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Disentangling the Impact of Poor Sleep from Depressive Symptoms on Emotion Regulation

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by

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Abstract

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Depressive symptoms and sleep are both strongly associated with deficits in emotional functioning (Durmer & Dinges, 2005; van der Helm & Walker, 2010). Although sleep and depression are tightly intertwined, understanding their independent and conjoint impact on emotional functioning is imperative. Given the limitations of previous designs, the primary goal of this study was to examine the separate impact of poor sleep quality and depressive symptoms on emotion regulation. In order to accomplish this goal, we preselected groups on the basis of their sleep and depression profiles: individuals with mainly sleep problems (N = 30), individuals with mainly depressive symptoms (N = 10), individuals who scored highly on both problems (N = 37), as well as individuals who are low on both problems (N = 33). Main predictions were that sleep would be uniquely associated with poor trait and laboratory emotion regulation. Results were contrary to hypotheses in that we found main effects of depression on all self-reported measures of emotion regulation and a main effect of sleep on rumination alone. Sleep and depression both predicted affective consequences of laboratory emotion regulation but in the opposite of the expected direction: greater severity scores predicted more benefit from instructed emotion regulation strategy use in the laboratory. Further discussion centers on the complexity of sleep problems and future directions for a greater understanding of sleep within emotion regulation.

Disentangling the Impact of Poor Sleep from Depressive Symptoms on Emotion Regulation

Major Depressive Disorder (MDD) is one of the most common mental health problems in the United States, with a 6.7% annual prevalence rate in the adult population (Kessler et al., 2005). Moreover, depression rates appear to be rising in younger birth cohorts: compared to adults 60 and older, young adults are now 70% more likely to experience an episode of depression in their lifetime (Kessler et al., 2005). In sum, MDD is a major public health concern, the fourth leading cause of disease burden worldwide (Ustin, 2004) and contributes to other health problems (Wang et al., 2005).

MDD is classified as a mood disorder (DSM-5; American Psychiatric Association, 2013), and is characterized by a profound disturbance in emotional functioning, with symptoms ranging from loss of interest to appetite problems. One key somatic symptom of depression is sleep disturbance (e.g., hypersomnia, insomnia, sleep awakenings.). Sleep disturbance is among the symptoms of depression, and are thought to contribute to the emotional disruptions that are characteristic of this disorder (e.g. van der Helm & Walker, 2010).

Although connected to depression, sleep problems have also been seen as an important public health problem in their own right. There are a number of primary sleep disorders (e.g. insomnia disorder, restless leg syndrome) that warrant independent diagnosis and require comprehensive care. Recent research has highlighted the social and economic burdens of sleep disorders (Kessler et al., 2011). For example, several commentators have called attention to a significant reduction in sleep time worldwide (Bixler, 2009; Matricciani et al., 2012; Mindell et al., 2009), which can contribute to negative outcomes such as accidents and increased absenteeism (Kessler et al., 2011). Moreover, impaired sleep quality has been tied to poor mental health, including internalizing disorders (Touchette et al., 2012) and prevailing low mood (Bower et al., 2010).

Difficulties in sleep have been implicated extensively in deficits in emotional functioning. Laboratory designs involving sleep deprivation replicate observational conclusions that disturbed sleep increases negative emotions and impairs overall emotion regulation (Durmer & Dinges, 2005; Mauss et al., 2013; van der Helm & Walker, 2010). The close link between sleep and emotion manifests in the ways that poor sleep contributes to high

prevailing negative affect and low prevailing positive affect (for a review, see Baglioni, 2010). In fact, sleep and depressive symptoms are so highly correlated (e.g. Agargun, Kara, & Solmaz, 1997) that it has been difficult for scientists to disentangle their impact on emotion.

Because of the high rate of occurrence of sleep complaints within major depression (e.g. ~ 90%, Tsuno, Besset, & Ritchie, 2005), one cannot readily determine from a cross-sectional study of depressed people whether poor sleep quality or depression have independent or conjoint impacts on emotional functioning. Profoundly, impairment in *emotion regulation (*efforts to modify the experience and expression of emotions) is a common consequence of sleep deprivation, as well as a common difficulty among depressed individuals (i.e. Fairholme et al., 2013). Emotion regulation (Gross, 1998) specifically has been a target of intervention attempts within depressive disorders (Kovacs et al., 2006), and emotion regulation deficits are a common complaint among those with sleep problems (Baum et al., 2014). At present, because it co-occurs with high rates of depression, it is unclear whether poor sleep quality contributes uniquely to impairments in emotion regulation.

While problematic sleep quality commonly co-occurs with depressive symptoms, there is value in attempting to disentangle their contribution to emotion dysregulation. For example, isolating variables contributing to disturbed emotional functioning are potentially relevant to future clinical targets and intervention efforts (e.g. aiding in comprehensive treatment planning based on the predictor variable and its course). Detailing the impact of certain symptoms (i.e. disturbance in sleep) on relevant difficulties in emotion has the potential to provide more concrete, specific, and individual-focused interventions. In this study, we attempted to isolate key factors to begin to disentangle the effects of poor sleep quality and depression on emotion regulation. The following sections explain the rationale for the study and how sleep and depression may contribute, both independently and jointly, to disturbance in emotional functioning (and specifically, emotion regulation).

Poor Sleep Quality and Depression: Correlation and Temporal Precedence

While poor sleep quality and depression are related, exactly how and when they are related has remained elusive, in part because study samples typically include individuals who are high on both sleep problems and depressive symptoms (e.g. Tsuno, Besset, & Ritchie, 2005). However, recent research suggests that sleep problems may precede depression (i.e. Alvaro, Roberts, & Harris, 2013).

A tight connection between sleep complaints and depression has been consistently documented (e.g. Rodin, McAvay, & Timko, 1988). In fact, during the creation of the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), the gold standard in self-report of sleep quality, the authors used depressed inpatients as one of the two original groups labeled as "poor" sleepers – with the only other group being those diagnosed with a primary sleep disorder (Buysse et al., 1989). Interestingly, though sleep problems is one symptom in all diagnostic measures of depression, work is still needed to clarify their place within depression and their role in emotion regulation. Regardless, the strong tie between disordered sleep and depression is robust across samples (e.g. inpatients, Selvi, 2010, adolescents, Urrila et al., 2012, and suicidal patients, Agargun, Kara, & Solmaz, 1997), demonstrating that poor sleep quality is associated with a high incidence of depression. In a comprehensive literature review, one group concluded that as many as 90% of depressed patients complain about their sleep quality (Tsuno, Besset, & Ritchie, 2005).

Recently, a new line of research has begun to examine why these variables are so strongly correlated: specifically, what are the processes that explain their interconnectedness (i.e. Baglioni et al., 2010). Although this research is in its early stages, one initial goal involved understanding temporal precedence between these variables (Alvaro, Roberts, & Harris, 2013). One review of longitudinal studies assessed the effect of early childhood sleep problems on the same children later in life and concluded that sleep problems in childhood put children at four times the risk for internalizing problems at an 18 year follow up compared to those without sleep problems in childhood (Touchette et al., 2012). Interpreting these findings in terms of the underlying processes, some scholars have suggested that deficits in emotion result from sleep impairment and contribute to later depression (i.e., Gruber & Cassoff, 2014). Disordered sleep, as it is, has the potential to be a trigger for future depression; however, it may manifest as a prodromal symptom or a warning sign of an emerging disorder. Various mechanisms have been suggested to explain this link, ranging from biological (increased inflammatory dysregulation; Muller, Myint, & Schwarz, 2011) to common factors (genetic, familial, social, or environmental; Kaneita et al., 2009). Given the high rate of emotion regulatory impairment seen in both depressed and sleep-disordered individuals, understanding the differential impact of each of these problems may be valuable, not only for defining pathways, but for improving intervention efforts as well. Disentangling the unique impact of each on emotional functioning was a main goal of the present study.

Method and Approach: Isolating the Unique Impact of Disordered Sleep and Depression on Emotional Functioning

There are several potential research strategies that could be utilized to isolate the impact of sleep and depression on emotion. For example, manipulating sleep deprivation in healthy individuals is one way to assess the unique impact of sleep on emotion regulation. Manipulating sleep involves a period of forced sleep deprivation (typically in one night's sleep) and observing subsequent effects; however, these paradigms primarily model the effect of acute sleep loss, which could prove quite different from the effects of chronically poor sleep quality (Talbot et al., 2010; Hamilton et al., 2008). Other strategies involve naturalistic observation of key variables as they vary over time. While naturalistic observation has the advantage of having good ecological validity, longitudinal studies are often costly, time-intensive, and challenging to carry out. Given how few efforts there have been to isolate the effects of sleep and depression variables, selective sampling can be a reasonable first step, as has been done elsewhere (i.e., burnout and insomnia, Vela-Bueno, et al., 2008). Therefore, preselecting pure groups may be one means to examine the separate and conjoint effects of depression and sleep quality on emotion regulation. In this instance, "pure" groups refer to groups with specific profiles of depression and sleep complaints (e.g., one group consisting of participants with high rates of sleep disturbances, but little to no reported depressive symptoms).

