Subjective and Objective Sleep Measures and Symptomatology

in Internalizing Psychopathologies

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LIST OF ABBREVIATIONS

CBT	Cognitive Behavioral Therapy
ERQ	Emotion Regulation Questionnaire
ERQ-R	Emotion Regulation Questionnaire-Reappraisal Subscale
ERQ-S	Emotion Regulation Questionnaire-Suppression Subscale
GS	Good Sleep
GSQ	Good Sleep Quality
HAM-A	Hamilton Anxiety Rating Scale
HAM-D	Hamilton Depression Rating Scale
HC	Healthy Controls
IP	Internalizing Psychopathology
MANOVA	Multivariate Analysis of Variance
PS	Poor Sleep
PSQ	Poor Sleep Quality
PSQI	Pittsburgh Sleep Quality Index
PT	Patients
SCID-5	Structured Clinical Interview for DSM-5 Disorders
TST	Total Sleep Time
VIF	Variance Inflation Factor
WASO	Wake After Sleep Onset

SUMMARY

Internalizing psychopathologies (IPs) such as anxiety and depression are commonly associated with disturbances in sleep (e.g., problems falling asleep, staying asleep) and emotion dysregulation. Accumulating data indicate problematic sleep is transdiagnostic and may exacerbate symptoms and emotion dysregulation in IPs. However, the majority of studies have relied on self-report measures of sleep, which are often discrepant from actigraphy, a validated objective measure of sleep. This study involved un-medicated patients with a principal anxiety disorder (n = 35) or unipolar depressive disorder (n = 12) and healthy controls (n = 20). Chi-square and multivariate analysis of variance were used to examine hypothesized groups effects and multiple regression analysis was performed to investigate associations between naturalistic sleep assessed with self-report and actigraphy, symptom severity, and habitual emotion regulation tendencies (i.e., reappraisal, suppression). We hypothesized patients would experience worse sleep, which is associated with higher symptom severity and emotion dysregulation. Study results showed that hypotheses were supported for self-reported sleep but not for actigraphy-based sleep. Specifically, poor subjective sleep, endorsed by more patients, is related to higher anxiety, higher depression, and lower reappraisal. Findings did not show sleep, either subjective or objective, to be related to suppression.

I. INTRODUCTION

Problematic sleep is observed in both the general population (Kahn et al., 1989; Owens & Matthews, 1998) and individuals experiencing internalizing disorders, such as anxiety and depression (Alvaro et al., 2013; Taylor et al., 2005). Findings from previous studies suggest poor sleep is transdiagnostic, dimensional, and contributes to worsening of mood and deficiencies in emotion regulation. However, there has been minimal integration of actigraphy, an objective estimate of sleep, and studies have been largely disorder-specific. Therefore, this investigation aimed to examine both subjectively and objectively measured sleep and how it relates to anxiety and depression symptoms and emotion regulation (i.e., reappraisal, suppression), in patients with internalizing disorders and healthy control participants.

A. Literature Review

1. Problematic Sleep in General Population

Sleep problems are common in the general population, with complaints such as difficulties falling asleep or maintaining sleep (Bixler et al., 1979; Mellinger et al., 1985; Welstein et al., 1983). Many individuals also report not feeling rested or getting the amount of sleep they desire or need (Bliwise et al., 1992). These complaints are often persistent and recurrent, suggestive of the symptoms of insomnia. In an epidemiologic study of three large community samples, more than half of the participants reported such complaints as occurring most of the time and between 23% and 34% reported symptoms of insomnia (Foley et al., 1995). Furthermore, more than one-third of the U.S. population reported sleeping less than the recommended 7 hours in a 24-hour period (Y. Liu et al., 2016). This is problematic as sleep is a

combination of complex physiological and behavioral processes well-intertwined with the ability to carry out everyday functions, therefore, altering its course contributes to clinical conditions and consequences (Bonnet & Arand, 2003; Fortier-Brochu et al., 2012). Accordingly, sleep disturbances have been found to be predictive of mortality rates. For instance, in a cohort of working men and women, self-reported short sleep duration was associated with an increased risk of mortality (Heslop et al., 2002). Additionally, healthy older adults with low sleep efficiency, as assessed with polysomnography, were at a greater risk of death, beyond age and medical burden (Dew et al., 2003). With the high prevalence of poor sleep, it is expected that many factors may initiate and maintain sleep problems. For example, female gender and advancing age have been found to be risk factors of problematic sleep (Klink et al., 1992; Zhang & Wing, 2006). Physical health problems (e.g., cardiovascular disease) and substance abuse have also been consistently shown to be significant issues contributing to poor sleep quality (Johnson & Breslau, 2001; Roehrs & Roth, 2001; Sofi et al., 2014).

Sleep disturbances have also been shown to negatively impact psychiatric health (D. E. Ford & Kamerow, 1989; Sarchiapone et al., 2014), and problematic sleep is a prevalent symptom of many psychiatric disorders (Benca et al., 1992). Prior work has detailed the relevance of sleep effects on emotion, with a strong association between problematic sleep and emotional problems (e.g., excessive worrying, irritability, anger) (Goldstein & Walker, 2014; Paavonen et al., 2002). Moreover, sleep deprivation studies in healthy individuals highlight the relationship between sleep and negative mood. For example, in healthy adults, one night of acute sleep deprivation was associated with greater anxiety, depression, and general distress relative to a control (i.e., no sleep deprivation) condition (Babson et al., 2010). Similarly, in a sleep manipulation study of healthy adolescents, shortened sleep was associated with negative mood and the ability to

regulate negative emotions (Baum et al., 2014). Neuroimaging work indicates the deleterious impact of sleep loss on mood is due to shared neurobiology between sleep and emotion regulating systems (Gruber & Cassoff, 2014; Yoo et al., 2007).

2. Problematic Sleep in Internalizing Psychopathologies

Internalizing psychopathologies (IPs) represent a spectrum of conditions characterized by negative emotion (Krueger & Markon, 2006) that are frequently disabling, cause severe impairment, and carry chronic burden (Kessler et al., 1999). In particular, anxiety and depression are frequently found to be highly comorbid disorders (Kaufman & Charney, 2000; Sartorius et al., 1996). A review of previous literature demonstrated that more than 50% of patients diagnosed with an anxiety or depressive disorder also suffer from a comorbid depressive or anxiety disorder, respectively. Moreover, the presence of this comorbidity is associated with increased chronicity, slower recovery, higher recurrence rate, and negative psychosocial impact (Hirschfeld, 2001). Thus, not only are anxiety and depressive disorders commonly cooccurring psychiatric conditions, they share overlapping characteristics marked by excessive or incongruous negative affect and biases (Lonigan et al., 1994; Seligman & Ollendick, 1998).

Individuals with anxiety and/or depression also frequently experience problematic sleep (Shanahan et al., 2014). For example, sleep disturbances are observed across different anxiety disorders such as panic disorder, generalized anxiety disorder, and posttraumatic stress disorder (Mellman, 2006). There are also close ties between depression and sleep difficulties, which may be bi-directional as individuals complaining of sleep difficulties are 3 to 4 times more likely to be depressed (Almeida & Pfaff, 2005). In a community sample of adolescence followed through their transition into adulthood, high comorbidity and longitudinal associations have been established between sleep disturbances and psychiatric symptoms as well (Fichter et al., 2009).

Altogether, problematic sleep is ubiquitous in IPs and considered to be transdiagnostic (Gorman, 1996).

Specific characteristics regarding the quantity and quality of sleep vary from person to person (Hale, 2005). Sleep disturbance may encompass insomnia (i.e., problems falling and staying asleep), hypersomnia (i.e., excessive sleepiness), or both insomnia plus hypersomnia. All forms of disturbed sleep are risk factors for the development of anxiety and depression (D. E. Ford & Cooper-Patrick, 2001; Gillin, 1998). Research has also shown that those presenting with combined insomnia and hypersomnia were more severely depressed and likely to have comorbid anxiety disorders (X. Liu et al., 2007; Soehner et al., 2014). Moreover, insomnia and hypersomnia, separately, are significant predictors of both anxiety and depression (Garvey et al., 1984; Manber & Chambers, 2009; Ohayon & Roth, 2003). Even so, the majority of individuals with IPs have sleep complaints consistent with insomnia. For example, around 90% of patients report difficulty falling asleep, staying asleep, or early morning awakenings (Tsuno et al., 2005).

