of both depression and anhedonia.

One specific measure of sleep is subjective sleep quality. Like the previously mentioned measures of sleep, sleep quality is also correlated with depressive symptoms. Illustrating this, Norra et al. (2012), found that participants with worse sleep quality had more symptoms of depression and that improving sleep may improve depressive symptoms. Worse sleep quality is also correlated with suicidality. A study by Ağargün, Kara, & Solmaz (1997) found that worse subjective sleep quality is associated with increases in suicidal behavior. Given the strength of the relationship between poor sleep quality, depression, and suicidal symptoms, mediators of sleep and depression must be investigated.

Reward motivation could play a key role in the mediation of these two factors. It is well established that reward motivation and sensitivity are highly associated with depression symptomatology (Nestler & Carlezon, 2006; Admon & Pizzagalli, 2015; Shankman et al., 2007; Boland et al., 2020). One key symptom of depression is a lack of pleasure or anhedonia. While there are several biomarkers of reward response, researchers Bress et al. (2012) addressed reward response specifically in feedback negativity (FN). Similar to adult samples, higher scores of depression in adolescence were correlated with decreased FN response. Further research, like that from Heshmati & Russo (2015), has provided a more concrete biological framework for reward, detailing the role of the nucleus accumbens and surrounding neuroanatomy activated in reward sequences but dysregulated in the presence of depression.

Central to the subject matter of this paper, the relationship between depression and reward generalizes to adolescents. Through a verbal memory task with varied rewards, Henriques & Davidson (2000) evaluated reward motivation between depressed and nondepressed individuals. They found that depressed adolescents had a decreased response to rewards. This is consistent with the literature surrounding the nature of anhedonia in depressed individuals. These findings are also consistent with the leading hypothesis that the left prefrontal hypoactivation in depressed populations reflects a decrease in approach-related behavior.

Less is understood about the relationship between sleep quality and reward, however, preliminary research demonstrates that worse sleep is associated with disruptions in multiple reward systems. Through an fMRI, researchers Holm et al. (2009) found that during the anticipation of a reward, less activation in the caudate nucleus was correlated with decreased sleep duration, sleep onset time, and worse sleep quality. During reward outcome, less activation in the caudate was again correlated with later sleep onset time, earlier sleep offset time, and

lower sleep quality. Other literature has established relationships between sleep disturbance and decreased effort expenditure for reward (Engle-Friedman et al., 2010) alongside worse sleep quality and diminished reward responsiveness (Wieman et al., 2022). Lastly, a study by Casement et al. (2016) found a mediating effect of the neural activity preceding a reward in regard to nonrestorative sleep and adolescent depression. Altogether, these findings suggest that sleep disruption is tied to deficits in reward processing, and that reward motivation may play a significant role in understanding the relationship between sleep quality and depression.

Previous studies provide a substantial knowledge base of sleep, depression, and reward processing. This literature describes the interrelationships between depression, its comorbidities, and persistence across both gender and age groups, alongside evaluating worse sleep as a predictor of both depression and suicidality. Furthermore, it lays the foundation for how poor sleep affects mood, and how sleep disruption impacts reward anticipation, motivation, and effort expenditure. What has yet to be investigated is the potentially mediating factor of reward motivation between sleep quality and depression. In the current study, women were specifically sampled due to their heightened rates of depression prevalence and the need for targeted intervention. The present study aims to investigate reward motivation as a mediator of poor sleep quality and depressive symptoms within this population.

Chapter 2: Methods

Participants

The participants of this study were young women, ages 18-22 years old. These women suffered both insufficient sleep and moderate to high scores of depressive symptoms. Insufficient sleep was determined by a score of daytime ≥ 6 on the Epworth Sleepiness Scale (Johns, 1999) or a score of ≥ 16 on the PROMIS Sleep-Related Impairment Scale (Yu et al., 2012,) alongside regularly sleeping less than 8 hours per night. Depression was operationalized through the Center for Epidemiological Studies for Depression Scale (CESD; Radloff, 1977), and the inclusion criteria for this study was a score ≥ 15 , which is slightly above the average score in young adults (Radloff, 1991).