The Impact of Disordered Sleep and Depression: Deficits in Emotion Regulation

Arguably, depression and sleep exert their strongest impact in the realm of emotion. For instance, preliminary work shows more robust effect sizes for sleep impairment of affect and mood than on cognitive or motor performance (Pilcher & Huffcutt, 1996; Kahn, Sheppes, & Sadeh, 2013). Various sleep deprivation paradigms have found that reduced sleep duration is associated with an increase in negative affect (Durmer & Dinges, 2005), low positive affect (Bower et al., 2010) and delayed recovery from negative events (Hamilton et al., 2008; for a more comprehensive review, see Kahn, Sheppes, & Sadeh, 2013). In addition, both problematic sleep and depressive symptoms have been associated with a host of maladaptive emotion responses/strategies: emotion regulation impairment (including worry and rumination, Mauss, Troy, & LeBourgeois, 2013), higher levels of maladaptive emotion regulation strategies (e.g. Lei et al., 2014), and emotion dysregulation in general (Fairholme et al., 2013).

In a study testing mediation, we found that poor sleep had detrimental effect on emotion regulation (and subsequently, on depression; O'Leary, Bylsma, & Rottenberg, 2016). Both impaired sleep quality and duration have been associated with poorer emotion regulation in children and adolescents, as indexed by self- and observer-reports

(Baum et al., 2014; Vriend et al., 2013). Poor sleep has been shown to lead to disturbed emotion regulatory ability in the laboratory (Mauss, Troy, & LeBourgeois, 2013). Sleep may interfere with a number of putative emotion regulatory mechanisms; for example, disordered sleep impairs executive functioning, which is likely to interfere with emotion regulatory capacity (for a review, see Gruber & Cassoff, 2014).

In turn, impairments in emotion regulation have been linked to depressive symptoms (O'Leary et al., 2016). Depressed individuals report higher use of maladaptive emotion regulation strategies (Gilbert & Gruber, 2014), including tendencies towards switching to and spontaneously using maladaptive strategies (Joormann & Gotlib, 2010). Additionally, improvement in emotion regulation ability can predict recovery from depression (Ardite & Joorman, 2011; Berking et al., 2014).

Because these variables and processes are likely complex and bidirectional, few studies have attempted to assess all variables involved; however, some work has tried to provide indirect answers via post-hoc statistical control. For instance, one study found that emotion dysregulation was still associated with depression even when controlling for insomnia symptoms (Fairholme et al., 2013). However, given the high incidence rate of sleep problems within depression and depressive symptoms among troubled sleepers, insomnia alone may not account for these factors. Additionally, in experimental work, controlling for sleep quality can render associations between depression and emotion regulation indices nonsignificant (e.g., the relationship between depression and blunted respiratory sinus arrhythmia – an index of impaired emotion regulation – disappears when including sleep quality as a variable; Bylsma et al., 2014). Additionally, disordered sleep may be exerting a key differentiating emotional impact. When controlling for depressive symptoms, sleep problems remain associated with emotion dysregulation, albeit decreasing to a trend-level effect (e.g. Mauss, Troy, & LeBourgeois, 2013). In a model study on emotion regulation, depression, anxiety, and stress, the authors found that emotion regulation played a large role in poor quality sleepers compared to good quality sleepers. This impact was seen across domains, in affecting depression, anxiety, and stress (Markarian et al., 2013).

Specificity of Impact: Non-Emotion Domains

Although both impaired sleep quality and depression have historical ties with problematic emotional functioning, each construct is also known to affect other domains of functioning. For example, poor sleep has been related to physical health problems, including low physical activity and obesity (i.e. Calhoun et al., 2011); this

negative relationship is also seen with depression (Uebelacker et al., 2013). Additionally, poor sleep has been associated with deficits in cognitive functioning, including reduced attentional information capacity (e.g. Alfarra et al., 2015) and working memory (e.g. Ozdemir et al., 2013). Several reviews have covered the relationship between depression and cognitive impairments (i.e. Wagner et al., 2014). Research in other domains suggests that poor sleep and depression may both contribute to impairment in nonemotional functioning as well. One question, then, is whether poor sleep quality and depression has differential ties to the domain of emotion regulation (Pilcher & Huffcutt, 1996) than they do to other domains.

The Present Study

Although sleep and depression are tightly intertwined, understanding their independent and conjoint impact on emotional functioning is imperative. As worldwide sleep problems increase (e.g. Matricciani et al., 2012) and the burden of depression escalates (e.g. Ustin, 2004), it becomes all the more important to establish how each might respectively contribute to problems in emotional functioning. As it stands, current study paradigms model this with acute sleep deprivation (which can look different from poor sleep quality, e.g. Hamilton et al., 2008) or in naturalistic samples of participants with co-occurring poor sleep and depressive symptoms. Given the limitations of these designs, the primary goal of this study was to examine the separate impact of poor sleep quality and depressive symptoms on emotion regulation.

In order to accomplish this goal, our sample consisted of groups that were preselected on the basis of their sleep and depression profiles: individuals with mainly sleep problems, individuals with mainly depressive symptoms, individuals who were high on both problems, as well as individuals who were low on both problems. This strategy provided several advantages relative to previous methodology in its ability to isolate effects. In order to represent emotion regulation (and emotion problems more broadly), self-report scales and laboratory measures were utilized. Depressive symptoms were measured using the Beck Depression Inventory II (BDI-II; Beck et al., 1996). We assessed sleep quality using the Pittsburgh Sleep Quality Index (PSQI; Buysse, 1989).

The current study addressed three main questions. (1) What are the specific contributions of poor sleep quality and depression (both separately and together) on maladaptive emotion regulation as indexed by (a) self report scales and (b) emotion regulation strategy use in the laboratory? And (2) to explore if there are there specific

components of sleep that are uniquely associated with impairment in emotion regulation? And (3) Do sleep and depression differentially relate to aspects of nonemotional functioning?

Hypotheses

Aim 1: What are the specific contributions of poor sleep quality and depression to maladaptive emotion regulation?

Hypothesis 1a: Sleep's detriment on reports of emotion regulation and specific problems (e.g. worry, rumination) are both strong and immediate (for a review, see Palmer & Alfano, 2016). Therefore, we predicted a significant effect of sleep disturbance on self-reports of emotion regulation. Additionally, we predicted that groups higher in sleep problems would score uniquely worse on deficits in emotion regulation.

Hypothesis 1b: Similarly, there is ample evidence that poor sleep produces acute deficits in functioning (e.g. quick changes in affect, Baglioni et al., 2010). Therefore, we predicted a significant impact of sleep on emotion regulation strategy use in the laboratory. Additionally, we predicted that groups with high sleep problems would perform worse on emotion regulation strategy use in the laboratory.

Aim 2 (exploratory): Additionally, if we find sleep is the key ingredient in Hypothesis 1 (i.e. groups with poor sleep are significantly worse on multiple ER scales than groups with good sleep), we aimed to explore several specific components of poor sleep quality that may be uniquely associated with impairment in emotion regulation. Although sleep quality as a construct is oftentimes referred to as a unitary concept, there are several aspects of sleep that can be impaired. Sleep quality subcomponents have been shown to differentially impact emotional functioning (e.g. O'Leary, et al., 2016; Tavernier & Willoughby, 2014). Follow up analyses would focus on three specific subcomponents of sleep quality: duration, disturbances, and quality.

Aim 3 (exploratory): Do sleep and depression differentially impact areas of nonemotional functioning? There is abundant evidence that poor sleep (whether chronicled by acute sleep loss or ongoing difficulties with sleep) has an impact on several domains, particularly cognitive and motor functioning (i.e. Pilcher & Huffcutt, 1996). Additionally, similar deficits are seen in individuals with depression (i.e. Wagner et al., 2014). As such, we included measures of physical activity to represent a nonemotional functioning domain. Because there is insufficient research in this area to make strong directional predictions, this aim was exploratory.

Method

Participants

Undergraduate students at the University of South Florida were selected for this study based on their responses to self-report assessments of sleep quality and depressive symptoms. Students signed up online via an internet-based research system within the university for a laboratory visit. All study procedures took place within the Mood and Emotion Laboratory rooms.