Not only are anxiety and depression often associated with poor sleep, poor sleep is found to worsen symptom severity as well (Cox & Olatunji, 2016). For instance, it has been shown in a large cohort of community-dwelling women either at risk for, or with anxiety disorders, that poor sleep efficiency, measured by actigraphy, is related to elevated anxiety symptoms (Spira et al., 2009). In particular, poorer sleep quality was associated with higher social anxiety symptom and severity scores in individuals with generalized social anxiety disorder (Zalta et al., 2013). Self-reported sleep-related problems were also positively correlated with anxiety severity in children and adolescents with generalized anxiety disorder (Alfano et al., 2007). Sleep disturbances have been found to be associated with increased symptom severity in obsessive-compulsive disorder (Storch et al., 2008) and posttraumatic stress disorder (Casement et al., 2012) as well. In children

suffering from concurrent disturbed sleep and a current major depressive episode, findings showed that they experienced more severe depressive symptoms (X. Liu et al., 2007). Poor sleep quality is also associated with symptom severity of postpartum maternal depression (Park et al., 2013). Taken together, disrupted sleep may contribute to the development and exacerbation of anxiety and depressive symptoms. However, the majority of findings are based on self-report. The incorporation of objective sleep measures when examining anxiety and depression symptomatology continues to be an area that is generally under-researched.

3. Emotion Regulation and Internalizing Psychopathologies

Emotion and cognitive processing are negatively impacted in anxiety (Cisler & Olatunji, 2012; Reinholdt-Dunne et al., 2013) and depression (Gotlib & Joormann, 2010; Joormann & Gotlib, 2010). The ability to modulate emotional states is thus emphasized in IPs (Aldao et al., 2016). Previous research, independent of sleep, has commonly shown that internalizing problems are associated with deficits centered around emotion dysregulation, particularly in reaction to negative stimuli (Hatzenbuehler et al., 2008; Zeman et al., 2002). In the execution of emotion regulation, difficulties in mitigating high negative emotionality is a factor specifically tied to internalizing symptomatology (Eisenberg et al., 2001). In this manner, the diminished ability to effectively process affective information contributes to symptoms of excessive negative mood and biases observed in IPs.

Emotion regulation approaches can be organized into those that are antecedent-focused (i.e., occurring prior to an emotional response to a stimulus to lessen the experienced arousal) and response-focused approaches (i.e., involving the alteration of emotions following the emotional experience), each with specific underlying strategies (Gross, 2002). Among these strategies, habitual reappraisal and suppression are deemed to be representative of each

approach, commonly used in daily life, and subject to individual differences (Gross et al., 2007). In a series of studies examining self-reported emotion regulation, it was found that the use of reappraisal led to experiences of more positive and less negative emotions, while the use of suppression led to experiences of less positive and more negative emotions (Gross & John, 2003). Its relevance in IPs include evidence of more frequent use of suppression in individuals with social anxiety disorder (Werner et al., 2011) or generalized anxiety disorder (Aldao et al., 2010). In depression, there is evidence of a positive correlation between suppression and depressive symptoms and a negative correlation between reappraisal and depressive symptoms in both clinical and nonclinical samples (Aldao et al., 2010). In summary, those suffering from internalizing problems are found to have a more habitual use of the suppression instead of the more adaptive reappraisal regulatory strategy.

Forms of emotion regulation have also been found to coincide with sleep problems and negative mood. In a veteran sample of individuals who suffered from posttraumatic stress disorder, there was a significant relationship between self-reported poor sleep quality and maladaptive emotion regulation strategies, particularly expressive suppression, in predicting anxiety (Mantua et al., 2018). Additionally, sleep deprivation studies highlight links between sleep loss and emotion (dys)regulation. In a laboratory cognitive reappraisal task, healthy participants who reported worse sleep exhibited less ability to implement reappraisal (Mauss et al., 2013). Yet, despite evidence of relationships between sleep and emotion regulation, more research is needed to address the impact on the severity of internalizing symptoms.

4. <u>Common Sleep Measures</u>

Wrist actigraphy is an objective measure of sleep. Actigraphy has been shown to have high agreement with ‘gold standard’ polysomnography, with the capability to monitor and record

continuous periods of activity and inactivity to estimate sleep quality (Girschik et al., 2013; McCall & McCall, 2012; Van Ravesteyn et al., 2014). Moreover, actigraphy is endorsed by the American Academy of Sleep Medicine for assessing sleep patterns in community settings (Morgenthaler et al., 2007). It is typically worn on the wrist like a watch and measures sleep by using an accelerometer to assess movement during the day which is contrasted with activity during rest (i.e., sleep). Common validated measures of actigraphy include total sleep time (TST), sleep efficiency, and wake after sleep onset (WASO). Greater TST and sleep efficiency are indices of better sleep, whereas greater WASO signifies more sleep loss. See Methods for how variables are defined (Lichstein et al., 2006; Meltzer et al., 2012).

The Pittsburgh Sleep Quality Index (PSQI) is a subjective measure of sleep assessed over the period of a month with inquiries about quantitative and qualitative aspects of habitual sleep. The PSQI global score is derived from responses to 19 questions that are condensed into seven different components of sleep, which are then summed to yield the global measure of sleep quality. It is the most commonly used measure of sleep quality in clinical and research settings, as it has been validated on individuals with good and poor sleep habits (Buysse et al., 1989). It is also a diagnostically sensitive (89.6%) and specific (86.5%) measure in distinguishing between sleep quality, with a global score greater than 5 denoting clinically problematic sleep (Backhaus et al., 2002).

Although they are both standard measures, there are more often than not, discrepancies between the wrist actigraphy and PSQI. For example, self-reported sleep time and actigraphy-estimated total sleep time were shown to be weakly correlated in both treatment-seeking overweight individuals (O'Brien et al., 2016) and pregnant women with a psychiatric illness (e.g., major depressive, anxiety, or personality disorder) (Van Ravesteyn et al., 2014). Also,

across patients diagnosed with anxiety or depression, the PSQI global score was not found to be associated with actigraphy estimates of sleep efficiency, duration, or WASO (Klumpp et al., 2017). Furthermore, inconsistencies were revealed between the sleep measures and symptom severity as well, with the PSQI positively correlated with both anxious and depressive symptoms, while sleep efficiency negatively correlated and WASO positively correlated with depressive symptoms only (Klumpp et al., 2017). In a nonclinical sample of younger and older adults, the PSQI global score did not correlate with any of the actigraphy variables of total sleep time, wake after sleep onset, or sleep efficiency (Grandner et al., 2006). However, in a sample of healthy children, parental reports of sleep managed to correlate well with the actigraphy recordings (Iwasaki et al., 2010).

Notably, evidence of the frequent discrepancy between subjective and objective measures indicates that both measures are important when evaluating sleep effects. It is likely that these measures capture different aspects of sleep that both will contribute to the understanding of IPs. Additionally, sleep is a gradual rather than discrete process, which may also contribute to the discrepancy between subjective and objective measures (Tryon, 2004).

B. Current Study

Despite the prevalence of problematic sleep, particularly in IPs, and its role in negative mood and emotion (dys)regulation, previous work is largely based on self-perceived sleep in disorder-specific samples. Given evidence that problematic sleep is transdiagnostic and disagreement between self-perceived sleep and actigraphy is common, the purpose of the present study was to examine differences between ‘good’ and ‘poor’ sleep groups and relationships between sleep measures, symptoms, and emotion regulation in treatment-seeking individuals with anxiety and/or depression and healthy control participants. See Methods for assignment into

sleep groups. Emotion regulation, specifically, reappraisal and suppression, were examined with the Emotion Regulation Questionnaire (ERQ; Gross, 2002).

1. Aims and Hypotheses

Aim 1: Examine distribution of patients and healthy controls, based on the 'good' and 'poor' sleep groups.

Hypotheses:

1. For subjective sleep/PSQI, more patients than controls will be in the poor than good sleep quality group.
2. For objective sleep/actigraphy, more patients than controls will be in the poor than good actigraphy-defined sleep group.

Aim 2: Examine group differences, based on the 'good' and 'poor' sleep groups, in symptom severity and self-reported emotion regulation.