Exclusion criteria included the diagnosis of a sleep disorder (e.g. insomnia, sleep apnea, hypersomnia), psychotic symptoms, and substance use disorder. Study exclusion was also based on present suicidal ideation, use of psychiatric medications, or a history of a neurological disorder. These conditions aimed to exclude participants whose sleep extension would be ineffective or contraindicated as well as control for comorbidities in psychopathology that could present challenges to sleep. Exclusion criteria also worked to control for variance in response that could not be modeled with a small sample size.

Materials

Adolescent psychiatric disorders: The Structured Clinical Interview for DSM 5 Psychiatric Disorders (SCID; First, Williams, Karg, & Spitzer, 2015) was used to screen for psychotic symptoms, and diagnose mood, anxiety, substance use, and trauma-related disorders. These diagnostic interviews were provided by a postbaccalaureate research coordinator or a clinical psychology doctoral student. All interviewers were trained and supervised by Dr. Melynda Casement.

Adolescent depression: Depression was measured through the CESD (Radloff, 1977). The CESD is comprised of 20 self-reported Likert-style questions that measure mood and behavior over the past week. Some of these questions include "I was bothered by things that usually don't bother me," "I talked less than usual," or "I felt lonely." Participants then indicate how often they have felt that way on a 0-4 scale — 0 specifying "Rarely or none of the time (less than 1 day)" and 4 indicating "most or all of the time (5-7 days)." After completion of the scale, scores are added. These scores range from 0-60, and a score of 16 or higher is considered depressed. Epidemiological data specify a mean CESD score of 12.51 (*SD* = 9.41) in adolescents (Radloff, 1991). The CESD has high internal consistency (α = 0.79–0.87; Radloff, 1977; Radloff, 1991) and 2-week test-retest reliability (0.51; Radloff, 1977).

Subjective sleep characteristics: Past month's sleep quality was measured through the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is an 18-item questionnaire with seven component scores ranging from 0 to 3. These result in an overall sleep quality score (range 0–21). A score of five or greater indicates a high probability of sleep disorder (Buysse et al., 1989). The PSQI is internally valid ($\alpha = 0.80-0.85$), has high test-retest reliability (r = 0.85-0.87) and concurrent validity with other assessments (r > 0.69; Backhaus, Junghanns, Broocks, Riemann, & Hohagen, 2002; Buysse et al., 1989; Carpenter & Andrykowski, 1998).

Sleep disorders: Sleep disorders including but not limited to insomnia, hypersomnia, sleep-disordered breathing, circadian rhythm, and sleep movement disorders were measured with the Structured Clinical Interview for DSM 5 Sleep Disorders (SCIDSLD; Wong, Samuelsson,

Casement, & Buysse, 2016). The SCIDSLD is comprised of screening questions that include "do you have excessive daytime sleepiness?" and "do you feel excessively sleepy even when you have enough opportunity to sleep at night?" If a participant agrees with a statement, the interviewer proceeds to the next indicated module to ask more refining questions with regard to episode history, insomnia, hypersomnolence, narcolepsy, circadian rhythm sleep-wake disorders, non-rapid eye movement sleep arousal disorders, nightmare disorder, restless leg syndrome, and substance/medication-induced sleep disorder. These diagnostic interviews were provided by a post-baccalaureate research coordinator or a clinical psychology doctoral student. All interviewers were trained and supervised by Dr. Melynda Casement.

Reward motivation: Reward motivation was measured through The Effort Expenditure for Rewards Task (EEfRT; Treadway et al., 2009). The EEfRT is a multi-trial game in which participants choose between two tasks varying in difficulty to obtain monetary rewards. During each trial, participants repeat manual button presses within short successions of time. More difficult tasks are rewarded with higher compensation. For example, successful completion of the difficult task required 100 buttons pressed with the non-dominant pinky within 21 seconds, while completion of the easy task trials simply required the participants to make 30 button presses using the dominant pointer finger within 7 seconds. To aid participants in choosing which trials they were most likely to win, participants were presented with probability cues at the beginning of each trial, ranging from high (e.g. 88%) to low (e.g. 12%). After making a decision, participants' screens displayed a 1-second "ready" screen, then they completed the task. Afterward, participants were shown a 2-second feedback screen that displayed if the task was successfully or unsuccessfully completed. If the task was successfully completed, then a second feedback screen appeared for 2 seconds which displayed their monetary prize value (*See Figure* *2).* On average, easy-task trials took approximately 15 seconds, and hard-task trials took approximately 30 seconds.

