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# Does emotional processing mediate the link between disordered sleep and depression?

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Does Emotional Processing Mediate the Link Between Disordered Sleep and Depression?

by

Kimberly O'Leary

A thesis submitted in partial fulfillment  
of the requirements for the degree of  
Master of Arts  
Department of Psychology  
College of Arts and Sciences  
University of South Florida

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## **ABSTRACT**

Disordered sleep is strongly linked to depression, but reasons for this are not well understood. One possibility is that this link is partially explained by deficits in the emotional processing system. This model is substantiated based on the strong link between sleep and emotions, as well as ties between affect and depression. Therefore, this study tested whether various emotional and non-emotional deficits mediated the link between poor sleep quality and depression. Two hundred undergraduate students were recruited via an online university system. Participants completed self-report scales of depression, sleep quality, emotion recognition, and affective response to pre-tested pleasant or unpleasant stimuli. Mediation models were tested for viable emotion and non-emotion mediators, as well as using other mediators as covariates. The indirect effect for all models was tested using bootstrapping. Only affective response to unpleasant stimuli emerged as a significant mediator of the relationship between sleep quality and depression and accounted for 5% of the variance in that relationship; it remained a mediator after controlling for non-emotion related mediators. Recently, sleep problems have gained attention due to serious consequences for public health, including a strong association with psychological disorders. This study was a first step in testing pathways by which disordered sleep leads to increases in depression symptoms. In our sample, blunted emotional responding to unpleasant images partially accounted for the link seen between sleep and depression. Future research may aim to extend the study of process and pathway-related models, particularly in the realm of emotional responding in the relationship between sleep and depression.

## **DOES EMOTIONAL PROCESSING MEDIATE THE LINK BETWEEN DISORDERED SLEEP AND DEPRESSION?**

Mood disorders are debilitating conditions with high and increasing prevalence rates (Klerman & Weissman, 1989). Major Depressive Disorder (MDD) is one of the most common mental disorders in United States adults, with a 12-month prevalence rate of 6.7%. Compared to adults 60 and older, 18-29 year olds are now 70% more likely to experience depression within their lifetime and 200% more likely to have an episode in the last year (Kessler et al., 2003). However, only just over half of those with MDD receive treatment and only a fraction of those treated is treated adequately (Wang et al., 2005). Currently, MDD is the fourth leading cause of disease burden worldwide (Ustun, 2004) and it is known to contribute to other health problems (e.g., Raposa et al., 2014).

One critically important problem that is associated with depression is disordered sleep. Although problematic sleep patterns are associated with multiple psychiatric problems (e.g., internalizing problems, Touchette et al., 2012; suicidal thoughts and anhedonia; Urrila et al., 2012), sleep complaints have a particularly strong relationship to depressive disorders (Alvaro, Roberts, & Harris, 2013). In fact, a growing body of research has examined the relationship(s) between depression and sleep problems.

### **Link between Mood Disorders and Disordered Sleep**

There has long been discussion of the correlation between sleep complaints and depression (e.g., longitudinal studies in the 80's; Rodin, McAvay, & Timko, 1988). This

discussion is natural, especially since sleep disturbance is included as one symptom in all diagnostic measures of depression. Although measured by a single question (usually assessed as insomnia or hypersomnia), sleep seems closely tied to depression on many levels. In fact, during the creation of the Pittsburgh Sleep Quality Index (PSQI), authors of the survey used depressed inpatients as one of their two groups labeled as “poor” sleepers – the other group being those with sleep disorders (Buysse et al., 1989). Since then, many studies have observed sleep problems in mood-disordered groups worldwide. Adolescents in Finland diagnosed with MDD also reported nonrestorative sleep (69%) and insomnia (51%) (Urrila et al., 2012). A study on psychiatric inpatients found that poor sleep quality and more depressive symptoms were both associated with a diagnosis of depression (Selvi, 2010). Similarly, one study found suicidal depressive inpatients had significantly higher scores of subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, and global PSQI scores than other inpatients (Agargun, Kara, & Solmaz, 1997). In a comprehensive review that included 205 articles, the authors concluded that 90% of depressed patients complain about sleep quality (Tsunno, Besset, & Ritchie, 2005).

### **Temporal Relationships**

Depressive symptoms and sleep problems are highly correlated. Recently, researchers have begun to examine why these variables are so strongly correlated. Given the potential for third variables, determining causality has also become imperative, not the least because of the implications for treatment. One review of longitudinal studies assessed the effect that early childhood sleep problems have on the children later in life.