Hypotheses:

1. Both poor sleep groups (measured subjectively and objectively) will endorse greater symptom severity, with higher scores on both the HAM-A and HAM-D, when compared with the good sleep groups.
2. Both poor sleep groups (measured subjectively and objectively) will endorse worse emotion regulation, with lower scores on the ERQ-Reappraisal subscale (ERQ-R) and higher scores on ERQ-Suppression subscale (ERQ-S), when compared with the good sleep groups.

Aim 3: Evaluate correspondences of sleep (measured subjectively and objectively), symptom severity, and emotion regulation across sleep groups.

Hypotheses:

1. PSQI global scores will be positively predicted by anxiety (i.e., HAM-A total score), depression (i.e., HAM-D total score), and suppression (i.e., ERQ-S subscale score). PSQI global scores will be negatively predicted by reappraisal (i.e., ERQ-R subscale score).
2. For actigraphy variables, TST and sleep efficiency will be negatively predicted but WASO will be positively predicted by HAM-A, HAM-D, and ERQ-S scores. TST and sleep efficiency will be positively predicted but WASO will be negatively predicted by ERQ-R scores.

II. METHODS

The present study used cross-sectional data collected from clinical trials; all data were collected prior to the participants beginning treatment. Groups consisted of un-medicated treatment-seeking patients and healthy control participants.

A. Participants

The current study consisted of 67 participants in total. Regarding principal diagnosis, 47 were diagnosed with a principal anxiety disorder [social anxiety disorder (n = 17); generalized anxiety disorder (n = 15); panic disorder (n = 2); posttraumatic stress disorder (n = 1)] or principal major depressive disorder (n = 12). Additionally, 20 demographically-matched healthy controls completed all of the same measures as patients. See Table I for details.

TABLE I

PRINCIPAL DIAGNOSIS AND COMORBIDITY

Principal Diagnosis	**N**	**%**
Social anxiety disorder	17	36.17
Generalized anxiety disorder	15	31.91
Major depressive disorder	12	25.53
Panic disorder	2	4.26
Post-traumatic stress disorder	1	2.13

Comorbidity	**N**	**%**
Social anxiety disorder	15	31.91
Major depressive disorder	13	27.66
Generalized anxiety disorder	11	23.40
Panic disorder	10	21.28
Persistent depressive disorder	9	19.15
Specific Phobia	6	12.77
Post-traumatic stress disorder	5	10.64
Obsessive compulsive disorder	2	4.26
Substance abuse	1	2.13

Participants were drawn from two treatment-outcome studies. One consisted of an open trial of cognitive behavioral therapy (CBT) involving patients with major depression or social anxiety disorder. In the other study, patients with a common anxiety or unipolar depressive disorder were randomized to CBT or pharmacotherapy. Treatment-seeking patients were recruited from the community (i.e., self-referred) and from professional referrals from the Mood and Anxiety Disorders Program at the University of Illinois at Chicago (UIC). Additionally, participants were recruited through web, newspaper, radio, and flyer advertisements. Potential participants were first screened over the phone to determine if they met initial inclusion criteria. Inclusion criteria for the patient groups were as follows: ages 18 to 65 years old, a principal diagnosis of depression and/or anxiety disorders, the ability to give informed consent, and no major active medical or neurological illness. Comorbidity was permitted, provided that depression or anxiety was the principal diagnosis. The inclusion criteria for healthy control participants was the same as that of patient groups, except that they were required to be free of a lifetime diagnosis of any Axis I disorder.

Exclusion criteria for all participants included: lifetime history of severe psychiatric illness (e.g., bipolar disorder, schizophrenia); cognitive impairment (e.g., traumatic brain injury, dementia); developmental disorder (e.g., pervasive developmental disorder); current[1] alcohol or drug abuse or dependence; current suicidal plan and/or intent; and positive drug screen test results.

[1] Current was defined as occurring within the past six months.

B. Instruments

All participants provided consent approved by the local Institutional Review Board at the University of Illinois at Chicago (UIC). All participants met with a master's-level clinician or PhD/MD assessor who administered the Structured Clinical Interview for DSM-5 Disorders (SCID-5; First et al., 2015) and symptom severity to determine the participants' diagnosis. Self-report was used to assess emotion regulation. Sleep measures, including the self-report Pittsburgh Sleep Quality Index (PSQI) and wrist actigraphy, were collected within a week of this initial study entry visit.

1. Clinician-Administered Measures

In addition to the SCID-5, participants completed the Hamilton Anxiety Rating Scale (HAM-A; Hamilton, 1959) and the Hamilton Depression Rating Scale (HAM-D; Hamilton, 1960) for the assessment of anxiety and depressive symptoms, respectively. Since the HAM-A and HAM-D contain sleep-related items, these items were removed to minimize confounding when analyzed with the sleep measures.

2. Emotion Regulation Questionnaire (ERQ)

The Emotion Regulation Questionnaire is a 10-item self-report measure assessing the habitual use of cognitive reappraisal and expressive suppression (ERQ; Gross & John, 2003). The reappraisal (ERQ-R) and suppression (ERQ-S) subscales are theorized as trait variables, reflective of either reframing experiences to regulate emotional salience or engage in expressive suppression, respectively. They have exhibited good internal consistency and test-retest reliability. They have also demonstrated good convergent and discriminant validity (Gross & John, 2003). Scores are specified using a 7-point Likert-type scale.

3. Subjective and Objective Sleep Measures

a. Pittsburgh Sleep Quality Index (PSQI)

The Pittsburgh Sleep Quality Index (PSQI) is a 19-item self-report questionnaire. This measure assesses sleep quality in adults over the previous month (Buysse et al., 1989). The total global score comprises of 7 component scores, which consist of: 1) subjective sleep quality, 2) sleep latency, 3) sleep duration, 4) habitual sleep efficiency, 5) sleep disturbances, 6) use of sleep medication, and 7) daytime dysfunction. Global scores range from 0 to 21. Higher scores signify worse sleep, with a global score greater than 5 denoting clinically problematic sleep (Buysse et al., 1989).

Assignment of sleep quality groups based on PSQI. Participants who had a PSQI global score greater than 5 were assigned to the poor sleep quality group and those with a PSQI global score less than or equal to 5, the good sleep quality group.

b. Actigraphy

All participants were instructed to: 1) wear an actigraph device, the Actiwatch 2 (Minimitter, Philips Respironics, Andover MA), on their nondominant wrist for 7 days/7 nights; 2) press the event marker button on their Actiwatch 2 upon getting into or out of bed; and 3) complete a simultaneous sleep diary to record habitual sleep times and disturbances. The Actiwatch was programmed with a start time and data collection interval. As the device is an omnidirectional accelerometer, it uses a piezoelectric sensor to monitor the occurrence and degree of motion. The motion sensor integrates the degree and speed of motion, storing this information as activity counts. The light sensor records the duration of light exposure and calculates the white light illuminance. Both activity count and light intensity were collected in 15-s epochs. The Actiwatch 2 has a memory capacity of 2 weeks and is water resistant.

Data was downloaded onto Windows XP and retrieved for analysis with the Actiware 6.0.9 scoring software, provided with the device. Actiware's algorithm was used to score each epoch as sleep or wake, following the default settings of a wake threshold activity count of 40. Then, all actigraphy data were manually evaluated in order to assign rest and active intervals through a hierarchical approach (Patel et al., 2015). Rest intervals, or periods of when the participant was trying to sleep, were set for analysis based on 4 inputs: event marker, sleep diary, activity count, and light intensity, in order of importance. For each rest interval, the most likely time in bed was identified for each input in isolation. For activity and light, time in bed was distinguished by a sudden drop in signal intensity, with activity levels needing to drop to zero for more than 10 epochs and light levels needing to fall below 1 lux for more than 10 epochs. Concordance within a 15-min interval was examined across inputs to determine the highest ranked input and defined time in bed. The same process was applied for time out of bed. One main rest interval at night was identified to determine the period of sleep. Once rest intervals were set, Actiware's algorithm then used 10 immobile or mobile minutes to define sleep onset and offset, respectively, to generate the sleep intervals. Actiware algorithms have been well-validated against polysomnography (Kushida et al., 2001; Marino et al., 2013).

Actigraphy variables of interest were: total sleep time (TST; average number of minutes spent sleeping), sleep efficiency (average percentage of time asleep), and wake after sleep onset (WASO; average number of minutes awake after initial onset of sleep). Sleep latency (average number of minutes between going to bed and falling asleep) was also derived but not used in analysis due to its reduced reliability (Martin & Hakim, 2011). Sleep variables were scored within the main rest interval for each 24-hr period, and means were computed.