The EEfRT model explored interactions between anhedonia and probability and reward magnitude. A pattern was found in the initial study by Treadway et al. (2009) that those with higher levels of anhedonia were far less likely to choose a hard task *(See Figure 3)*.

Chapter 3: Procedure

Written informed consent was gathered from all participants, and participants were debriefed following all study procedures. Participants received up to \$361 for their time and participation. Procedures were approved by the Institutional Review Board.

Participants were recruited through advertisements distributed on social media platforms and campus email listservs. Those who were interested registered on a web-based participant recruitment platform and completed a short pre-screening questionnaire. Of those who met gender and age inclusion criteria, participants then completed additional screening assessing sleep characteristics, and symptoms of depression. Those who continued to meet inclusion criteria were then invited to complete a telephone screening. During this assessment, the study coordinator provided select items from the SCIDSLD to exclude participants who likely had a sleep disorder. Those who remained eligible were then invited to complete an in-office screening for sleep and psychiatric disorders using the SCIDSLD and SCID, respectively.

During baseline sleep monitoring, bedtimes and wake times were created based on the participants' respective typical sleep duration plus an additional 30 minutes (obtained during screening). Participants were instructed to keep to a regular sleep schedule within the parameters that both accommodate their daytime obligations and remained similar to their typical sleep timing. During this time, participants received automated text reminders (30 minutes before bedtime and 30 minutes after wakeup) about the sleep monitoring protocol, and a link to complete the daily sleep diaries and questionnaires. Sleep diary entries and actigraphy data were routinely reviewed to monitor for adverse reactions. Participants who deviated significantly from their sleep schedule were removed from the study and were compensated for their participation.

After baseline observation, participants were randomly assigned to the typical sleep opportunity (TSO) or extended sleep opportunity (ESO) condition. At this time, participants received additional instructions for their sleep schedule. ESO participants were asked to increase their sleep opportunity by 90 minutes, while TSO participants were asked to continue with the same sleep schedule as the previous week. ESO condition participants could increase their sleep opportunity by either advancing their bedtime, delaying their wake time, or taking naps. These participants were also instructed to contact the study investigator if they experienced difficulty falling asleep or staying asleep during this extension phase.

To investigate the hypothesized mediating effect of reward motivation on depression symptomatology and sleep quality, I planned to utilize regression analysis. This analysis was composed of three regression sets: $X \rightarrow Y, X \rightarrow M$, and $X + M \rightarrow Y. X \rightarrow Y$: See *Figure 4*; $X \rightarrow$ *M*: See *Figure 5*; $X + M \rightarrow Y$: See *Figure 6*.

Chapter 4: Results

Figure 1 displays a flowchart depicting participant exclusion and withdrawal, and *Figures 7* and *8* provide descriptive characteristics for those who completed the 2-week sleep protocol (N = 22). The breakdown of demographics for those who began the sleep protocol is as follows. Of the 11 individuals randomly assigned to the typical sleep, control group, *M* age = 20.41, *SD* = 1.24. 8 (72.7%) of these individuals were White, 1 (9.1%) of them were Asian, and 2 (18.2%) of them were other. Of the other 11 individuals randomly assigned to the extended sleep, experimental group, *M* age = 19.58, *SD* = 1.02. Of these individuals, 7 (63.6%) of them were White, 1(9.1%) was Asian, 2 (18.2%) were other, and 1 (9.1%) were multiracial. Of those who withdrew from the study (N = 10), *M* age = 20.52, *SD* = 1.34. Of these 10, 5 (50%) were White, 2 (20%) were Asian, 1 (10%) was other, and 2 (20%) were multiracial. The average participant was 20 years old, enrolled in college (84%), had at least one parent with a college degree (75%), and was Caucasian (62.5%). Furthermore, 56% of this sample was diagnosed with a lifetime mood disorder diagnosis (Casement et al., 2021).