They found that childhood sleep problems put children at 4 times the risk for internalizing problems at an 18 year follow up (Touchette et al., 2012). Another systematic review of longitudinal studies found that childhood sleep problems predicted later depression – but not the other way around (Alvaro, Roberts, & Harris, 2013). Although research thus far suggests disordered sleep generally arises before depression, the responsible pathways have not been established.

### **Pathways in this Relationship**

Despite efforts listed above, the mechanisms by which sleep disturbances produce depression are not yet clear. Disordered sleep, as it presents, may be a trigger for future depression or may simply be a prodromal symptom or warning sign of an emerging disorder. Either way, it is important to understand pathways involved in the sleep-depression process. This aim is in line with the NIH's current Research Domain Criteria (RDoc) proposal initiating research focus onto basic dimensions of functioning (Insel et al., 2010). Although disordered sleep, sleep disorders, and depression all fall under different diagnostic categories (or none at all), they share a number of key symptomatic points, which we aim to identify. Searching for mediators of the relationship between sleep and depression is a key next step for understanding their interrelationship.

### **Potential Emotion-Based Mediators**

Researchers have begun to examine why these variables are correlated – specifically, what is the mutual influence? Problems in sleep and depression are damaging to both physical and psychological health– and tend to overlap with one

another. Various mechanisms underlying the relationship between sleep and depression have been suggested, ranging from biological (increased inflammatory dysregulation in response to sleep disturbance: Muller, Myint, & Schwarz, 2011) to independently contributing common factors (genetic, familial, social or environmental; Kaneita et al., 2009; personality characteristics such as neuroticism, Batterham et al., 2012).

Although both sleep quality and depressive symptoms have been used separately as mediators for other outcomes (i.e. Anderson, 2000), what mediates the link between the two is unknown. One important task is to examine if there are consequences of sleep disturbance that may account for much of the later decline into depression. In this project, three kinds of mediating processes involving emotion, working memory, and health impairments are investigated.

### *Emotional Processing*

Emotional processing is a construct that broadly involves interactive neural networks regarding the processing of cognition and emotion (Brosch, Pourtois, & Sander, 2010). Current literature has suggested that emotional processing can strongly affect brain systems responsible for face recognition and responses, with implications for psychiatric disorders (Vuilleumier & Pourtois, 2007). However, interestingly, the sleep literature has focused more on sleep and affective memory. Walker and van der Helm (2009) note the peculiar absence of emotion processing (instead of memory) research, considering the abundance of evidence suggesting an intimate relationship between sleep and emotion. Similarly, further reviews on sleep and emotion speculate as to underlying mechanisms of this relationship (Kahn, Sheppes, & Sadah, 2013).

Studies on the affective consequences of sleep or depression generally assess two types of testing constructs: recognizing and responding to emotional “events.” Both are defined below and assessed as potential mediators. The presence of problems within these two domains throughout depression and disordered sleep points to probable impairment in emotional processing as a viable potential mediator.

### *Emotion Recognition.*

Recognition of emotion is undoubtedly a skill central to emotional processing; without accurate detection of type of emotion, information processing is skewed at an early stage (Brosch, Pourtois, & Sander, 2010). Recognizing and accurately identifying facial emotions has been associated with both disordered sleep and depression in the past. As recognizing emotions occurs within a social environment, problems with emotion recognition can interfere with communication, interpersonal relationships, and the ability to receive/give social support (Szanto et al., 2012). Wrongly assessing emotions in a social context can increase the likelihood of behaving inappropriately emotionally and experiencing negative consequences in social situations– which describes the interpersonal problems seen in many mental health issues (Kornreich et al., 2002). The causal link between sleep deprivation and disturbed ability to accurately identify emotions, combined with emotional impairment as a predictor of depressive status makes disturbance in emotion recognition a plausible mediator.

Results from acute experimental sleep deprivation shows that it impairs ability to correctly categorize emotions. Sleep deprived individuals are, on the whole, less accurate at identifying all emotional expressions (Cote et al., 2013; Pallesen et al., 2004; Schwarz

et al., 2013; Wagner et al., 2003). More in depth correlational assessments have found similar patterns: elevated night awakenings and decreased sleep efficiency as assessed by actigraphy predict poorer performance in emotional information processing conditions (identifying emotional expressions), but not in neutral identification (i.e. gender identification) conditions. (Soffer-Dudek et al., 2011). Impairing various aspects of sleep seems to consistently affect accuracy in identifying emotional expressions.