Participants were selected based on their performance on initial sleep and depression screener measures. Details about cut-off scores and rationale are below. Based on previous lab scores, it was estimated that around 400 participants would need to be screened to result in an N of 120 (30 for each group). Power analyses are included below as well. See Appendix A for an outline of laboratory procedures.

Key Materials

Beck Depression Inventory (BDI-II). The Beck Depression Inventory is a 21-item self-report measure that assesses depression severity. The BDI-II has previously shown good psychometric properties (Beck et al., 1996). A cut-off score of 10 (Oliver & Simmons, 1984) was utilized in this sample to split participants into "high" and a score below 8 into "low" depression severity groups, with 10 marking the minimal scoring required before a participant was classified as showing mild levels of self-reported depression symptoms. Although it is difficult to determine a cut-off that can produce separate groups, previous analyses within this population have found mean BDI scores around 9 represent a cut-off for mild symptoms. Therefore, a range of below 8 and above 10 has the greatest potential to split based on different depressive characteristics (see Appendix B). Alphas for all self-report measures are included in Table 1.

Pittsburgh Sleep Quality Index (PSQI). The PSQI is a 19-item self-report questionnaire, which assesses sleep quality over the last month. Items are rated on 0-3 scales (with 3 indicating worse functioning). Although the PSQI can be used to derive different sleep component scores (subjective sleep-quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, medicine to sleep, and daytime dysfunction), it is typical to combine these components into an overall sleep quality index (Buysse, 1989). In this sample, a cut-off score of 5 (Backhaus et al.,

2002) was used to split participants into "high" and into "low" sleep problem groups; this cut-off has been psychometrically shown to distinguish those with significant sleep problems from those with minimal sleep issues. Similarly, previous analyses within this student population have found average scores of 6. Therefore, a cutoff of 5 (in the literature cited as the cut-off score for clinical sleep problems) has the greatest potential to split this particular population based on sleep characteristics. See Appendix C.

Groups were assessed on age, gender, and income level.

Emotion Regulation Scales: Self-Report Outcome Measures

Emotion regulation (ER) refers to a variety of efforts to modify the experience and expression of emotions, including how emotions are monitored and evaluated (Gross, 1998). In examining maladaptive ER, researchers often use self-report scales that are "broadband" (e.g. overall difficulties in emotion regulation strategies) or specific problematic emotion regulation strategies (e.g. worry or rumination). Because ER is a complex construct, there is currently no obvious single measure of ER (e.g. Gross, 2014). In order to provide a more comprehensive picture of ER, we have included a broadband self-report ER measure and two specific ER strategy measures, as well as laboratory measures of emotion regulation success.

Penn State Worry Questionnaire (PSWQ). The PSWQ is designed to assess pathological worry, with questions concerning excessive worry, duration, and uncontrollability (Meyer et al., 1990) This instrument has shown high reliability and stability within large samples compared to other measures (Wuthrich, Johnco, & Knight, 2014). The use of pathological worry and its uncontrollability are widely considered to be key emotion regulatory problems, and exhibit significant relationships with anxiety and depression (Topper et al., 2014). See Appendix D.

Response Style Questionnaire (RSQ). The RSQ measures the potential to respond to low mood in a ruminative fashion. The scale has good internal consistency and predictive validity (Butler & Nolen-Hoeksema, 1994). High scores on the rumination response scale falls have been considered a problematic form of emotion regulation (Ciesla & Roberts, 2007). See Appendix E.

Difficulties in Emotion Regulation Scale (DERS). The DERS is a broadband emotion regulation scale, designed to conceptualize emotion regulation as involving awareness, understanding, and acceptance of emotions (Gratz & Roemer, 2004). Psychometric properties of the measure support its high internal consistency, construct, and predictive validity across various populations (Mitsopoulou et al., 2013; Dan-Glauser & Scherer, 2013;

Giromini et al., 2012). The DERS contains six subscales: nonacceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to emotion regulation strategies, and lack of emotional clarity, which can be averaged into total difficulties score. See Appendix F.

Emotion Regulation Scales: Strategy Success

In addition to self-reports, laboratory manipulations of emotion regulations strategies are useful, given errors in how people retrospectively report on their emotion regulation strategies (Robinson & Clore, 2002). Therefore, to assess emotion regulation in the lab, participants were instructed to watch three different movie clips meant to elicit sadness. Before each clip, participants received each of three assignments: cognitive reappraisal, suppression, or the control condition. Therefore, all participants received all three strategies (one per video) in order to assess success at each strategy. Success was defined by comparing the level of negative affect between a baseline and following use of the emotion regulation strategy. Further post-hoc analyses utilize definition of success as control versus strategy use for comparative and interpretive purposes.

The instructions are included for each type below.

Film clip

In order to induce sadness during emotion regulation tasks, films were used as stimuli to elicit negative emotion. Films have generally been shown to elicit emotions successfully and reliably (i.e. Rottenberg et al., 2007). Three 2-3 minute sad films were chosen to induce sadness and their order was counterbalanced to control for film effects. The three sad films were *Lion King*, *Return to Me*, and *Champ* that tested high on self-reported sadness in a validation study (Rottenberg et al., 2007). In order to minimize potential carryover effects, a neutral puzzle task was utilized between sad films in order to help emotional state to return to baseline.

Cognitive Reappraisal

Cognitive reappraisal involves generating a different – and more positive/neutral – interpretation of a situation in order to reduce emotional distress (Gross, 1998). In this particular instance, we instructed participants to think about the film in such a way that they would feel less distress. Reappraisal can be seen as interpreting emotional stimuli in unemotional terms (Speisman, Lazarus, Mordkoff, & Davison, 1964).

The reappraisal instructions were follows: “We will now be showing you a short film clip. It is important to us that you watch the film clip carefully, but if you find the film too distressing, just say, "stop." This time, please try to adopt a detached and unemotional attitude as you watch the film. In other words, as you watch the film clip, try to think about what you are seeing objectively, in terms of the technical aspects of the events you observe. Watch the film clip carefully, but please try to think about what you are seeing in such a way that you don't feel anything at all” (Gross, 1998).

Suppression

Suppression is another emotional regulation strategy that is generally used to inhibit emotional expression. Although there is some indication that suppression reduces subjective experience of emotion, it may be less effective than other strategies (e.g. resulting in a paradoxical increase in negative mood, Gross, 1998). Suppression generally refers to behaving in such a way that an observer would not know that a target person is experiencing emotion. Although cognitive reappraisal is generally seen as a more “adaptive” strategy within the literature, some have questioned separating emotion regulatory attempts into invariably “adaptive” or “maladaptive” categories (i.e. when context and other strategy presence may impact functioning, Nolen-Hoeksema & Aldao, 2011; Bonanno & Burton, 2013). Therefore, we included both strategies to provide a more comprehensive view of the emotion regulatory strategy spectrum.

The instructions for suppression were as follows: “We will now be showing you a short film clip. It is important to us that you watch the film clip carefully, but if you find the film too distressing, just say, "stop." This time, if you have any feelings as you watch the film clip, please try your best not to let those feelings show. In other words, as you watch the film clip, try to behave in such a way that a person watching you would not know you were feeling anything. Watch the film clip carefully, but please try to behave so that someone watching you would not know that you are feeling anything at all” (Gross, 1998).

Control condition

Participants received instructions to watch the film naturally:

“We will now be showing you a short film clip. It is important to us that you watch the film clip carefully, but if you find the film too distressing, just say, "stop." Please watch this clip and let your emotions come naturally, as you would watching it at home.”

They were not given further instructions based on emotion or behavioral response.

Emotion Regulation Manipulation Outcomes:

Experience of Emotion. Participants rated how they felt before each film (baseline), as well as reporting how they felt after. They rated their levels of sadness, happiness, disgust, as well as a number of other distractor emotion variables taken from the PANAS (see Appendix H). This subjective experience and report of emotion similarly serves as a metric for "successful" emotion regulation. Downregulation of emotion is the goal following the films; therefore, our perception of successful emotion regulation within this study is defined by change to more neutral/baseline scores (Gross, 1989). Additionally, participants completed a post-task manipulation check following each video to determine whether the appropriate emotion regulation strategy was utilized.

Manipulation Checks

In order to test whether the correct strategy was attempted during film, participants were given three questions once the film ended. The questions inquired to whether they tried not to feel anything, tried to hide emotions, or reacted normally. See Appendix L.

Other Measures

International Physical Activity Questionnaire (IPAQ). The IPAQ is a set of 4 questions that is used to obtain internationally comparable data on health-related physical activity. The scale assesses time spent during the past week in vigorous, moderate, walking, and sitting activities. Although brief, the IPAQ has achieved good psychometric properties in test-retest reliability (Brown et al., 2004), international reliability and validity in 12 countries (Craig et al., 2003), and criterion-related validity (Ekelund et al., 2006). See Appendix J.