Of the 11 individuals randomly assigned to the typical sleep control group, CESD scores (M = 18.18, SD = 9.61) were higher than those who completed the sleep extension protocol (M = 13.55, SD = 7.98) following the sleep duration manipulation. PSQI scores also varied between groups with the control group reporting worse overall sleep quality (M = 7.18, SD = 2.18) than the experimental group (M = 6.36, SD = 1.50) after sleep manipulation.

Sleep quality and depression: A linear regression was used to test if worse sleep quality predicts higher depression scores. Results showed that there was not a significant relationship between sleep quality and depression scores ($R^2 = 0.0151$, F(1, 20) = 0.307, p = 0.585). The

effect size of this relationship was small such that depression (as measured by CESD on the 2nd visit) accounted for just 1.513% of the variability in sleep quality. These results indicate that as subjective sleep quality worsens, depression scores do not necessarily increase. This does not support the hypothesis that worse sleep quality would predict increased depressive symptomatology in adolescent girls. See *Figure 9* for a scatterplot depicting the relationship between sleep quality and depression scores.

Reward motivation and sleep quality: A second linear regression was used to test the relationship between sleep quality and reward motivation, operationalized by the mean proportion of hard tasks chosen throughout EEfRT trials. The results of this regression displayed a nonsignificant relationship between sleep quality and reward motivation ($R^2 = 0.0404$, F(1, 19) = 0.799, p = 0.383). Again, the effect size was minimal such that reward motivation accounted for 4.04% of the variability in sleep quality. Indicating a nonsignificant relationship between variables, these results do not support the hypothesis that worse sleep quality would predict a lower proportion of hard tasks chosen. See *Figure 10* for a scatterplot depicting the relationship between sleep quality and reward motivation.

Mediation analysis: This analysis aimed to explore the impact of sleep quality on depression as mediated by reward motivation. It was hypothesized that having worse sleep quality would positively predict depression. Furthermore, it was predicted that reward motivation would be a mediating variable. A series of regression analyses were used to test these hypotheses. The results demonstrate a nonsignificant mediation relationship such that B = -0.0460, z = -0.705, p = 0.481 (95% *CI*, -0.832 to 0.392). Alongside this, results showed that sleep quality had a nonsignificant relationship with reward motivation (B = 0.2009, z = 0.940, p = 0.347), and that reward motivation had a nonsignificant relationship with depression scores (B = 0.347).

-0.2290, z = -1.066, p = 0.286). Direct effects were also null; sleep quality had a nonsignificant relationship with depression scores (B = 0.1894, z = 0.882, p = 0.378).

Chapter 5: Discussion

Through regression analyses, the current study found no significant associations between sleep quality, depression, and reward motivation. Furthermore, reward motivation did not mediate the relationship between sleep quality and depression. These results did not support the hypothesis that these factors would be both correlated and also mediated by reward motivation.

As stated before, the relationship between sleep quality and reward motivation was nonsignificant. In analyzing the relationship between these variables, the line of best fit is not only inconsistent with the initial hypothesis, but it follows the opposite direction, such that as sleep quality worsens, subjects chose a higher proportion of hard tasks. This could potentially be explained by an increased drive for reward following worse sleep.

In 2013, researchers Telzer et al. (2013) sought to explain the relationship between poor sleep quality and reward-related brain function in adolescents. Their results demonstrated that subjects with poorer sleep also experienced increased reward motivation, also noting that this relationship is connected to decreased cognitive control and increased risk-taking behaviors. Moreover, Telzer et al. (2013) describe that poor sleep may exaggerate the already imbalanced relationship between affective and cognitive control systems. These findings could explain the mechanism by which increased risk-taking behaviors occur in adolescents with worse sleep. Increased reward motivation following poor sleep quality was also observed in a population of pregnant and postpartum mothers. Betts et al. (2021) found that depressive symptoms, stress, and worse sleep quality were all associated with increased hedonic eating, or the desire to eat foods in the absence of hunger cues.