Several studies suggest close links between impaired emotion processing and depression; in fact, many suggest that earlier impairment in emotional facial recognition may be a risk factor for depression. Cross-sectionally, Csukly and colleagues (2011) reported that the recognition rate for a happy face decreased with higher BDI scores. Those with depression score at a higher level of impairment on emotional recognition and expression, understanding emotions, emotional management, and emotional control (Nolidin et al., 2013). Other studies have reported that facial recognition problems can serve as risk factors for future internalizing disorders. Impairments on emotion recognition are associated with future suicidal risk in aging depressed inpatients (Szanto et al., 2012). Joormann and colleagues found that high risk girls of mothers with depression made significantly greater errors in identifying angry faces and required higher intensity to identify sad faces (Joormann, Gilbert, & Gotlib, 2010). Given these foundational results, it appears as though impairment in facial recognition is a good candidate in accounting for some of the relationship between disordered sleep and depression.

### *Affective Responding.*

Responses to emotional stimuli are another important aspect of emotional processing. Emotions are defined as quick-moving reactions that follow the processing of a meaningful stimulus, which typically involve changes in subjective feelings and behavior (Ekman, 1992; Keltner & Gross, 1999). Responding to emotional events is an integral part of successfully interacting appropriately with our social environment.

When experimentally manipulating sleep deprivation (the most common method when studying sleep), reactivity results are mixed. Researchers interested in sleep have mainly operationalized sleep problems in the form of experimental acute deprivation. While this method has its advantages, there are often differences between quantity and quality of sleep (in terms of predictors, Davidson, 2013; and outcomes, Pilcher, Ginter, & Sadowsky, 1997). Unsurprisingly, then, the evidence is split in the case of emotional reactivity as an outcome variable. Certain studies do find an increase in reactivity in healthy controls following problematic sleep (Franzen et al., 2009; Rosales-Lagarde et al., 2012); however, other studies report that sleep deprivation decreases facial reactions in response to emotional stimuli (Minkel, 2011; Schwarz et al., 2013). While research suggests that affective responding is altered by sleep difficulties, the direction and strength of this response is unclear.

Reactions to emotional stimuli are similarly associated with depression. For example, a meta-analysis of 19 laboratory studies on MDD found a blunting of emotional reactivity consistent with Emotion Context Insensitivity theory (ECI: Bylsma, Morris, & Rottenberg, 2008). In fact, recent genetic research suggests that in depressed individuals the interleukin 1 beta gene is associated with less responsiveness to emotional stimulation

and, in a longitudinal component, also associated with an increased risk for nonremission (Baune et al., 2010). Given that impairments in sleep lead to changes in affective responses and decreased emotional reactivity is predictive of depression, altered emotional reactivity is a prime candidate for mediation.

### **Non-Emotion Based Mediators**

In the following paragraphs, other non-emotion based mediators are defined and considered. Other factors, such as working memory or physical health, contain literature documenting their relationship with both sleep and depression as well (e.g. Ozdemir et al., 2013). These factors are distinguishable from the main proposed mediators in that they represent non-emotion based tasks/skills.

#### *Working Memory*

Executive function involves several mental processes connecting past actions and dictating future ones (Baddeley & Della Sala, 1998). Working memory is an important aspect of executive functioning, tied to coding past memories for future use (Baddeley, 1992). Because there is some evidence that working memory is affected in the cases of both depression and disordered sleep, it is presented as another potential mediator.

In order for working memory to serve as an adequate mediator, indications of disordered sleep impairing working memory, as well as impaired working memory predicting depression, would need to be established. In terms of sleep and working memory, several studies have indicated that shift work, which is associated with poor sleep, has a negative influence on cognitive performance, particularly working memory (i.e. Ozdemir et al., 2013). In fact, good and poor sleepers differ on which aspects of

cognitive performance are impaired: working memory, but not aspects such as episodic memory (Nebes et al., 2009). Generally, individuals who report difficulty falling asleep fare significantly worse on measures revolving around short-term and working memory (Gamaldo, Allaire, & Whitfield, 2008).