Assignment of sleep groups based on actigraphy. TST was used to assign groups. Specifically, the National Sleep Foundation recommends 7 to 9 hours of sleep for adults 18 to 64 years of age. Therefore, participants with less than 7 hours of sleep per night were assigned to the poor sleep group and those with sleep duration of 7 to 9 hours, the good sleep group (Watson et al., 2015).

C. Analytic Approach

Sleep measures were examined to determine if assumptions of normality were met and if data transformation was necessary. Assumptions of homogeneity of variance and collinearity were tested for, with the variance inflation factor (VIF) required to be < 4 (Allison, 1999). The appropriate adjustments were made for any violations. Questions regarding sleep in the HAM-A/D were excluded to minimize the potential of confounding derived relationships. All analyses were two-tailed with an alpha level of 0.05. Statistical analyses were performed in R (3.5.2).

Significant main effects and interactions were followed up with simple effects analyses. Significant regression results were followed up with Pearson's correlations to clarify the direction and magnitude of associations. Furthermore, significant effects encompassing the PSQI global scores were followed up with analyses of its component sub-scores in order to better pinpoint the influence of specific aspects of overall sleep quality.

Chi-square analyses were performed to evaluate whether the PSQI-derived and actigraphy-derived sleep groups were similar in principal diagnosis as well as general demographics (i.e., gender, race, ethnicity) of participants. Likewise, independent samples t-tests were used to examine whether the PSQI-derived and actigraphy-derived sleep groups were similar in age and education level. Emerging differences were adjusted for by including relevant demographic characteristics as covariates in subsequent analyses.

1. **PSQI Manipulation Check**

For an examination of whether grouping based on the PSQI cutoff point had the intended effect, an independent samples t-test was conducted to confirm the PSQI global score and component sub-scores differed between the subjective 'good' and 'poor' sleep groups. Specifically, it was expected that mean scores would be higher in the PSQI-defined poor sleep quality (PSQ) than the good sleep quality (GSQ) group.

2. **Aims and Hypotheses Based on PSQI (Aims 1 and 2)**

To test the hypotheses that more patients than controls would be assigned to the PSQ group, chi-square analyses were performed.

To test for expected differences in symptomatology between sleep quality groups, a 2 (PSQI group: GSQ, PSQ) x 4 (measure: HAM-A, HAM-D, ERQ-R, ERQ-S) multivariate analysis of variance (MANOVA) was performed. Although moderate associations between anxiety and depression symptoms and reappraisal and suppression components were expected, they nonetheless reflect distinct dimensions of symptomatology. We followed up significant omnibus findings with independent t-tests, controlling for multiple comparisons by conducting Bonferroni corrections. Since units for independent variables (IVs) differed, IVs were standardized (i.e., z-scored).

3. **Actigraphy Manipulation Check**

In order to evaluate whether grouping based on the actigraphy threshold had the intended effect, an independent samples t-test was used to confirm whether the total sleep time (TST), sleep efficiency, and WASO variables differed between the actigraphy-defined good sleep (GS; more than 6 hours of TST) and poor sleep (PS; 6 hours of TST or less) groups. Specifically, it

was expected for mean values of TST and sleep efficiency to be lower and that of WASO to be higher in the actigraphy-defined PS than GS group.

4. **Aims and Hypotheses Based on Actigraphy (Aims 1 and 2)**

A 2 (actigraphy group: GS, PS) x 4 (measure: HAM-A, HAM-D, ERQ-R, ERQ-S) MANOVA was used to determine if mean total scores on the HAM-A, HAM-D, ERQ-R, ERQ-S differed by objective sleep group. Again, we followed up significant omnibus findings with independent t-tests and controlled for multiple comparisons by applying Bonferroni corrections. Since units for independent variables (IVs) differed, IVs were standardized (i.e., z-scored).

5. **Evaluate Associations Between Sleep and Symptomatology Across Sleep Groups (Aim 3)**

To evaluate individual differences, hierarchical multiple linear regressions were conducted to examine predictive relationships among subjective and objective sleep as well as symptom severity and emotion regulation.

A forward-fitted hierarchical multiple regression evaluated the independent variables/predictors of anxiety (i.e., HAM-A) and depression (i.e., HAM-D) symptoms on the dependent variable of subjective sleep (i.e., PSQI global score). Next, the emotion regulation reappraisal (i.e., ERQ-R) and suppression (i.e., ERQ-S) subscales were included as independent variables/predictors as well to test whether they contributed to the effects. Lastly, the actigraphy measurements (i.e., TST, sleep efficiency, WASO) were inputted as the final set of independent variables/predictors to assess associations between subjective and objective sleep measures across participants.

A similar forward-fitted hierarchical multiple regression was applied to test whether the four symptomology measures (i.e., HAM-A, HAM-D, ERQ-R, ERQ-S) predicted each of the actigraphy variables (i.e., TST, sleep efficiency, WASO) as the dependent variable.

III. RESULTS

Transformations were applied to clinical and sleep measures to adhere to assumptions of normality. With actigraphy-derived variables, the distribution for sleep efficiency was negatively skewed, so reflection and log transformations were applied. For WASO, the distribution was positively skewed, so square-root transformation was applied.

From the total of 67 participants, the majority were primarily patients (n = 47; 70.1%) and female (n = 45; 67.2%). The average age across participants was 28.13 (SD = 10.00) years and education level was 16.18 (SD = 3.13) years. In regard to race and ethnicity, 70.1% (n = 47) were Caucasian, 10.4% (n = 7) were African American, 11.9% (n = 8) were Asian, 3.0% (n = 2) reported more than one race, 4.5% (n = 3) endorsed 'other or unknown,' and 20.9% (n = 14) were Hispanic or Latino. For clinical symptom severity (excluding sleep items), the average anxiety level assessed with the HAM-A was 10.82 (SD = 8.72), and the average depression level assessed with the HAM-D was 6.40 (SD = 5.17). As for measures of emotion regulation, the average ERQ subscale scores were 26.57 (SD = 7.56) for reappraisal and 15.60 (SD = 5.95) for suppression. In relation to subjective sleep, the average PSQI global score was 7.96 (SD = 5.02). For objective sleep with actigraphy, the average number of days/nights participants wore the device was 6.85 (SD = 1.49), and 89.6% (n = 60) of participants completed the nightly sleep diary. Actigraphy-derived average TST was 6.54 (SD = 1.29) hours, average sleep efficiency was 91.6% (SD = 3.7%), and average WASO was 36.48 (SD = 17.56) minutes.

A. Assignment of Sleep Quality Groups Based on PSQI

According to the PSQI global score cutoff of > 5, 43 participants were assigned to the PSQ group (66.7% female; M_{age} = 28.74, SD_{age} = 10.02) and 24 were assigned to the GSQ group (67.4% female; M_{age} = 27.04, SD_{age} = 10.07). Chi-square analyses and independent samples t-

tests revealed that groups were generally similar in gender, race, ethnicity, age, and education level (all p's > .05). See Table II for demographic and clinical characteristics across PSQI-defined groups.

B. PSQI Manipulation Check

Independent samples t-tests confirmed the PSQI global score differed significantly between the subjective sleep groups ($t(62) = -11.34$, $p < .001$). The PSQ group ($M = 10.77$, $SD = 3.91$) endorsed higher global scores compared to the GSQ group ($M = 2.92$, $SD = 1.72$). All PSQI component sub-scores also differed between groups, with scores for the PSQ group being significantly higher than those of the GSQ group (all p's < .05).

C. Group Distributions and Differences Based on PSQI (Aims 1 and 2)

Chi-square analysis revealed diagnostic status (healthy controls vs. patients) differed between groups ($\chi^2(1) = 21.54$, $p < .001$), such that more patients were in the PSQ group ($n = 39$; 90.7%) and more healthy controls were in the GSQ group ($n = 16$; 66.7%).

The MANOVA for PSQI-derived groups revealed a main effect of sleep group across all measures of symptomatology ($F(4, 62) = 11.01$, $p < .001$). Follow-up analyses, with corrections for multiple comparisons, showed that there were significant differences in HAM-A ($F(1, 65) = 29.19$, $p < .001$), HAM-D ($F(1, 65) = 28.15$, $p < .001$), ERQ-R ($F(1, 65) = 10.91$, $p = .002$), and ERQ-S ($F(1, 65) = 8.74$, $p = .004$) scores between subjective sleep groups.