The work of Betts et al. (2021) corresponds with existing literature that explores the role of poor sleep in relation to reward motivation. In a paper from Gujar et al. (2011), researchers

iterate that poor sleep and sleep deprivation contribute to reward reactivity and motivation. Researchers demonstrate that these effects can be witnessed through fMRI across activation differences. Critically, Gujar et al. (2011) find that sleep deprivation activates increased activity in the mesolimbic system, a key dopaminergic pathway that responds to pleasure and rewards. Researchers also describe how this activation enhances response to positive emotional stimuli (Gujar et al., 2011) and negative emotional stimuli (Yoo et al., 2007). Yoo et al. (2007) report that this activation in the amygdala is correlated with decreased activity in the medial prefrontal cortex.

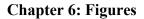
With increased activation in areas of the brain that process emotional stimuli and under activation of the prefrontal cortex and other cognitive control systems, the results of these articles offer a foundation for understanding the consequences and risks of reduced sleep and poor sleep quality. Consistent with these findings, the results of the current study demonstrate a slight trend whereby as adolescent women worsen in sleep quality, they are more likely to choose a harder task for an increased reward. Because poor sleep quality is pervasive in adolescents (Saxena, Koreti, & Gaur, 2016), its effects on reward motivation and consequences related to risky decision-making and decreased cognitive control must be addressed. In the same way, researchers Harrison and Horne (2000) mark poor sleep as not only a developmental concern but also a public health issue that impacts adolescent well-being. The physical, emotional, and social developments during adolescence are indispensable. Being that decreased sleep quality is associated with markers of decreased cognitive control and risky decisionmaking, it is important that adolescents increase sleep and overall sleep quality to promote their well-being.

Unlike sleep quality and reward, the relationship between sleep quality and depression follows the hypothesized direction of correlation, but not to a significant extent. This could be due to the limited age range of this study's sample, only including women ages 18-22. According to Villarroel & Terlizzi (2020), depression prevalence is highest among those aged 18-29. Furthermore, a study from Madrid-Valero (2017) found that increases in age are directly related to decreases in subjective sleep quality, especially among women. Thus, including ages 23-29 in this sample could impact mean CESD scores and the interrelationship between depression and sleep quality. Lastly, expanding this study's sample size could increase generalizability and provide context for future interventions within this group.

This study was not without its limitations. The primary limitation of this study was its small number of participants (N = 22). Having too small of a sample increases the margin of error, and reduces the statistical power of a study. Alongside having a smaller sample size, this study's age range was limited. Participants were all young women, aged 18-22. Because of its limited range, age may have modified results. Another limitation of the present study was the restricted manipulation of the PSQI. In Casement et al's (2020) study, sleep duration was the only component of the PSQI that was manipulated.

In an effort to mitigate the limitations of the present study, directions for future research could include an increased sample, participants from a wider age group, increased manipulation of PSQI components, and the search for methods to promote sleep health within this age group. In increasing sample size, future researchers could utilize greater statistical power and better controls of sample variance, however, having too large of a sample may inflate small differences within the study. Secondly, due to the increased prevalence rates of depression in younger adolescents, future studies may wish to include a wider breadth of age groups, including but not

limited to a wider range of adolescents or greater youth (10-24). Furthermore, although Casement et al. (2020) found that increasing sleep duration is associated with diminished depressive symptoms, it may be worthwhile to manipulate more components of sleep quality to better understand how depression varies with sleep disturbance, sleep latency, daytime dysfunction due to sleep iness, sleep efficiency, overall sleep quality, and sleep medication use. Knowing the prevalence and risk of depression and diminished sleep quality particularly among adolescents, directions for future research could also include finding methods, clinical or otherwise, to promote increased sleep health within this age group. Depression is one of the leading causes of global disease (Kessler et al., 2005), and its diagnosis is associated with the risk of suicide (Birmaher et al., 1996), anxiety, conduct disorders, substance abuse disorders, risky behaviors (Brooks et al., 2002), deficits in interpersonal relationship, and physical health (Beekman et al., 1997). With poor sleep as a marker for MDD (Geoffroy, et al., 2018), and ties between both of these variables and reward response (Treadway et al., 2012; Luking et al., 2016; Holm et al., 2009); Wieman et al., 2022), finding successful sleep and depression interventions for adolescents is paramount.