Depression and depressive symptoms have been associated with working memory impairment (i.e., Wagner et al., 2014). There is an indication of dopaminergic signaling impairment and as such executive dysfunction seen in those with depression (Schwartz & Nihalani, 2011); however, type or onset of depression does not seem to correlate with working memory (Richard-Devantoy et al., 2013). One review of the meditational effects of cognitive deficits in MDD on workplace impairment over time did find evidence of these deficits in 20-30% of depressed individuals; cognitive deficits, rather than with a focus on working memory, tend to be nonspecific (McIntyre et al., 2013).

### *Health*

Multidisciplinary research has shown that physical health impacts psychological functioning (Myeong, 2012). In general, the more clinically significant concepts associated with sleep and depression have been activity levels and obesity – which are, of course, linked as well. Some studies have shown that problematic sleep adversely impacts health even when controlling for psychological issues (e.g. Calhoun et al., 2011; Vaatainen et al., 201).

In order for health factors to serve as an adequate mediator, indications of disordered sleep impairing health, as well as impaired health predicting depression would need to be established. In a sample of children with excessive daytime sleepiness (EDS),

authors found that sleep difficulties were more associated with obesity than sleep disordered breathing (SDB) or objective sleep disruption (Calhoun et al., 2011). Similarly, nocturnal awakenings have a strong independent association with decreased health related quality of life; obese adolescents also had significantly later bedtimes than healthy youngsters (Vaatainen et al., 2013). However, several studies have investigated longitudinally into the link between sleep and risk of obesity, but have been unable to find any link or change over time (i.e., Lytle et al., 2013).

The link between activity levels and depression has been well documented. Higher levels of physical activities are associated with lower depressive symptoms (Uebelacker et al., 2013). It is clear that, among those individuals with depression, profiles of those who are physically active differ from those who are inactive in that the physically active are less likely to endorse insomnia, fatigue, guilt, and suicidality (McKercher et al., 2013). In a comprehensive study on BMI, depression, and sleep disturbances, higher BMI was associated with decreased sleep efficiency and more time spent awake and moving in depressed individuals; this effect was not found in the control group (Wojnar et al., 2010).

### **The Construct of Sleep Quality**

The concept of sleep quality is an important one because poor sleep quality is associated with many medical and psychological disorders (i.e., Touchette et al., 2012). Although sleep quality has accepted clinical relevance, defining it objectively is difficult. Many sleep quality measures consider several components of the construct, such as duration, arousals, and depth of sleep. Some studies report only moderate to low



correlations between various sleep assessments (in polysomnography and self-report; Unruh et al., 2008), a large body of research continues to validate self-report measures across various other types of sleep measurement (i.e. Monk et al., 2013; Birrell, 1983; Lichstein, 1983).

The PSQI was an effort to develop a more valid self-report measure of sleep quality (Buysee et al., 1989). Although overall sleep-quality as assessed by the PSQI has proven both reliable and valid (see above), aggregate measures of sleep may be too broad (i.e. cover too many separate domains). That is, subscale constructs of sleep may be more predictive of depression or its symptoms than overall sleep quality scores. For instance, suicidal inpatients score higher than nonsuicidal patients on specific subscales of the PSQI: namely, subjective sleep quality, sleep latency, sleep duration, and sleep efficiency (Agargun, Kara, & Solmaz, 1997). Bower and colleagues (2010) found that PSQI subscales subjective sleep quality and daytime dysfunction were both strongly tied to low ambulatory PA. Therefore, an exploratory aim in the present study will be to assess the sleep-depression relationship and its mediators using subcomponents of sleep quality.

### **The Present Study**

Although the temporal association between sleep and depression has repeatedly been documented, with sleep problems predicting later depressive symptoms (i.e. Alvaro, Roberts, & Harris, 2013), it remains unclear why there is a predictive relationship. One proposed pathway for this relationship is impairments in two emotional processing constructs that are moderately correlated but separate constructs: facial recognition and affective response.

The present study aimed to understand the pathways between disordered sleep and depressive symptoms. Specifically, the study assessed both sleep problems and depressive symptoms as continuous measures in an undergraduate sample, a group that increasingly reports both sleep problems and depression symptoms (Iarovici, 2014; Nyer et al., 2013). We assessed potential mechanisms via a cross-sectional study of impairments in emotional processing and other suggested mediators, including working memory and physical health issues.