Specifically, results showed that participants in the PSQ group ($M = 14.42$, $SD = 7.79$) endorsed higher anxiety symptom severity than the GSQ group ($M = 4.38$, $SD = 6.30$). The PSQ group ($M = 8.51$, $SD = 4.59$) also endorsed higher depression symptom severity than the GSQ group ($M = 2.63$, $SD = 3.88$). Results also showed that those in the PSQ group ($M = 24.44$, $SD = 7.30$) reported lower use of reappraisal than those in the GSQ group ($M = 30.38$, $SD = 6.56$). On

the other hand, the PSQ group (M = 17.12, SD = 5.69) reported higher use of suppression compared to the GSQ group (M = 12.88, SD = 5.53). See Table II and Figure 1 for details.

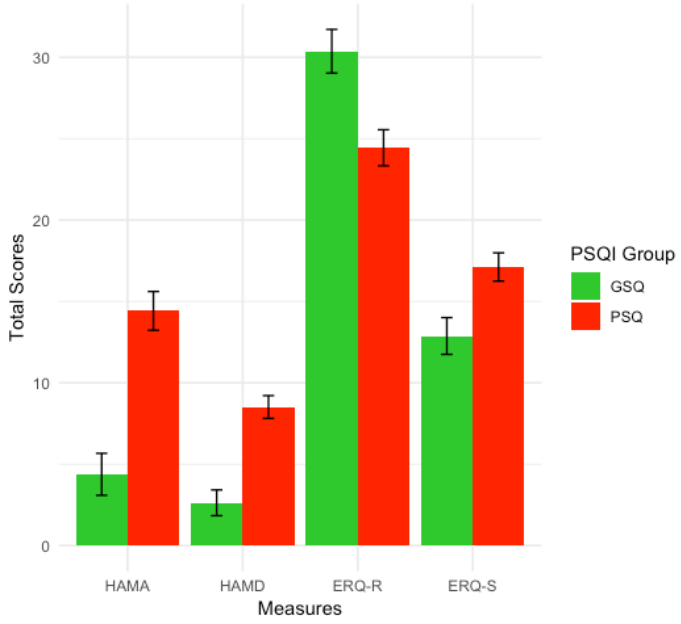
TABLE II

PARTICIPANT CHARACTERISTICS ACROSS PSQI GROUPS

		Good Sleep (PSQI ≤ 5)				**Poor Sleep (PSQI > 5)**			
		HC (*n* = 16)		**PT (*n* = 8)**		**HC (*n* = 4)**		**PT (*n* = 39)**	
		N	%	N	%	N	%	N	%
Gender	Male	5	31.3	3	37.5	1	25.0	13	33.3
	Female	11	68.8	5	62.5	3	75.0	26	66.7
Race/Ethnicity	Caucasian	9	56.3	7	87.5	2	50.0	29	74.4
	African American	2	12.5	0	0.0	1	25.0	4	10.3
	Asian	4	25.0	0	0.0	1	25.0	3	7.7
	Hispanic/Latino	3	18.8	3	37.5	0	0.0	8	20.5
	Other or Unknown	1	6.3	0	0.0	0	0.0	2	5.1
	More than one race	0	0.0	1	12.5	0	0.0	1	2.6
		M	SD	M	SD	M	SD	M	SD
Age (yr)		24.75	10.02	31.63	9.04	24.00	4.24	29.23	10.34
Education (yr)		15.13	2.66	18.38	3.02	16.00	2.71	16.18	3.24
PSQI*		2.31	1.74	4.13	0.83	9.25	4.57	10.92	3.88
Actigraphy	TST (hr)	6.55	0.84	6.63	1.39	6.77	1.54	6.50	1.43
	Sleep Efficiency (%)	92.27	3.17	90.27	3.27	92.81	1.20	91.55	4.06
	WASO (min)	34.26	10.06	43.46	13.86	33.20	12.28	36.30	19.31
ERQ-R*		33.00	5.85	25.13	4.55	25.00	12.06	24.38	6.89
ERQ-S*		11.50	3.81	15.63	7.52	15.75	1.71	17.26	5.94
HAM-A*		0.69	0.79	11.75	5.99	2.50	3.00	15.64	7.06
HAM-D*		0.31	0.48	7.25	3.49	0.50	0.58	9.33	3.98

Note. HC=Healthy Controls, PT=Patients; TST=Total Sleep Time, WASO=Wake After Sleep Onset; PSQI=Pittsburgh Sleep Quality Index, ERQ-R/S=Emotion Regulation Questionnaire Reappraisal/Suppression, HAM-A/D=Hamilton Anxiety/Depression Rating Scale
*$p < 0.05$

Figure 1. Pittsburgh Sleep Quality Index (PSQI) group differences in relation to symptomatology measures.



D. Assignment of Sleep Groups Based on Actigraphy

Based on the actigraphy TST threshold of < 7 hours per night, 44 participants were assigned to the PS group (61.4% female; M_{age} = 28.23, SD_{age} = 10.14) and 23 were assigned to the GS group (78.3% female; M_{age} = 27.96, SD_{age} = 9.95). Chi-square analyses and independent samples t-tests did not reveal significant differences between groups for gender, race, ethnicity, age, education level, completion of sleep diaries, and number of days/nights the actigraph device was worn (all *p*'s > .05). See Table III for demographic and clinical characteristics across actigraphy-defined groups.

E. Actigraphy Manipulation Check

When examining the assignment of groups based on actigraphy, independent samples t-tests partially confirmed the effect of the set threshold, with objective sleep groups differing

between measures of TST ($t(62) = 8.44$, $p < .001$) but not sleep efficiency ($p = .253$) or WASO ($p = .264$). TST for the PS group ($M = 5.92$, $SD = 1.07$) was significantly higher than that of the GSQ group ($M = 7.73$, $SD = 0.69$), but sleep efficiency and WASO were generally equivalent.

F. <u>Group Distributions and Differences Based on Actigraphy (Aims 1 and 2)</u>

Chi-square analysis did not reveal significant differences in diagnostic status between actigraphy-defined groups ($p = .940$). Thus, the number of patients and healthy controls in both the PS and the GS groups were similar. The MANOVA results for actigraphy-defined groups did not reveal group effects ($F(4, 62) = 0.28$, $p = .892$). There were no significant differences in HAM-A ($p = .576$), HAM-D ($p = .667$), ERQ-R ($p = .763$), and ERQ-S ($p = .501$) scores between objective sleep groups. See Table III and Figure 2 for details.