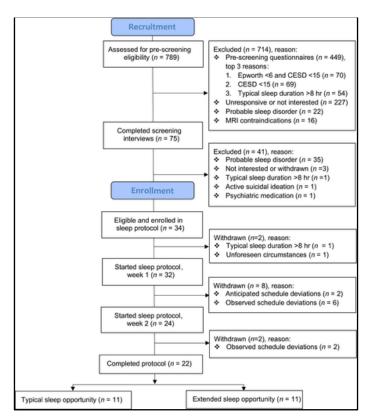
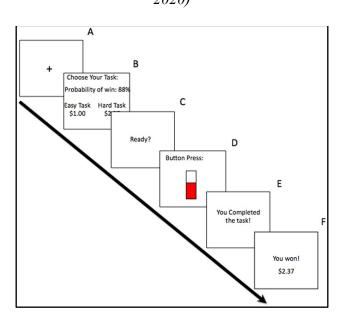


Figure 1: Participant recruitment flow chart (Casement, Livingston, Allen, & Forbes,



2020)

Figure 2: Overview of EEfRT procedure (Treadway et al., 2009)

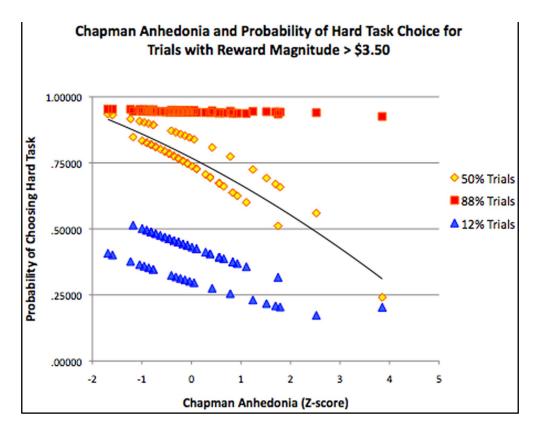


Figure 3: Anhedonia and hard task choice (Treadway et al., 2009)

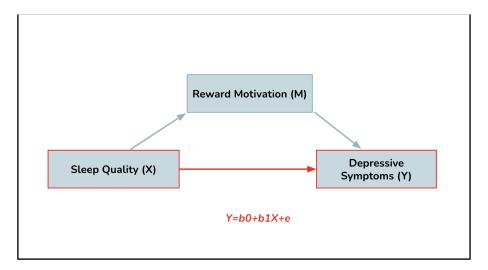


Figure 4: Display of sleep quality effect on depressive symptoms

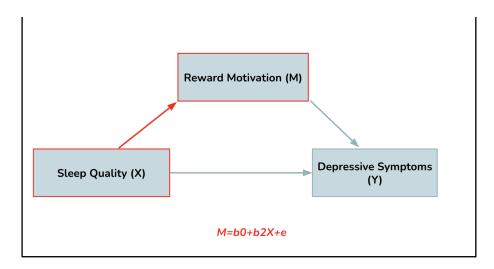


Figure 5: Display of sleep quality effect on reward motivation

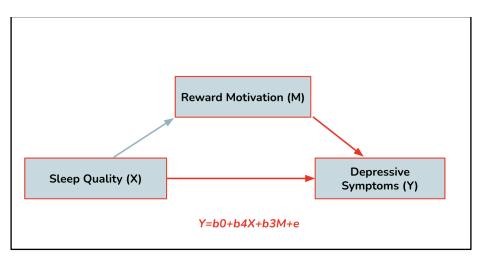


Figure 6: Display of reward motivation's effect on the relationship between sleep quality and