Procedure

Participants signed up online for specific time slots and came to the lab for initial testing. Participants first completed the PSQI and the BDI, which was scored immediately by a research assistant. If the participant did not meet entry criteria, they were debriefed, awarded all earned points, and escorted out. For those who qualified, participants were included in one of four groups based on their score on the depression severity and sleep quality measures (as detailed above): high sleep-high depression, low sleep-low depression, high sleep-low depression, and low sleep-high depression.

Following initial screening, participants were seated in a comfortable chair in a room by a research assistant, who explained the remaining procedure. Participants completed a number of emotion regulation measures and manipulations, as described above. Following completion of all measures, participants were thoroughly debriefed and escorted out.

Changes to Recruiting Strategy

During data collection, three of the four groups were collected utilizing the above described preselection procedure. However, the high depression/low sleep difficulties group – proved particularly difficult to recruit. We put additional procedures in place in order to recruit these individuals. The BDI depression cutoff was reduced from 14 to 10 (as noted above), which yielded an additional 1-2 participants per semester. In an effort to further preselect for these individuals, mass pre-selection testing was utilized prior to in-person procedures. This recruiting procedure allowed for a more refined method of finding individuals within this group. Despite these additional steps, it was not possible to identify additional persons with high depression/low sleep difficulties and analyses were run on a reduced sample size, N=10 (this issue is discussed in detail below).

Power Analysis

The above design involves testing the effect of sleep and depression represented by pre-selected groups (high depression-high sleep, high depression-low sleep, low depression-high sleep, and low depression-low sleep) on specific self-report and behavioral indices of emotion regulation. In order to be able to detect a small-moderate effect (similar to the impact of sleep quality on emotion regulation and depressive symptoms on emotion regulation alone; e.g. *r*'s~.20, Mauss, Troy, & LeBourgeois, 2013), an estimated sample size of 100-120 would be desirable (25-30 participants per group). At minimum, in order to detect a large effect, power analyses suggest that groups would need to contain 10-15 participants.

During data collection, we collected 30-35 participants for three of the four groups. One group – high depression/low sleep problems – proved impossible to recruit and contained 10 participants total at the termination of data collection. Further details are included above regarding revision to recruiting procedures and below in an extensive discussion of the impact of power on this study.

Hypothesis Testing

For all aims, the main target analyses focused on the main effects of sleep and depression on various indices (e.g. ER trait measures, ER strategies, nonemotion domain). Specifically, all analyses tested the main effect (sleep, depression) and interaction effect (sleep by depression) on these key outcome variables.

For hypothesis 1a, a 2x2 factorial design was utilized with sleep and depression as independent variables and all three emotion regulation indices as outcome variables. Our prediction was that sleep would emerge as the primary main effect for all three indices. Any significant interaction terms were further unpacked.

For hypothesis 1b, a 2x2x2 repeated measures design was utilized with time, sleep, and depression as independent variables and behavioral emotion regulation (negative affect) as outcome variables. These analyses were done separately on each strategy use type for theoretical basis and clarity in interpretation purposes. Our prediction was that sleep by time would emerge as a significant effect on behavioral measures.

Hypothesis 2 was conditional based on Hypothesis 1 revealing sleep as a main effect in multiple measures of trait and behavioral emotion regulation measures in stated directions. In this case, further analyses would focus on specific sleep component scores (e.g. sleep quality, Tavernier & Willoughby, 2014) rather than on sleep quality as a whole. However, as Hypothesis 1 was not fulfilled, no further analyses were completed for Hypothesis 2.

For Hypothesis 3 to test domain specificity, the same 2x2 factorial design was utilized with physical activity as the dependent variable. Significant main effects and interactions would be unpacked. Additional variables (e.g. demographics) were examined for differences across groups. Manipulation checks on key experimental variables were conducted as well.

Results

Demographics and Clinical Descriptive Statistics

The sample consisted of 110 participants recruited via an online system at a southeastern university. A total of 316 individuals were initially screened. Group 1 (high depression, high sleep problems) contained 37 participants; Group 2 (low depression, high sleep problems) contained 30; Group 3 (high depression, low sleep problems) contained 10 participants; and Group 4 (low depression, low sleep problems) contained 33 participants. Most participants were young adults in the age range of 18-22 (85% of the sample). The ethnic composition of the sample was similar to those previously reported within the university (51% white, 20% Hispanic, 15% Black, 14% other). Sample means and other characteristics (without sorting by group) are available in Table 1. The final sample was characteristic of previous college student samples within this laboratory including a mean depression score of 10.86 (*SD* = 10.54) and mean sleep problems score of 6.57 (*SD* = 3.17).

Correlations among key variables are included in Table 2. Additional descriptive statistics for the sample by group are displayed in Table 3. Group differences on measures of sleep and depression were as expected given the preselection criteria. Age, gender, and ethnicity were tested as potential variables impacting dependent variables, but none were significant (p's all >.05). As such, demographic variables are not included in the main analyses.

Aim 1a: Impact of Group on ER: Self-Report

In order to assess the separate impact of sleep and depression, a 2x2 factorial design was utilized in which factors sleep (high/low) and depression(high/low) were considered, as well as their interaction. Analyses were run on all self-reported emotion regulation indices as outcome variables.

Depression had a significant main effect across all measures on self-reported emotion regulation. Specifically, for difficulties in emotion regulation, the main effect of depression was significant, $F(1, 103) = 178.00$, $p = .048$. Individuals high in depression ($M = 101.235$) experienced greater difficulties with emotion regulation than individuals low in depression ($M = 78.008$). However, contrary to our hypothesis, neither the main effect of sleep, $F(1, 103) = 8.96$, $p = .205$, nor the interaction effect of sleep and depression, $F(1, 103) = 0.44$, $p = .5$, were significant. Similar results were observed using worry as the outcome. There was a main effect of depression, $F(1,$

104) = 545.54, p = .027, but no effects of sleep $F(1, 104) = 45.00$, $p = .094$, or a sleep x depression interaction, $F(1, 104) = 0.09$, $p = .769$. As with difficulties in emotion regulation, individuals high in depression ($M = 61.875$) experienced more worry than individuals low in depression ($M = 44.452$). In both cases, higher depressive groups reported worse emotion regulatory outcomes than their low depressive counterparts. There was no effect of sleep.

In contrast, when predicting response styles/rumination, a main effect was found for both depression, $F(1, 101) = 15371.88$, $p = .005$, and sleep, $F(1, 101) = 4138.33$, $p = .010$. The sleep x depression interaction, however, was not significant, $F(1, 101) = 0.00$, $p = .955$. For rumination, as with other emotion regulatory problems, higher depressive groups reported worse response styles ($M = 51.447$) than those with low depression ($M = 34.947$); additionally, groups with worse sleep problems reported increased rumination ($M = 47.478$) compared to their healthier sleep counterparts ($M = 38.917$). See Tables 4-6 for complete statistical results. Figures 1 through 3 present graphical representation of results.

Taken together, these findings denote a pattern in which the main effect of depression was consistent across self-reported emotion regulation strategies. By contrast, the main effect of sleep was less consistent and depended on the type of emotion regulation.

Aim 1b: Impact of Group on ER: Laboratory Success

In order to assess the separate impact of sleep and depression on the effectiveness of laboratory emotion regulation strategy use, a 2x2x2 repeated measures factorial design was utilized in which assessment point (baseline/post-strategy; within subjects), sleep (high/low; between subjects) and depression (high/low; between subjects) were considered. Of interest were the interaction effects of sleep and depression with assessment point (i.e., assessment point x sleep and assessment point x depression), as well as the three-way interaction across all factors (i.e., assessment point x sleep x depression). The outcome measure was self-reported negative affect. Each strategy was analyzed separately to determine the impact of depression, sleep, and their interaction on the effectiveness of particular emotion regulation techniques.

For suppression, there were significant assessment point x depression, $F(1, 106) = 4.36$, $p = .039$, $\eta p^2 = .04$, and assessment point x sleep interactions, $F(1, 106) = 3.95$, $p = .049$, $\eta p^2 = .036$. Post-hoc analyses of assessment point at individual levels of depression revealed a significant effect at high levels of depression, $F(1, 45), = 16.569$, $p = .000$, but not low levels of depression, $F(1, 63) = .026$, $p = .872$. Contrary to our hypothesis, individuals with

higher self-reported depression symptoms exhibited greater reductions in negative affect from baseline to post-strategy than did those low in depression. Unexpectedly, individuals with high sleep problems also exhibited the same effect: a significant effect at high levels of sleep problems, $F(1, 67) = 9.534$, $p = .003$, but not low levels of sleep problems $F(1, 41) = .111$, $p = .740$. However, there was no significant assessment point x depression x sleep interaction, $F(1, 106) = 2.54$, $p = .114$.