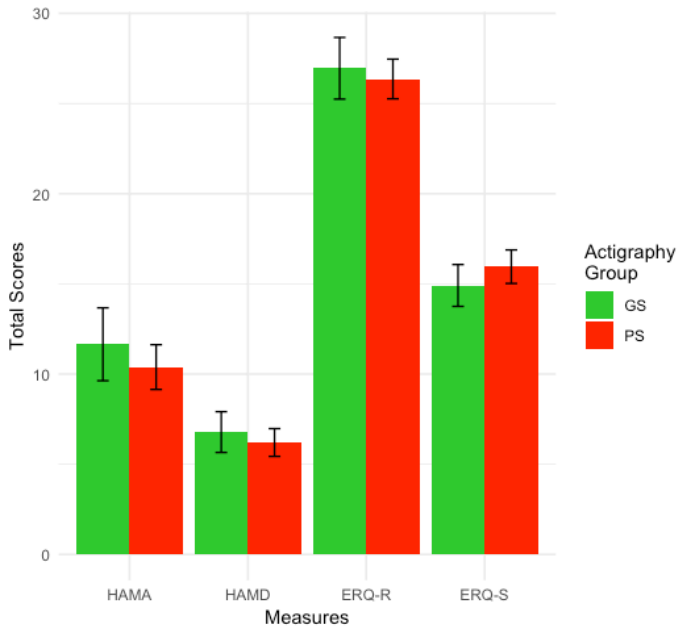
TABLE III

PARTICIPANT CHARACTERISTICS ACROSS ACTIGRAPHY GROUPS

		Good Sleep (TST = 7-9 hrs)				Poor Sleep (TST < 7 hrs)			
		HC (*n* = 7)		PT (*n* = 16)		HC (*n* = 13)		PT (*n* = 31)	
		N	%	N	%	N	%	N	%
Gender	Male	1	0.1	4	0.3	5	0.4	12	0.4
	Female	6	0.9	12	0.8	8	0.6	19	0.6
Race/Ethnicity	Caucasian	6	0.9	13	0.8	5	0.4	23	0.7
	African American	0	0.0	2	0.1	3	0.2	2	0.1
	Asian	1	0.1	1	0.1	4	0.3	2	0.1
	Hispanic/Latino	0	0.0	2	0.1	3	0.2	9	0.3
	Other or Unknown	0	0.0	0	0.0	0	0.0	0	0.0
	More than one race	0	0.0	0	0.0	0	0.0	0	0.0
		M	SD	M	SD	M	SD	M	SD
Age (yr)		23.71	5.06	29.81	11.08	25.08	10.81	29.55	9.72
Education (yr)		15.71	2.98	16.88	3.36	15.08	2.50	16.39	3.28
PSQI		5.00	5.51	11.00	4.38	3.00	2.27	9.13	4.31
Actigraphy	TST (hr)*	7.70	0.54	7.75	0.76	5.99	0.50	5.88	1.24
	Sleep Efficiency (%)	92.92	3.45	91.97	3.21	92.09	2.60	91.01	4.27
	WASO (min)	37.25	19.95	41.06	19.92	32.32	12.34	35.69	17.88
ERQ-R		28.71	10.61	26.19	7.15	32.85	5.79	23.65	6.10
ERQ-S		12.71	3.55	15.88	6.06	12.15	4.16	17.55	6.25
HAM-A		1.29	2.56	16.19	7.95	0.92	0.76	14.35	6.48
HAM-D		0.57	0.53	9.50	4.13	0.23	0.44	8.71	3.89

Note. HC=Healthy Controls, PT=Patients; TST=Total Sleep Time, WASO=Wake After Sleep Onset; PSQI=Pittsburgh Sleep Quality Index, ERQ-R/S=Emotion Regulation Questionnaire Reappraisal/Suppression, HAM-A/D=Hamilton Anxiety/Depression Rating Scale
*$p < 0.05$

Figure 2. Actigraphy group differences in relation to symptomatology measures.



G. Relationships of Sleep and Symptomatology (Aim 3)

1. Subjective Sleep/PSQI

Hierarchical multiple regression analyses with PSQI global score as the DV revealed a significant model (R^2 = .500, $F(2, 64) = 32.04$, $p < .001$) (VIF < 4), in which anxiety (HAM-A) (B = 0.360, *s.e.* = 0.158, $p = .026$) and depression (HAM-D) (B = 0.380, *s.e.* = 0.158, $p = .019$) were significant predictors. When reappraisal (ERQ-R) and suppression (ERQ-S) were included as predictors, the model remained significant (R^2 = .537, $F(4, 62) = 17.97$, $p < .001$) (VIF < 4). Anxiety (HAM-A) (B = 0.344, *s.e.* = 0.156, $p = .031$), depression (HAM-D) (B = 0.351, *s.e.* = 0.155, $p = .027$), and reappraisal (ERQ-R) (B = -0.199, *s.e.* = 0.009, $p = .034$) significantly predicted PSQI global score, but suppression (ERQ-S) did not ($p = .459$).

Following the submission of actigraphy measures of TST, sleep efficiency, and WASO as IVs, the model was still significant (R^2 = .546, $F(7, 59) = 10.12$, $p < .001$), but VIF was > 4 for

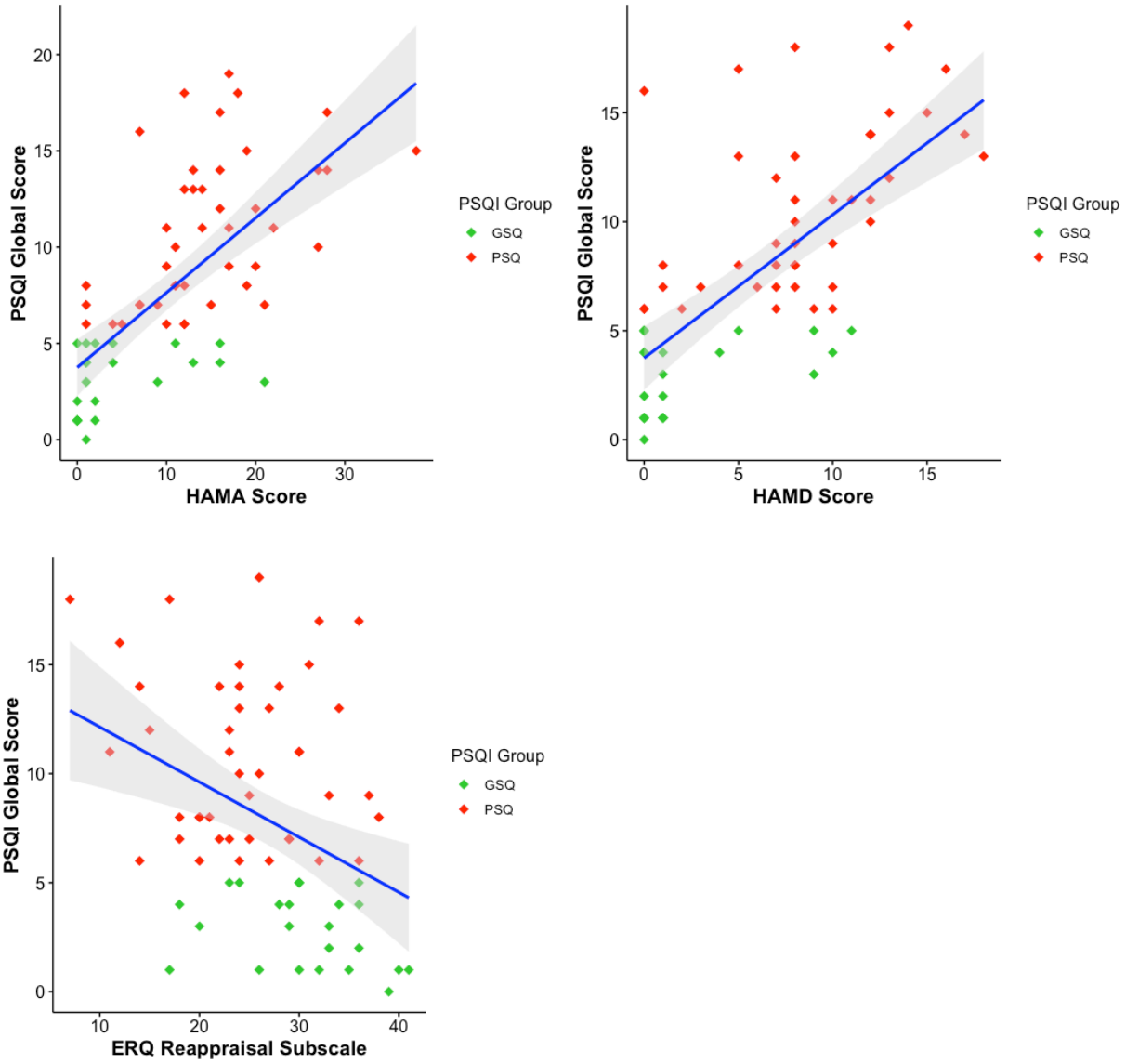
sleep efficiency and WASO. Therefore, the same analysis was performed without including these IVs simultaneously.

When the model included actigraphy-based sleep efficiency without WASO and TST, it was significant (R^2 = .546, $F(6, 60) = 12.00$, $p < .001$) (VIF < 4). However, results were similar to prior findings as sleep efficiency and TST did not predict subjective sleep/PSQI nor did suppression (ERQ-S) (all p's > .05). Rather the significant finding was explained by anxiety (HAM-A) (B = 0.334, *s.e.* = 0.158, p = .039), depression (HAM-D) (B = 0.376, *s.e.* = 0.160, p = .022), and reappraisal (ERQ-R) (B = -0.203, *s.e.* = 0.094, p = .035).

Similarly, with WASO but not sleep efficiency, the model was significant (R^2 = .544, $F(6, 60) = 11.94$, $p < .001$) (VIF < 4) but was again explained by anxiety (HAM-A) (B = 0.329, *s.e.* = 0.160, p = .044), depression (HAM-D) (B = 0.374, *s.e.* = 0.160, p = .023), and reappraisal (ERQ-R) (B = -0.200, *s.e.* = 0.094, p = .038) as suppression (ERQ-S), TST, and WASO did not predict PSQI global score. (all p's > .05). All results were maintained when controlling for the diagnostic status of participants.