depressive symptoms

Descriptive Characteristics

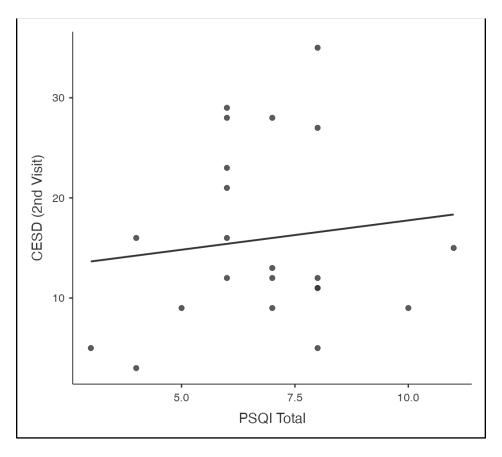
Of participants who completed the sleep protocol (N = 22)

| | Group | Ν | Mean | Median | SD | Range | Minimum | Maximum |
|------------------|---------------------------|----|-------|--------|------|-------|---------|---------|
| Age | Typical sleep opportunity | 11 | 20.41 | 20.44 | 1.24 | 4.26 | 18.64 | 22.90 |
| | Sleep extension | 11 | 19.58 | 19.39 | 1.02 | 3.15 | 18.24 | 21.39 |
| CESD (2nd Visit) | Typical sleep opportunity | 11 | 18.18 | 15.00 | 9.61 | 30.00 | 5.00 | 35.00 |
| | Sleep extension | 11 | 13.55 | 12.00 | 7.98 | 25.00 | 3.00 | 28.00 |
| PSQI Total | Typical sleep opportunity | 11 | 7.18 | 7.00 | 2.18 | 8.00 | 3.00 | 11.00 |
| | Sleep extension | 11 | 6.36 | 7.00 | 1.50 | 4.00 | 4.00 | 8.00 |