For cognitive reappraisal, there was a significant assessment point x depression, $F(1, 106) = 5.69$, $p = .019$, $\eta p^2 = .051$, and assessment point x sleep interaction, $F(1, 106) = 4.45$, $p = .037$, $\eta p^2 = .040$. Individuals exhibited a similar pattern seen in suppression: post-hoc analyses of assessment point at individual levels of depression revealed a significant effect at high levels of depression, $F(1, 45), = 26.834$, $p = .000$, but not low levels of depression, $F(1, 63) = .795$, $p = .376$. Additionally, post-hoc analyses unpacked a significant effect at high levels of sleep problems, $F(1, 67) = 19.858$, $p = .000$, but not low levels of sleep problems $F(1, 41) = .438$, $p = .512$. Those with high depressive symptoms exhibited greater reductions in negative affect and individuals with high sleep problems exhibited greater reductions in negative affect. It is also notable that for depression effects, while high depression groups were associated with more benefit from strategy use, negative affect still remained higher at post-strategy use than low depression groups (means are displayed in figures 4-7). This was not found for the sleep effect (i.e. post affect decreased to levels similar to low sleep problem counterparts).

In the uninstructed control condition, there was a significant assessment point x depression effect, $F(1, 106) = 7.83$, $p = .006$, $\eta p^2 = .069$. Post-hoc analyses of assessment point at individual levels of depression revealed a significant effect at high levels of depression, $F(1, 45), = 17.910$, $p = .000$, but not low levels of depression, $F(1, 63) = 1.149$, $p = .288$. The effect of assessment point x sleep, $F(1, 106) = 2.98$, $p = .087$ was observed at a trend level. Additionally, an interaction between sleep, depression, and assessment point was observed at a trend level, $F(1, 106) = 2.98$, $p = .087$. Patterns were similar to other behavioral strategies in that high depression groups benefited significantly more from strategy use than low depressive groups. See tables 7-9 and figures 4-7 for complete results.

Impact of Group on ER: Laboratory Success – Post Hoc Analyses

Because our initial results were strikingly counterintuitive, additional analyses were run in order to further understand the impact of sleep and depression on ER success. Although we originally defined ER success as baseline to post-strategy reduction in negative affect, an alternative operationalization of ER success may result

from a comparison of affect in the sad film in the instructed control condition with those of the emotion regulation strategy conditions. As such, a 2x2x2 repeated measures factorial design was utilized in which alternate laboratory ER instruction (control vs. suppression or control vs. cognitive reappraisal; within subjects), sleep (high problems/low problems; between subjects) and depression (high symptoms/low symptoms; between subjects) were considered. Of interest were the two-way interaction effects of sleep and depression with ER instruction type (i.e., ER instruction type x sleep and ER instruction type x depression), as well as the three-way interaction across all factors (i.e., ER instruction type x sleep x depression). The outcome measure was self-reported negative affect after strategy use.

There was no significant impact of ER instruction type, F (1, 106) = .005, p = .941, ηp^2= .00 when comparing control and suppression conditions. Subsequently, there were no significant ER instruction type x depression interaction, F (1, 106) = 2.259, p = .136, ηp^2 = .021, or ER instruction type x sleep interactions, F (1, 106) = .005, p = .941, ηp^2= .00. Additionally, there was no significant ER instruction type x depression x sleep interaction, F (1, 106) = .244, p = .622.

By contrast, there was a significant main effect of ER instruction type when comparing the control and cognitive reappraisal conditions, F (1, 106) = 6.465, p = .012, ηp^2 = .057. Use of cognitive reappraisal resulted in significantly lower negative affect scores following strategy use (M = 18.03, SD = .66) versus following no strategy use (M = 19.16, SD = .68). However, there were no significant ER instruction x depression, F (1, 106) = 1.147, p = .287, ηp^2 = .011, or ER instruction x sleep interactions, F (1, 106) = .151, p = .698, ηp^2 = .001. Finally, there was no significant assessment point x depression x sleep interaction, F (1, 106) = .239, p = .626, ηp^2 = .002.

Aim 2

As Aim 2 was contingent on multiple sleep effects, further analyses on sleep quality subscores were not run.

Aim 3: Impact of Group on non-ER: Physical Activity

In order to assess the separate impact of sleep and depression on a non-emotional domain, a 2x2 factorial design was utilized in which factors sleep (high/low) and depression(high/low) were evaluated, as well as their interaction on all self-reported physical activity as the outcome variable.

No significant main or interaction effects were observed for physical activity. Neither depression nor sleep yielded a main effect on physical activity, $F(1, 103) = 11.21$, $p = .185$ for sleep; $F(1, 103) = 21.46$, $p = .135$ for depression). Similarly, the interaction between sleep and depression was nonsignificant, $F(1, 103) = .105$, $p = .746$.

Note about Power

Given the discrepancies between the sample size expected versus the one collected for the high depression/low sleep problems group, further discussion on power and the implications of these results is warranted. In the power analysis above, we discussed the potential to detect a small-moderate effect (similar to the impact of sleep quality on emotion regulation and depressive symptoms on emotion regulation alone; e.g. *r*'s~.20, Mauss, Troy, & LeBourgeois, 2013) at an estimated 25 participants for group, which three of the four groups exceeded. Furthermore, in order to detect a large effect, power analyses suggest that groups would need to contain 10-15 participants (~13 for most calculators). The high depression/low sleep problems group concluded with 10 participants; as such, a large effect may be detected with this group. However, other effects may be more difficult to detect.

A more thorough discussion of conceptual and practical implications is presented below within the discussion section.

Discussion

Although sleep and depression are both strongly associated with deficits in emotional functioning (Durmer & Dinges, 2005; van der Helm & Walker, 2010), their independent and conjoint impact on emotional functioning is not well established. This study was the first to our knowledge to examine the separate impact of poor sleep quality and depressive symptoms on emotion regulation via groups preselected on their sleep and depression profiles: mainly sleep problems (N = 30), mainly depressive symptoms (N = 10), high on both problems (N = 37), and low on both problems (N = 33). While depression yielded significant effects on indices of both laboratory and self-reported emotion regulation, the patterns that emerged were not aligned with our hypotheses, which predicted that sleep would emerge as a significant main effect for all areas of emotion regulation. Specifically, self-report data suggested that trait emotion regulation deficits were associated with higher depression scores, and impaired sleep quality was associated with a higher rumination response style only. Sleep and depression were both associated with the degree of instructed emotion regulation success in the laboratory; however, the direction of these effects was contrary to what we predicted. Specifically, in our primary analyses, persons with higher levels of depression symptoms and greater impairment of sleep quality obtained greater benefit of emotion regulation strategy use.

As worldwide sleep problems increase (e.g. Matricciani et al., 2012) and the burden of depression escalates (e.g. Ustin, 2004), it becomes all the more important to establish how each of these facts might respectively contribute to problems in emotional functioning. In previous work, sleep has been shown to amplify negative emotions and affective volatility (Walker & van der Helm, 2009**)**. Sleep and depressive symptoms are so highly correlated (e.g. Agargun, Kara, & Solmaz, 1997) that it has been difficult to disentangle their impact on emotion. Theories have suggested that deficits in emotion that result from sleep impairment contribute to later depression (e.g., Gruber & Cassoff, 2014) or that sleep may manifest as a prodromal symptom of depression. Competing theories provide a motivator to delineate pathways and specific effects of both sleep problems and depression on emotion regulation.

Interestingly, the pattern that emerged in this study was contradictory to our hypotheses. Because previous studies of emotion regulation have shown a consistent role for sleep above and beyond depression (e.g. that sleep predicts emotional reactivity in both healthy and mood disordered individuals, O'Leary et al., 2016), one key

question is why sleep emerged as a main effect for only one of the three trait emotion regulation scales, rumination. One potential explanation for the association between rumination and impaired sleep quality is their common emphasis on past events; that is, individuals who have these characteristics may be especially prone to perseverate on the past. While worry tends to involve future-focused orientation, rumination is associated with low mood and past causes (Nolen-Hoeksema & Morrow, 1991). Recent findings have shown a robust relationship between poor sleep and symptom-focused rumination, demonstrating a common pathological negative past focus in both insomnia and rumination (Carney et al., 2006). Although we favor a substantive interpretation, it is also possible that high correlations between depression measures and emotion regulation measures suggest potential content overlap rather than common processes. One need in future work is to suggest better ways to assess depression symptoms apart from emotion regulation problems.