Zero-order correlations, with Bonferroni correction, collapsed across sleep groups showed PSQI global score to be positively correlated with anxiety (HAM-A) ($r(65) = .67$, $p < .001$) and depression (HAM-D) ($r(65) = .68$, $p < .001$) while negatively correlated with reappraisal (ERQ-R) ($r(65) = -.38$, $p = .001$). See Figure 3.

Figure 3. Relationships between Pittsburgh Sleep Quality Index (PSQI) and anxiety (HAM-A), depression (HAM-D), and reappraisal (ERQ-R).



Follow-up post-hoc analyses were performed with each PSQI component sub-score as the DV alone and the significant predictors of anxiety, depression, and reappraisal as the IVs. Anxiety (HAM-A) was found to be a significant predictor of PSQI components 1 (subjective

sleep quality) (B = 0.295, *s.e.* = 0.140, p = .039) and 5 (sleep disturbances) (B = 0.251, *s.e.* = 0.120, p = .040). Depression (HAM-D) was found to be a significant predictor of PSQI components 1 (subjective sleep quality) (B = 0.289, *s.e.* = 0.140, p = .043), 2 (sleep latency) (B = 0.877, *s.e.* = 0.403, p = .033), and 7 (daytime dysfunction) (B = 0.389, *s.e.* = 0.159, p = .017). Reappraisal (ERQ-R) was a significant predictor of PSQI component 7 (daytime dysfunction) (B = -0.216, *s.e.* = 0.093, p = .023). All other component findings were not significant (all p's > 0.05).

Zero-order correlations, with Bonferroni correction, showed PSQI component 1 (subjective sleep quality) to be positively correlated with anxiety (HAM-A) ($r(65) = .66$, $p < .001$) and depression (HAM-D) ($r(65) = .65$, $p < .001$). PSQI component 2 (sleep latency) was positively correlated with depression (HAM-D) ($r(65) = .57$, $p < .001$). PSQI component 5 (step disturbances) was positively correlated with anxiety (HAM-A) ($r(65) = .55$, $p < .001$). PSQI component 7 was positively correlated with depression (HAM-D) ($r(65) = .59$, $p < .001$) and negatively correlated with reappraisal (ERQ-R) ($r(65) = -.39$, $p = .001$).

2. <u>Objective Sleep/Actigraphy</u>

Hierarchical multiple regression analyses were repeated with each of the actigraphy variables as the DVs. With TST as the DV, the initial model was not significant ($R^2 = .006$, $F(2, 64) = 0.19$, $p = .831$) (VIF < 4), and anxiety (HAM-A) ($p = .612$) and depression (HAM-D) ($p = .815$) were not significant predictors. The model remained non-significant when emotion regulation variables were included ($p = .711$), and neither reappraisal (ERQ-R) ($p = .264$) nor suppression (ERQ-S) ($p = .394$) were significant predictors of TST.

When sleep efficiency was the DV, the initial model with anxiety (HAM-A) ($p = .341$) and depression (HAM-D) ($p = .094$) as IVs was again not significant ($R^2 = .052$, $F(2, 64) = 1.76$,

p = .180) (VIF < 4). The model remained non-significant when reappraisal (ERQ-R) (p = .254) and suppression (ERQ-S) (p = .274) were included as predictors of sleep efficiency (p = .232).

Lastly, with WASO as the DV, the initial model was not significant (R^2 = .048, F(2, 64) = 1.61, p = .207) (VIF < 4). Anxiety (HAM-A) (p = .122) and depression (HAM-D) (p = .078) did not significantly predict WASO. Inclusion of reappraisal (ERQ-R) (p = .159) and suppression (ERQ-S) (p = .447) into the model also showed that they were not significant predictors, with the model remaining non-significant as well (p = .245).

IV. DISCUSSION

The study consisted of individuals with and without internalizing psychopathologies. The primary goal was to examine clinical symptoms (i.e., anxiety and depression) and emotion regulation between sleep groups based on subjective and objective sleep measures and relationships between sleep, symptoms, and emotion regulation. Overall, results revealed group effects based on subjective sleep/PSQI group assignment but not objective sleep/actigraphy-based groups. Also, significant relationships were found between self-reported sleep, clinical symptoms, and emotion regulation. No such relationships were detected for actigraphy. Findings suggest close links between mood, regulation, and self-perceived sleep particularly in patients with anxiety and/or depression.

Aims were partially supported. For Aim 1, the poor sleep quality group based on a standard PSQI cut-point comprised significantly more patients than healthy controls. As expected, more patients reported poor sleep quality, which is in line with the literature, indicating that more individuals with internalizing conditions report clinically problematic sleep compared to those without psychopathology (Almeida & Pfaff, 2005; Cox & Olatunji, 2016). Previous studies have not only shown that the presence of psychiatric symptoms predicted more self-perceived sleep disturbances but that sleep disturbances may also predict the development of related disorders, such as generalized anxiety and depression (Benca et al., 1992).

However, the poor sleep group based on actigraphy as demarcated by a total sleep time (TST) of < 7 hours did not yield significant differences between distributions of patients and healthy controls. Interestingly, the average TST was relatively short in this cohort, i.e., 6.54 (*SD* = 1.29) hours, which largely consisted of patients (70.1%), although 13 out of 20 healthy controls (i.e., 65%) experienced less than 7 hours of sleep over the course of a week highlighting

the prevalence of problematic sleep in the U.S. (Y. Liu et al., 2016). The Center for Disease Control and Prevention defines short sleep duration as less than 7 hours of sleep per 24-hour period. Therefore, our sample is more representative of individuals who experience inadequate sleep than adequate sleep, which may have impeded our ability to detect significant effects. In addition to relatively short sleep duration, our sample exhibited an average sleep efficiency of 91.6% and WASO of 36.48 minutes. Studies examining actigraphy parameters in patients with insomnia disorder suggest thresholds of sleep efficiency of less than 92% and WASO at > 25 minutes (Natale et al., 2009), providing further evidence that our sample more closely represented a population with problematic sleep than good sleep. Furthermore, results did not show significant differences for the other actigraphy measures of sleep efficiency and WASO. This suggests that using TST as a cut-point may not appropriately account for all of the distinct estimates of sleep derived through actigraphy (Patel et al., 2015).

For Aim 2, as hypothesized, the subjective sleep groups also differed in both symptom severity and emotion regulation in the expected directions. Specifically, the poor sleep quality group exhibited higher anxiety and depression levels and reported less habitual use of reappraisal, and more habitual use of suppression to regulate emotions, compared to those with the good sleep quality group. As discussed, more patients endorsed poor sleep quality, reinforcing that high levels of anxiety and depression symptoms are often observed with poor sleep (Cox & Olatunji, 2016). Prior work has shown individuals with worse self-reported sleep to have a poorer ability in implementing cognitive reappraisal (Mauss et al., 2013). Moreover, another study found decreased sleep quality, assessed with the PSQI, to be correlated with increased expressive suppression in individuals with high negative affect (Latif et al., 2019). However, this was again not found for objective sleep. The actigraphy-based sleep groups

exhibited similar levels of anxiety and depression and reported similar habitual use of reappraisal and suppression.

Regarding Aim 3, individual differences were detected for subjective sleep/PSQI global score, as anxiety and depression positively predicted self-reported sleep quality while reappraisal negatively predicted it. Dissection of the PSQI components revealed that increased anxiety was significantly related to worse sleep quality and more sleep disturbances. Increased depression was found to be significantly related to worse sleep quality, increased sleep latency, and more daytime dysfunction. With regard to emotion regulation, increased reappraisal significantly corresponded with less daytime dysfunction. This is consistent with previous work, as higher levels of symptom severity have been shown to correspond to worse sleep (Alvaro et al., 2013; Chorney et al., 2008; Gregory et al., 2011). Other studies (independent of sleep considerations) have also shown higher internalizing problems in relation to more deficits in emotion regulation (Hatzenbuehler et al., 2008; Zeman et al., 2002). Findings provide support for the role of sleep in both mood and emotion regulation.