Two separate components of emotional processing, facial recognition and affective response, were tested as separate mediators of the linear relationship between disordered sleep and depression. Affective response was gathered through reports in response to well-validated emotion-evoking stimuli (IAPS; Lang, Bradley, & Cuthbert, 2008) and emotional perception was indexed through accuracy in identifying facial emotions (Amsterdam dynamic facial expression set; Van der Schalk et al., 2011) among an undergraduate population. In order to establish discriminant validity in models (and to account for the potential that cognitive factors are influencing emotional processing), both non-emotional cognitive processing and health factors were tested as mediators. In addition, the separate components of sleep identified in the PSQI were studied as separate predictors as exploratory analyses.

Based on the strength of previous research, we expected impaired emotional processing to be the key factor in the relationship between sleep and depression, compared with the impact of non-emotional cognitive processing and health factors. Within the mediation framework, we hypothesized that impaired emotional processing (in the form of emotion recognition and affect) would partially mediate the relationship

between disordered sleep and depressive symptoms. Although both working memory and health factors have been connected to sleep and depressive symptoms, we did not predict that either would emerge as a unique component in the mediation model.

## **METHOD**

### **Participants**

200 participants were selected via an internet-based research system for undergraduates at the University of South Florida. All eligible students were invited to sign up for the study. Students signed up online for a specific time slot and all study procedures took place within the Mood and Emotion Laboratory rooms. Given the large gender skew within this particular population pool and gender differences seen in depression, our study was limited to females only. However, both ethnicity and age were tested and used as covariates, when necessary.

Given the nature of the sample, college-age participants were typically between the ages of 18-21. They were currently enrolled in a psychology course, through which by completing the experiment, they received credit in that course. Specifically, participants were given one course credit point per half hour. In general, college students represent a population that complains of both disordered sleep and depression (Iarovici, 2014; Nyer et al., 2013); in fact, these problems on college campuses have been documented extensively with various potential solutions offered (Kadison & DiGeronimo, 2004). And accordingly, duration and quality of sleep are key predictors of academic functions, psychological, and physical health in college students (Wong et al., 2014). These mental health issues are widespread within a college population and are therefore a key focus in terms of participant demographic.

## Materials

*Demographics.* Participants were given a demographic self-report to complete on a computer. Demographic information included age, gender (as a check), ethnicity, income, education, and other similar items.

*Beck Depression Inventory (BDI-II).* The Beck Depression Inventory is a 21-item self-report measure that assesses depression severity. The scale is a 0 to 3 metric, which ranks at 0 for no severity and 3 for the highest severity. The BDI-II has previously shown good psychometric properties (Beck, Steer, & Brown, 1996) and had adequate reliability within this sample ( $\alpha = .92$ ).

*Pittsburgh Sleep Quality Index (PSQI).* The PSQI is a 19-item self-report questionnaire which aims to assess overall sleep quality over the past month. This measure is rated on scales from 0 to 3 (with 3 indicating worse functioning) and contains seven different subscales (subjective sleep-quality, sleep latency, sleep duration, sleep efficiency, sleep disturbances, medicine to sleep, and daytime dysfunction). Sleep component scores can generally be combined for an overall measure of sleep quality (Buysse, 1989) and had adequate reliability within this sample ( $\alpha = .74$ ).

*Perceived Stress Scale (PSS).* Participants reported on their perceived stress in order to serve as a potential covariate. In this study, the version of the PSS with 10 items is used to assess the degree to which the participant's life events are appraised as stressful (Cohen, Kamarck, & Mermelstein, 1983). The original study found that the PSS measures a different and independent construct from depression and in this sample, presented adequate reliability ( $\alpha = .91$ ).

*International Affective Picture System (IAPS).* These pictures are a well-validated series of images from an international database (Lang, Bradley, & Cuthbert, 2008), chosen to represent either pleasant or unpleasant affective states. Pictures included 6 pleasant and 6 unpleasant stimuli, matched for content, valence, and arousal ratings. Positive content images included visuals such as families, animals, and nature; negative content images contained visuals such as injured children and insects.

*Affective Response.* Participants provided baseline ratings of their own affect (on a sliding scale from unpleasant to pleasant) before beginning any tasks, as well as affect in response to emotion-provoking pictures. Participants rated their affect after viewing either pleasant (valence > 8) or unpleasant (valence < 4) pictures. Participants used sliding scales from 0-10 to rate the dimensions of pleasantness (unpleasant-pleasant) and arousal (not aroused-highly aroused).