Our laboratory findings displayed a pattern in which those with high depression and/or high sleep problems benefited more from emotion regulatory instructions than their low symptom counterparts. There are several possible explanations of this finding. Although this was unexpected, one explanation specific to depression can be gleaned from a recent review of depression and ER, wherein depressed individuals successfully utilize strategies when instructed. The study suggested that ER skills are as successfully utilized by depressed individuals as by healthy controls when clearly directed (Liu & Thompson, 2017). Indeed, a study on depression and instructed strategy use found that for one of three strategies –distraction – depressed individuals were more effective in reducing negative affect than a never depressed group (Smoski, LaBar, & Steffens, 2014). Another potential explanation for why depressed persons appear to benefit focuses on regression to the mean. Depressed individuals begin their laboratory paradigm at higher levels of negative affect, providing the opportunity to regress to lower levels following any stimuli. Albeit speculative, the laboratory stimuli may serve as a distraction rather than a negative induction and therefore lead to decreased negative affect. Further, there is some suggestion that the sad stimuli did not have the intended effects, as seen through low sleep problems/low depression graphical representations. Potentially, a weak elicitation at best may have been evoked during laboratory manipulation, which would make finding effects difficult.

One methodological caution is that our findings were not stable, and appear to hinge upon how emotion regulation success is defined. Importantly, in post hoc analyses, emotion regulation differences largely disappeared when the instructed control condition was used as the point of comparison rather than baseline. Specifically, sleep

and depression were no longer significant predictors. The sensitivity of main effects to operationalization and methodology lends to additional questioning regarding the specificity of impact on emotion regulation within these groups. The fact that operational differences may inform what we currently know about emotion regulation is especially notable in this study.

Given what we know about the difficulty operationalizing emotion regulation, there are some potential explanations involving timing as well as coding of measurement. Significantly, there has been recent suggestion that timing of collecting study data (e.g. affect) is essential in assessing the impacts of sleep on emotion. In their review, Watling and colleagues (2017) describe the differences between null and significant findings on emotion following sleep loss. They posit that poor sleep impacts emotion by increasing the likelihood of a negative affective reaction in immediate response to an aversive stimulus; however, if given time to recover from this initial emotional reaction (i.e., in an instructed regulatory design, such as the one used in the current study), the impact of poor sleep fades. Thus, the experimental methods utilized in the current investigation may not have been adequate to capture this immediate impact of poor sleep on affective responding, as participants were given time to recover from the affect-inducing stimulus before their negative affect was assessed. In other words, we did not assess their immediate emotional reaction, but their delayed emotional reaction following implementation of an emotion regulation strategy. This speaks to the broader point mentioned above: our inferences about ER success often depend on how successful strategy use is operationalized.

Lastly, our non-emotion variable of physical activity was unrelated to key variables. Although both sleep and depression have been associated with physical activity, it is possible that would be easier to detect these effects in a clinical, rather than college sample. Additionally, there have been difficulties establishing the link between sleep and physical activity; for example, while increasing physical activity has been shown to improve sleep, improving sleep has not resulted in increased levels of physical activity (Kline, 2014). Additionally, there is some suggestion that only certain types of physical activity are associated with mental health (Joshi et al., 2016); as such, our general measure of physical activity may not capture the specificity involved in this relationship.

One potential limitation of this study is the modest sample size, particularly for the group with high depressive symptoms and low sleep problems. Despite extensive outreach for data collection (including screening large amounts of people, as well as introducing pre-screening to collect specific subsamples), the number of collected participants for this group remained low. Power notwithstanding, a number of significant effects involving

this group were detected, suggesting that sample size limitations may not have had a great impact. Additionally, we found that this group of college students reported high mean sleep difficulties ($M = 6.57$, with a clinical cutoff for significant sleep problems at 5). This high baseline level of poor sleep quality may provide some potential explanation for the lack of sleep effects.

Perhaps as interesting and worthy of comment was the difficulty in identifying and recruiting a sample of individuals high in depression but low in sleep problems. One potential explanation for the difficulty in obtaining this group is that sleep and depression problems are simply fused; for example, the underlying neurobiological connection between sleep and depression, particularly involving the serotonergic system (Adrien, 2002), may be too strong to allow identification of individuals who have depression but no sleep problems. If the underlying systems are fused, phenotypic presentation will reveal that one rarely occurs without the other. The variety of common neurobiological findings, such as EEG abnormalities and endocrine disturbances, found in both sleep restriction and depression, would seem to support this account (Spiegel, Leproult, Van Cauter, 1999). Importantly, insomnia often emerges prior to depression (Harvey, 2001), whereas depression typically occurs concurrently with poor sleep (e.g. early depressive symptoms do not predict later sleep problems, Alvaro et al., 2013). Although the relationship between sleep and depression is complex and likely bi-directional (Kahn, Sheppes, & Sadeh, 2013), it is fair to say that sleep problems often precede, and can occur in isolation of, depression. Thus, we were able to find a group of individuals with poor sleep and low depressive symptoms, but struggled to collect a group of participants with high depressive symptoms and good sleep.

This tie between sleep and depression, certainly known prior to this study, appears to be stronger than initially anticipated. In fact, our results suggest that the two concepts are near impossible to separate in a normative sample. Findings in this vein give rise to questions about what it is about depression that makes sleep problems so likely to co-occur. While this question lies beyond the scope of our current inquiry, it is the central question raised within this unique sample. It appears clear that depression and sleep are separate constructs (by content and measurement); however, their strong statistical tie speaks to the role of sleep as a fundamental characterological trait of depression, despite it being outside of the fundamental descriptive indices of depression. Further discussion along this line would be purely speculative; regardless, it remains curious that the two are so strongly tied and lends to the necessity of further exploration.

Finally, there are a few additional limitations that should be considered when evaluating this study. As with any creation of numerical cutoffs for group comparisons, there is the risk of losing the continuous nature of such symptoms – individuals who fall "in the middle" may provide valuable information. The difference between high and low symptoms for group cutoffs, though based on empirical evidence, can be somewhat artificial. Similarly, cutoffs were created utilizing self-reported symptoms, which can be less diagnostically accurate due to their shortened nature and lack of clinical interview.

Findings from the current study suggest important new avenues for investigation in future research. In the present sample, college students represent a population where difficulties with sleep and depression are commonplace. This is most notable in both sleep quality overall scores and overall depression scores, in which means (unseparated by group) of this sample were quite high on both sleep and depressive problems, suggesting the sample should not be regarded as a "healthy." Given the pathological nature of these comparisons, extending these findings to a clinical population with diagnostic subsamples would be a logical next step. Most specifically, individuals with unique presentations, such as high depressive symptoms and low sleep problems, are a theoretically interesting group by which to potentially draw information from regarding emotion regulation. This group may be easier to access within a clinical population, where rare symptom presentations – such as those in treatment for insomnia with residual depression symptoms – are more likely. Thus, replication of the current study in a clinical sample is an important goal for future research.

Although we found few significant effects of impaired sleep quality on trait emotion regulation variables, future studies can build upon questions identified in this study, such as timing of assessment and its impact on results. An experience sampling method specifically would enable researchers to time lock instances of depressed mood and days after poor sleep to specific events involving self-reported emotion regulation. Of course, self-reported and actual emotion regulation differ in empirical and theoretical ways, necessitating further laboratory studies. Future experimental paradigms could benefit from a within-study design and assessment points monitored more frequently throughout the emotion regulation process. Importantly, further research focus on unique clinical groups may provide a more lucrative look inside the sleep-depression link.

In summary, our study was the first to selectively sample groups based on their sleep and depression scores in order to compare across relatively pure categories. We found evidence that while sleep is not the primary ingredient driving impact on emotion regulation, it may selectively impact certain emotion regulatory processes,

particularly rumination. In order to explore this relationship further, additional investigation and data collection within clinical populations, particularly among individuals with low sleep problems but high depressive symptoms, would be highly valuable.

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Appendix A: Main Figures and Tables

Table 1. Mean and standard deviations of key variables for entire sample.

	Mean	Standard Deviation	N	α
PSQI	6.57	3.17	110	N/A
BDI	10.86	10.54	110	.945
DERS	87.92	17.08	107	.855
PSWQ	52.56	14.98	108	.944
RSQ	42.89	14.64	105	.940
PA	26.86	7.96	110	.900
NA	20.55	7.55	110	.862
IPAQ	3863.88	3055.81	107	N/A

*PSQI = Pittsburgh Sleep Quality Index; BDI = Beck Depression Inventory; DERS = Difficulties in Emotion Regulation Scale; PSWQ = Penn State Worry Questionnaire; RSQ = Rumination Style Questionnaire; PA = Positive Affect; NA = Negative Affect; IPAQ = International Physical Activity Questionnaire
*N's varied slightly due to response issues and computational impossibilities

Table 2. Correlations among key variables.