Contrary to hypotheses, there was no association between suppression and sleep despite finding the expected association between reappraisal and sleep. For those with anxiety and depression, emotion dysregulation is generally characterized with an inverse relationship such that individuals report more use of maladaptive forms of regulation (e.g., suppression) and less use of adaptive forms of regulation (e.g., reappraisal) (Aldao et al., 2010). Results suggests individual differences in suppression may not be robustly linked with self-reported sleep quality.

Continuing with Aim 3, individual differences were not detected for objective sleep/actigraphy. No significant associations were found between symptom severity or emotion regulation and TST, sleep efficiency, or WASO. This corresponded with a study also showing no

relationships between anxiety and depression levels and these exact actigraphy variables (Du-Quiton et al., 2010). Another study showed depressive symptoms but not symptom severity to be negatively correlated with sleep efficiency only (Pillai et al., 2014). However, these studies differed in populations of interest (e.g., inpatient, outpatient, college students). There seems to be a lack of research examining actigraphy and emotion regulation in naturalistic sleep settings. One study involved the manipulation of sleep in healthy adolescents showing that shortened sleep worsened mood and emotion regulation ability (Baum et al., 2014). Our null findings may be due to restriction of range. Therefore, further study is necessary before drawing firm conclusions.

Extending upon differences seen thus far between subjective and objective sleep and symptomatology, findings revealed no significant relationships when directly assessing the two methodologies as well. None of the actigraphy variables predicted the PSQI. Findings across all aims provide further support for the discrepancies between subjective and objective assessments of sleep (Bei et al., 2010; Girschik et al., 2012; Klumpp et al., 2017; McCall & McCall, 2012; O'Brien et al., 2016; Van Den Berg et al., 2008; Van Ravesteyn et al., 2014). In a previous study across patients diagnosed with anxiety or depression, the PSQI was not found to be associated with the same actigraphy estimates (Klumpp et al., 2017). Similar results of weak correlations were shown in nonclinical samples too (Grandner et al., 2006).

All in all, findings partially support our hypotheses, and disagreement between self-report and actigraphy findings are consistent with the literature. A primary explanation for the null results for objective sleep may be the comparison made with a subjective measure of emotion regulation. For emotion regulation, strategies are recalled retrospectively and thus frequently subject to bias (Lewis et al., 2010). Research also suggests that one's perception of engaging in emotion regulation is not equivalent to successfully objectively implementing it (B. Q. Ford et

al., 2017). Another explanation alludes to current literature suggesting that problematic sleep in patients also relies heavily on self-report. The PSQI has been shown to be closely correlated with psychiatric symptoms (Buysse et al., 2008), which may help explain significant PSQI findings and null actigraphy results.

Findings need to be taken in the context of important limitations. Participants were largely young adults and the majority of participants consisted of females diagnosed with an anxiety disorder, therefore, results may not generalize to cohorts who differ in clinical and demographic characteristics. Participants were not screened for sleep disorders (e.g., insomnia, sleep apnea) and participants were not instructed to abstain from substances that interfere with sleep (e.g., caffeine, alcohol). Also, not all participants completed sleep logs related to actigraphy.

Additionally, we used total sleep time to assign participants into good and poor sleep groups. The utility of actigraphy overall is well-validated but the relationships between actigraphy variables may be further delineated (Smith et al., 2018). Use of other actigraphy variables to assign participants into groups may have led to different findings. The cross-sectional and correlational nature of the study cannot allow for direct inferences between sleep, mood, and emotion regulation. Lastly, actigraphy involved 15-second epochs, consistent with some studies (Mantua et al., 2016) but not with others that implemented 30-second epochs (Marino et al., 2013).

Despite limitations, results contribute to addressing the fundamental gaps in the psychiatric literature regarding sleep. Results highlight how self-reported sleep quality differs from objective sleep parameters when examining symptomatology of internalizing conditions and suggests self-perceived sleep plays a role in clinical symptoms and emotion regulation. The

study also advances our understanding of sleep dysfunction as a transdiagnostic and dimensional construct.

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VITA

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PUBLICATIONS:	Feurer, C., Jimmy, J., **Chang, F.**, Langenecker, S. A., Phan, K. L., Ajilore, O., & Klumpp, H. (Under Review). Resting State Functional Connectivity Correlates of Rumination and Worry in Internalizing Psychopathologies. *Journal of Affective Disorders*. Kinney, K. L., Burkhouse, K. L., **Chang, F.**, MacNamara, A., Klumpp, H., & Phan, K. L. (Under Review). The Late Positive Potential during Working Memory Load as a Predictor and Index of Anxiety Improvement Following CBT and SSRI Treatment. *Progress in Neuropsychopharmacology & Biological Psychiatry*. **Chang, F.** & Klumpp, H. (Under Review). Sleep Quality and Emotion Recognition in Individuals With and Without Internalizing Psychopathologies. *Psychiatry Research*. Crane, N. A., **Chang, F.**, Kinney, K. L., & Klumpp, H. (Under Review). Individual Differences in Striatal and Amygdala Response to Emotional Faces Predict Symptom Severity in Social Anxiety Disorder. *NeuroImage: Clinical*. Conway, C. C., **Chang, F.**, Young, K. Y., & Craske, M. G. (In Press). Threat conditioning and trait-based vulnerability to personality disorder. *Journal of Personality Disorders*.

POSTERS:

Chang, F., Uribe, M., & Klumpp, H. (2020, March). *Actigraphy-Measured Sleep and Negative Repetitive Thinking in Depressed and Anxious Adults*. Invited poster for the 40th Annual Meeting of the Anxiety and Depression Association of America, San Antonio, TX.

Uribe, M., **Chang, F.**, & Klumpp, H. (2020, March). *Links Between Subjective Attentional Control and Behavioral Inhibition in the Context of Emotional Distractors*. Poster presented at the 6th Annual Meeting of the UIC Psychology Cross Program Conference, Chicago, IL.

Chang, F., Jimmy, J., & Klumpp, H. (2019, March & September). *Links Between Subjective and Objective Sleep and Emotion Processing in Anxiety and Depression*. Poster presented at the 39th Annual Meeting of the Anxiety and Depression Association of America, Chicago, IL & UIC Psychiatry's 10th Annual Research Forum (Extravaganza), Chicago, IL.

Shvartsur, A., **Chang, F.**, Lee, H., Wright, M. J., Apostolova, L., & Woo, E. (2018, February). *Longitudinal Assessment of Metamemory in Mild Cognitive Impairment and Alzheimer's Disease*. Poster presented at the 46th Annual Meeting of the International Neuropsychological Society, Washington, DC.

McNett, S., **Chang, F.**, Vujanovic, A., Dick, D., Kendler, K., Amstadter, A. B., & Berenz, E. C. (2017, November). *Sex Differences in the Association between Age of Alcohol Use Initiation and Internalizing Symptoms in Trauma-Exposed Young Adults*. Poster presented at the 33rd Annual Meeting of the International Society for Traumatic Stress Studies, Chicago, IL.

Garcia, C. B., **Chang, F.**, Gomez, K., Kreisel, C., & Woo, E. (2017, May). *Everyday Cognition Questionnaire Discrepancy Reports in Mild Cognitive Impairment Show No Significant Difference Between Subject and Study Partner Responses*. Poster presented at the UCLA Neuroscience Poster Day, Los Angeles, CA.

Liu, S., **Chang, F.**, Kreisel, C., Gomez, K., Woo, E., Ringman, J., Apostolova, L. G., Wolf, S., & Nuñez, C. S. (2017, February). *Cognition, Neuropsychiatric Symptoms and Everyday Functioning in Latino Older Adults*. Poster presented at the 45th Annual Meeting of the International Neuropsychological Society, New Orleans, LA.

Chang, F., Hanulik, I., Woo, E., Wright, M. J., He, A., De Leon, F., Roberg, B., Apostolova, L. G., Ringman, J., & Nuñez, C. S. (2016, August). *Components of Prospective Memory in Healthy Aging and Mild Cognitive Impairment*. Poster presented at the 2016 Turken Research Award Symposium, Los Angeles, CA.

Chang, F., Conway, C. C., & Craske, M. G. (2016, January). *Change In Expectancy for Aversive Events During Extinction*. Invited poster for the 36th Annual Meeting of the Anxiety and Depression Association of America, Philadelphia, PA.

Chang, F., Gautam, A., Conway, C. C., & Craske, M. G. (2015, April). *Interrelations among Diverse Measures of Distress Tolerance*. Poster presented at the 35th Annual Meeting of the Anxiety and Depression Association of America, Miami, FL.