*Facial Identification.* Participants were given the choice of 9 facial expressions to categorize the expression they had previously seen (anger, contempt, disgust, embarrassment, fear, joy, pride, sadness, and surprise). It was a forced choice categorization of pictorial stimuli from a dynamic facial stimuli set. Facial expressions include those listed above with Northern European males and females intermixed (Amsterdam dynamic facial expression set; Van der Schalk et al., 2011). Participants saw 36 facial trials (9 facial expressions, 2 females and 2 males). Mean recognition rates in undergrad students are .78, with good variability in recognition among different expressions (.59 for contempt; .87 for anger; Van der Schalk et al., 2011). Accuracy was coded as correct or incorrect; analyses utilized the facial identification variable as total number of items correct.

*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).

*Body Mass Index (BMI)*. Participants were asked to report height and weight, from which BMI was calculated.

*Digit Span Task*. To assess working memory, the Digit Span Task was used. This task is a computerized version of the forward digit span used in the WAIS-IV (Wechsler, 1997). This computerized task displays numbers on the screen at second intervals each and then prompts the participant to type the numbers they have seen. The average individual retains about 5-7 numbers in their working memory at one time. This particular test assesses working memory via memory for strings of numbers and has been used in those with sleep problems and depressive symptoms (Ozdemir et al., 2013). Computerized scores displayed the average number of strings assessed consistently.

## **Procedure**

Participants signed up via SONA systems for an hour and a half time slot at the day and time of their choosing. At the designated time, students arrived in the Mood and Emotion Lab and were escorted to the room next door where they were seated

comfortably with a computer. Tasks were fully computerized and responded to solely through the survey database.

Self-report measures that include depression, sleep, health, working memory, and affect were given first through the online survey program Qualtrics. Following the self-report measures, the participant began either the facial recognition or emotional response, which was randomly ordered for each participant. Facial recognition includes the viewing of 36 trials with 9 different facial expressions of North-European male and female models – with prompts after each picture requesting identification of the previous facial emotion (Amsterdam dynamic facial expression set; Van der Schalk et al., 2011).

The emotional response component included an affective response (as determined by a change score from baseline) to various pictorial emotion-provoking stimuli. Participants were prompted to rate their affect immediately following each image meant to provoke pleasant or unpleasant emotions. Following these computer tasks, students were debriefed, given course credit, and escorted out.

## **Hypothesis Testing**

To understand the pathways that influence the relationship between sleep and depression, the main focus of this investigation was testing emotion-based mediation models between disordered sleep and depression. Mediators tested separately within this framework included facial recognition and affective response, aspects of emotional processing. The secondary aim was to test non-emotional mediators: digit span (an index of working memory) and activity levels and BMI (as health factors). We statistically



controlled for several potential confounds, such as age, ethnicity, and other demographic variables.

Combining subscale sleep components into a measure of overall sleep quality is psychometrically valid (Buysse et al., 1989) and was the main predictor variable. However, a number of studies have provided reasoning for looking at the PSQI subscales separately (e.g. Nunes et al., 2008; Osorio et al., 2006; Robert et al., 2005). At times, subscales of the PSQI have proven more strongly associated with depression-relevant outcomes than the overall score: suicidality and positive affect, for example (Agargun, Kara, & Solmaz, 1997; Bower et al., 2010). As such, we limited our search to the relevant subscales from above: sleep duration (average number of hours in bed each night), sleep efficiency (the ratio of average number of hours in bed versus hours actually asleep), and sleep disturbances (awakenings during the night). These exploratory analyses were conducted on significant mediation models after main analyses were complete.

### **Power Analysis**

In order to detect a mediation effect (beta coefficient of .2) with an alpha of 0.05 and power of 0.80 with the planned analyses, a total adequate sample size would be 105. Similar power analyses conducted on chi-square designs similar to mediation reveal sample sizes between 100 and 120. Statisticians have reported that for bootstrapping methods in particular, an N of 100 is a good minimum in order to lower concern for statistical errors (Koopman et al., 2014). Based on this information, we targeted 120 as a sample size for this project.

## **Aims**

*Aim 1.* A.) To test the hypothesis that impairments in emotional processing – facial recognition and affective response - mediate the relationship between disordered sleep and depression and B.) To test the hypothesis that impairments in working memory, physical activity levels, and BMI mediate the relationship between disordered sleep and depression.