	BDI	PSQI	PA	NA	DERS	RSQ	PSWQ
BDI	1	.61**	-.48**	.65**	.75**	.66**	.65**
PSQI	.61**	1	-.39**	.44**	.45**	.52**	.41**
PA	-.48**	-.39**	1	-.22*	-.50**	-.38**	-.40**
NA	.65**	.44**	-.22*	1	.67**	.66**	.56**
DERS	.75**	.45**	-.45**	.66**	1	.73**	.72**
RSQ	.66**	.52**	-.38**	.66**	.73**	1	.63**
PSWQ	.65**	.41**	-.40**	.56**	.72**	.63**	1

**PSQI = Pittsburgh Sleep Quality Index; BDI = Beck Depression Inventory; DERS = Difficulties in Emotion Regulation Scale; PSWQ = Penn State Worry Questionnaire; RSQ = Rumination Style Questionnaire; PA = Positive Affect; NA = Negative Affect.

Table 3. Means by group.

	PSQI	*BDI*	*DERS*	*PSWQ*	*RSQ*	*PA*	*NA*	*IPAQ*
High Sleep Problems/High Depression	9.49	21.81	102.91	64.35	56.22	22.73	26.41	4234.17
High Sleep problems/Low Depression	7.13	4.47	80.89	46.69	37.83	26.43	16.63	2859.26
Low Sleep problems/High Depression	4.10	15.50	99.50	59.00	47.10	24.70	21.30	4791.17
Low Sleep problems/low Depression	3.55	3.00	74.67	42.25	31.23	32.55	17.30	4089.88

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Table 4. Results of 2x2 factorial design on difficulties in emotion regulation.

	SS	*Df*	*F*	*Sig*
Intercept	666142.02	1	57.00	.071
Depression	11186.57	1	178.00	.048
Sleep	563.15	1	8.96	.205
*Sleep*Depression*	62.85	1	.44	.511

Table 5. Results of 2x2 factorial design on worry.

	SS	*Df*	*F*	*Sig*
Intercept	235168.12	1	34.46	.084
Depression	6314.80	1	545.54	.027
Sleep	520.88	1	45.00	.094
*Sleep*Depression*	11.58	1	.09	.769

Table 6. Results of 2x2 factorial design on response styles.

	SS	*Df*	*F*	*Sig*
Intercept	153104.95	1	21.60	.072
Depression	5584.35	1	15371.88	.005
Sleep	1503.39	1	4138.33	.010
*Sleep*Depression*	.36	1	.003	.955

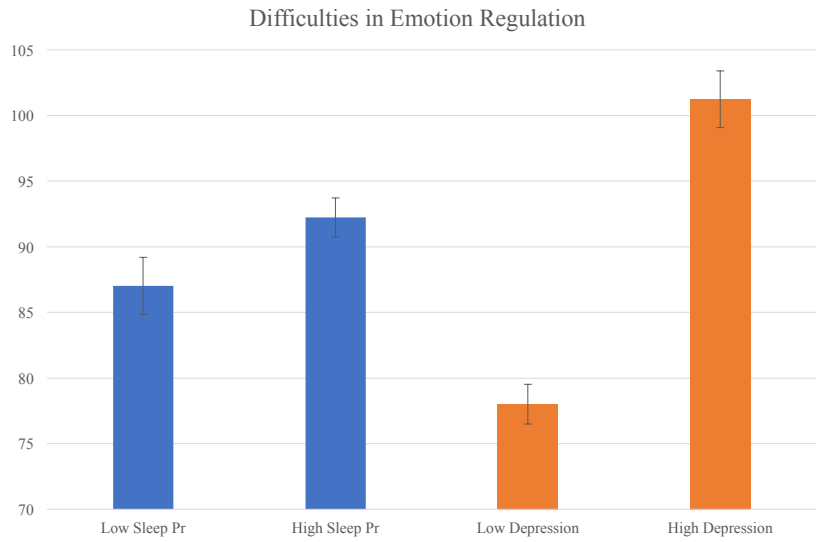

Figure 1. Main effects of sleep and depression difficulties in emotion regulation.

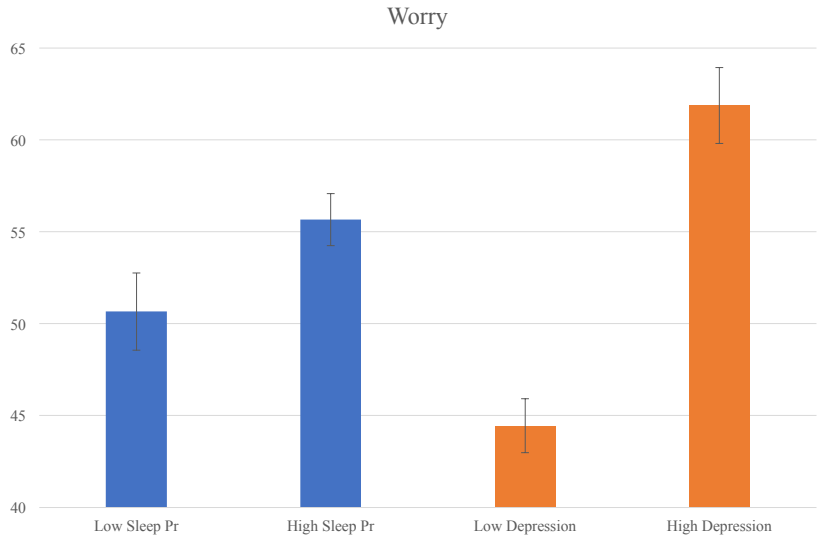

Figure 2. Main effects of sleep and depression on worry.

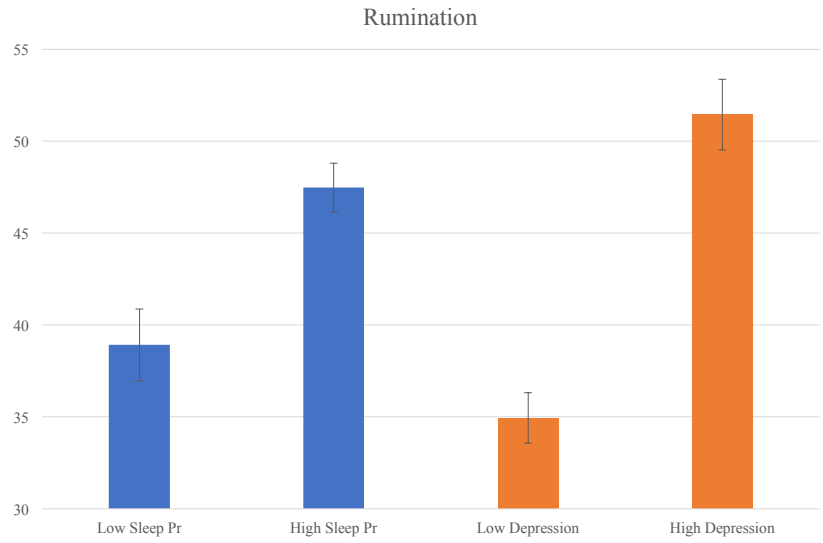

Figure 3. Main effects of sleep and depression on rumination.

Table 7. Results of 2x2x2 repeated measures factorial design on behavioral emotion regulation following suppression strategy use.

	SS	*df*	*F*	*Sig*
Time	77.73	1	3.66	.058
*Time*Depression*	92.68	1	4.36	.039
*Time*Sleep*	83.93	1	3.95	.049
*Time*Sleep*Depression*	53.84	1	2.54	.114

Table 8. Results of 2x2 factorial design on behavioral emotion regulation following cognitive reappraisal strategy use.

	SS	*Df*	*F*	*Sig*
Time	253.55	1	11.18	.001
*Time*Depression*	129.14	1	5.69	.019
*Time*Sleep*	100.94	1	4.45	.037
*Time*Sleep*Depression*	56.46	1	2.49	.118

Table 9. Results of 2x2 factorial design on behavioral emotion regulation following no instructed strategy use.