Figure 7: Descriptive characteristics of age, CESD, and PSQI scores

| requencies of R | ace | | | | Frequencies of Sexual 0 | Drientation | | | | |
|---|---|---|-------------------------------|------------------|---|-------------|--------------------------------|----------------------|---------|-----------|
| | | Group | | _ | | | Group | | | |
| Race | Typical sleep oppo | ortunity Slee | p extension | | Sexual Orientation | Typical sle | ep opportunity | Sleep exte | nsion | |
| Asian | 1 | | 1 | | Heterosexual | | 6 | 6 | | |
| White | 8 | | 7 | | Mostly Heterosexual | | 2 | 3 | | |
| Other | 2 | | 2 | | Bisexual | | 2 | 2 | | |
| Multiple Races | 0 | | 1 | | Gay | | 1 | 0 | | |
| requencies of Er | mployment Status | | | | Frequencies of Subject | Education | | | | |
| | | Group | | | | Luuuun | | Group | | |
| Employment S | tatus Typical sleep | opportunity | Sleep exte | nsion | Subject Educa | tion | Typical sleep o | opportunity | Sleep e | extension |
| | | | | | | | | | | |
| Student | | 8 | 9 | | Completed high school | l or GED | 1 | 2 | | 2 |
| Student | | 8 3 | 9 1 | | Completed high school Completed some colle | | | 2 8 | | 2 9 |
| Student Employed Unemployed | | | | | Completed some colle College degree | ge courses | 1 | | | |
| Student Employed Unemployed | arents' Education | 3 | 1 | | Completed some colle | ge courses | 1 | 8 | | 9 |
| Student Employed Unemployed | _ | 3 | 1 1 Group | Sieep extension | Completed some colle College degree | ge courses | | 8 | tension | 9 |
| Student Employed Unemployed 'requencies of Pr Parents' No formal educa | Education 1 | 3 0 | 1 Group pportunity | 0 | Completed some colle College degree Frequencies of Ethnicity Ethnicity Hispanic or Latina | ge courses | Group leep opportunity 2 | 8 1 y Sleep ex | 3 | 9 |
| Student Employed Unemployed Frequencies of Pri Parents' No formal educz Completed high | Education 1 ation school or GED | 3 0 Typical sleep o | 1 Group pportunity | 0 | Completed some colle College degree Frequencies of Ethnicity Ethnicity | ge courses | Group | 8 1 y Sleep ex | | 9 |
| Student Employed Unemployed Frequencies of Pi Parents' No formal educ: Completed high Technical schoo | Education 1 ation school or GED | 3 0 Typical sleep o 1 1 1 | 1 1 Group pportunity | 0 1 1 | Completed some colle College degree Frequencies of Ethnicity Ethnicity Hispanic or Latina | ge courses | Group leep opportunity 2 | 8 1 y Sleep ex | 3 | 9 |
| Student Employed Unemployed Prequencies of Pri Parents' No formal educa Completed high Technical schoo College degree | Education 1 ation school or GED I degree | 3 0 Typical sleep o 1 1 1 | 1 1 Group pportunity | 0 1 1 6 | Completed some colle College degree Frequencies of Ethnicity Ethnicity Hispanic or Latina | ge courses | Group leep opportunity 2 | 8 1 y Sleep ex | 3 | 9 |
| Student Employed Unemployed Prequencies of Pri Parents' No formal educa Completed high Technical schoo College degree | Education 1 ation school or GED | 3 0 Typical sleep o 1 1 1 | 1 1 Group pportunity | 0 1 1 | Completed some colle College degree Frequencies of Ethnicity Ethnicity Hispanic or Latina | ge courses | Group leep opportunity 2 | 8 1 y Sleep ex | 3 | 9 |
| Student Employed Unemployed Prequencies of Pr Parents' No formal educa Completed high Technical schoo College degree Masters or doct | Education 1 ation school or GED I degree orate level degree | 3 0 Typical sleep o 1 1 1 4 4 4 | 1 1 Group pportunity | 0 1 1 6 | Completed some colle College degree Frequencies of Ethnicity Ethnicity Hispanic or Latina | ge courses | Group leep opportunity 2 | 8 1 y Sleep ex | 3 | 9 |
| Student Employed Unemployed Frequencies of Pr Parents' No formal educa Completed high Technical schoo College degree Masters or doct | Education 1 ation school or GED I degree orate level degree ender Groug | 3 0 Typical sleep o 1 1 1 4 4 | 1 Group pportunity | 0 1 1 6 | Completed some colle College degree Frequencies of Ethnicity Ethnicity Hispanic or Latina | ge courses | Group leep opportunity 2 | 8 1 y Sleep ex | 3 | 9 |
| Student Employed Unemployed Frequencies of Pr Parents' No formal educa Completed high Technical schoo College degree Masters or doct | Education 1 ation school or GED I degree orate level degree | 3 0 Typical sleep o 1 1 1 4 4 | 1 Group pportunity | 0 1 1 6 | Completed some colle College degree Frequencies of Ethnicity Ethnicity Hispanic or Latina | ge courses | Group leep opportunity 2 | 8 1 y Sleep ex | 3 | 9 |

Figure 8: Descriptive characteristics of race, employment status, education, gender, sexual



orientation, and ethnicity

Figure 9: Scatterplot depicting the relationship between sleep quality and depression scores in

adolescent girls

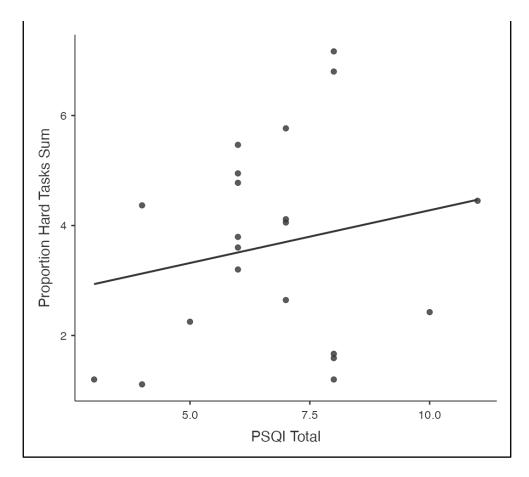


Figure 10: Scatterplot depicting the relationship between sleep quality and reward motivation in

adolescent girls

Chapter 7: Bibliography

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