	SS	*Df*	*F*	*Sig*
Time	73.62	1	2.76	.100
*Time*Depression*	208.99	1	7.83	.006
*Time*Sleep*	79.65	1	2.98	.087
*Time*Sleep*Depression*	79.65	1	2.98	.087

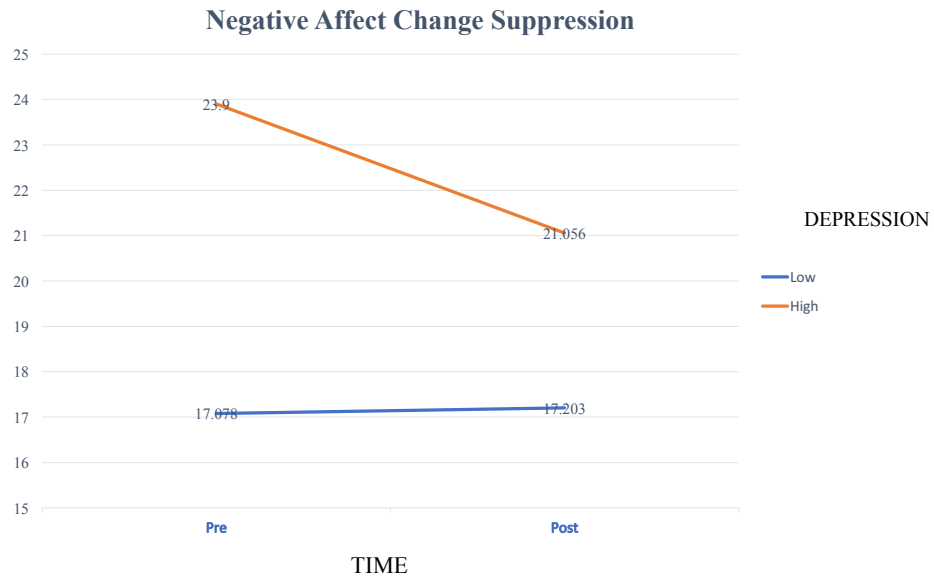

Figure 4. Effects of time x depression on negative affect following suppression strategy use.

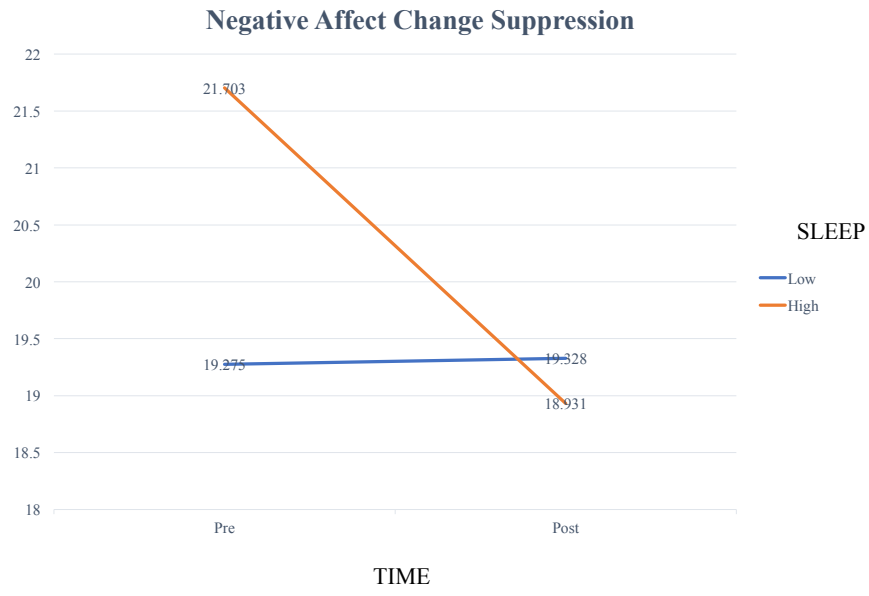

Figure 5. Effects of time x sleep on negative affect following suppression strategy use.

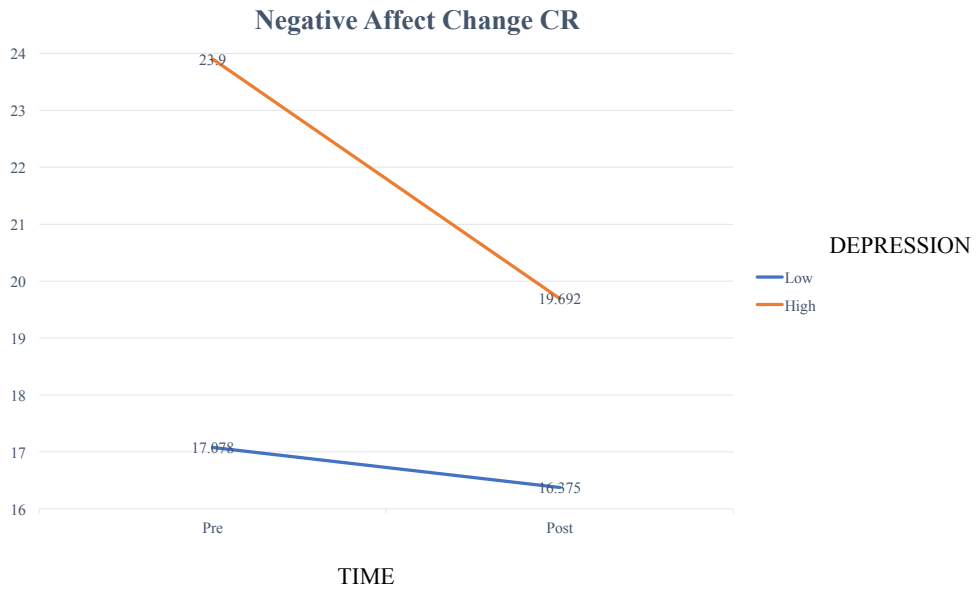

Figure 6. Effects of time x depression on negative affect following cognitive reappraisal strategy use.

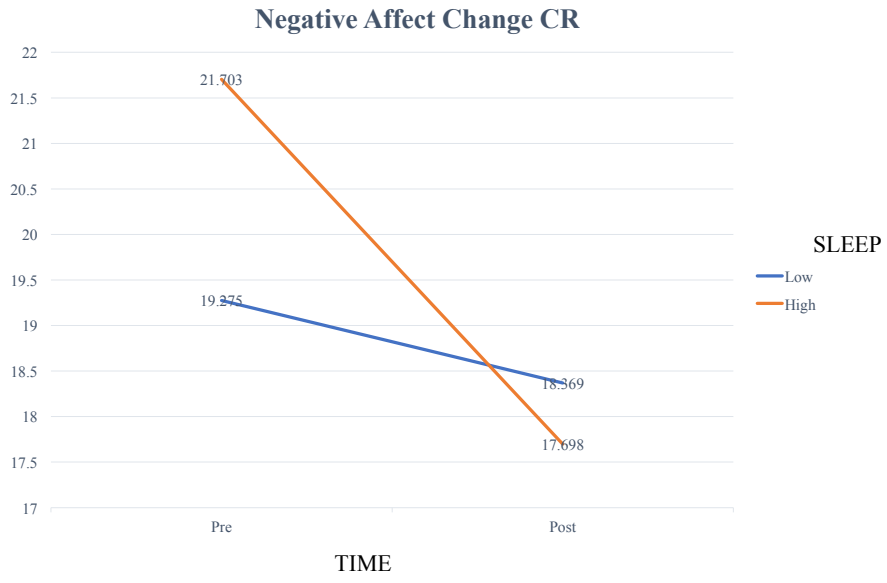

Figure 7. Effects of time x sleep on negative affect following cognitive reappraisal strategy use.

Appendix B: IRB Approval Letter.

5/12/2017

Kimberly O'Leary, B.S. Psychology
4202 East Fowler Ave. Tampa, FL 33620

RE: **Full Board Approval for Initial Review**
IRB#: Pro00029372
Title: Disentangling the impact of poor sleep from depressive symptoms on emotion regulation

Study Approval Period: 4/21/2017 to 4/21/2018

Dear Ms. O'Leary:

On 4/21/2017, the Institutional Review Board (IRB) reviewed and **APPROVED** the above application and all documents contained within, including those outlined below.

Approved Item(s): Protocol Document(s): IRB Protocol 050817

Consent/Assent Document(s)*:
Informed Consent Form Version 1 3312017.pdf

*Please use only the official IRB stamped informed consent/assent document(s) found under the "Attachments" tab. Please note, these consent/assent documents are valid until the consent document is amended and approved.

As the principal investigator of this study, it is your responsibility to conduct this study in accordance with IRB policies and procedures and as approved by the IRB. Any changes to the approved research must be submitted to the IRB for review and approval via an amendment. Additionally, all unanticipated problems must be reported to the USF IRB within five (5) calendar days.

We appreciate your dedication to the ethical conduct of human subject research at the University of South Florida and your continued commitment to human research protections. If you have any questions regarding this matter, please call 813-974-5638. Sincerely,

Kristen Salomon, Ph.D., Vice Chairperson USF Institutional Review Board