*Testing Aim 1.* We used the bootstrapping method to detect mediation. Although the Baron & Kenny method (the “classic” method of testing mediation) is the most cited, it also has the lowest power to detect mediation and other limitations (Pardo & Roman, 2013). Because of this disadvantage, we tested mediation using the bootstrapping method (Efron & Tibshirani, 1993; Shrout & Bolger, 2002). Bootstrapping involves randomly resampling from the data and providing confidence intervals for the indirect effect. It is analytically more complex, but allows for nonnormal indirect effects and decreases the rate of Type I and Type II errors. To estimate indirect effects with Sobel testing included, we utilized Hayes Indirect Process Model (Preacher & Hayes, 2008).

*Aim 2.* To test whether mediation of emotional processing components are a unique component of the relationship.

*Testing Aim 2.* After testing the mediation models for each potential mediator, additional analyses were conducted on each significant model. In the case of facial recognition and affective response, the second analyses controlled for the other non-

emotion based mediators (working memory, BMI, and physical activity levels), such that all other mediators served as covariates. A similar design was used for any mediation that tests significantly in order to determine which mediators were unique components of the relationship above and beyond their counterparts.

*Exploratory Aim 3.* To assess which (if any) subscales of the PSQI are significant IV's in the mediation model.

*Testing Aim 3.* For any mediation models that reached statistical significance, the independent variable of sleep problems was tested with selected subscales of the PSQI, in addition to the full PSQI sleep quality metric. These analyses were exploratory, and conducted to understand which aspects of sleep problems may be driving this mediation effect.

## **RESULTS**

### **Sample Characteristics**

The sample consisted of 200 female participants recruited online at a southeastern university. Most participants were young adults in the age range of 18-21 years old (82% of the sample), while 22-25 year olds accounted for 15%, 26-39 for .5% and 30+ accounting for 2.5% of the sample. The ethnic composition of the sample was 51% White, 20.5% Hispanic, 15% Black, 7% Asian, 3% Middle-Eastern, and 4.5% reporting other. Key variables (sleep quality, depression, affective response, etc.) were normally distributed. Ranges for sleep quality were 1-17 (with 5 being the “clinical” cutoff for problematic/disordered sleep on the PQSI) and 0-36 for depression (which adequately captures all levels of depression following the BDI-II cutoffs). Mean depression scores of the sample were 9.21, indicating that the students were, on average, reporting low to moderate level of depression. Similarly, mean PSQI scores were 6.41, slightly above the clinical cutoff for problematic sleep patterns. Sample means and other characteristics are included in Table 1.

### **Relationships between Key Variables**

Before analyzing our primary questions, we wanted to confirm the expected relationships between variables. As expected, depression was strongly correlated with impaired sleep quality ( $r = .58$ ). In line with one of the mediators, there was also a negative relationship between impaired sleep quality and unpleasant affective response ( $r$

= -.21), such that impaired sleep quality was associated with less affective responses to unpleasant stimuli. Following this, there was a negative relationship between depressive symptoms and unpleasant affective response ( $r = -.32$ ), in that as depressive symptoms increased, affective response to unpleasant stimuli decreased. The relationships between these three variables support the viability of our mediation analysis.

In terms of the other mediators, unexpectedly, there were no significant relationships between sleep quality, facial recognition, pleasant affective response, BMI, or working memory. However, there was a significant positive relationship between pleasant affective response and depressive symptoms ( $r = .17$ ), such that as depressive symptoms increased, affective response to pleasant stimuli increased. Given the usual negative relationship between depression and positive emotions, this correlation was unexpected. The lack of significant relationships between facial recognition, BMI, working memory, and the independent/dependent variables obviated a need to test mediation models.

### **Mediation Model: Testing Aim One**

A total of 200 participants' data was entered into the Hayes Process Model for Mediation in SPSS. In accordance with our first aim, all mediators were tested separately within the model: Three emotion-centric and two other-centric mediators with sleep quality as a predictor and depression symptom severity as the outcome. Although three potential emotion mediators - Facial Recognition (FR), Unpleasant Affective Response (UAR), and Pleasant Affective Response (PAR) and two non-emotion based mediators - Body Mass Index (BMI) and Working Memory (WM) – were hypothesized, the lack of

correlation between all mediators (excepting UAR) with the IV and DV precluded a test of mediation. In order to serve as a significant mediator for a bootstrapping design, the upper and lower bounds of the confidence intervals must not contain 0. UAR emerged as a significant mediator within a separate model (see Table 3).

The indirect path between sleep quality and depressive symptoms via UAR was significant. The specific indirect effect through UAR accounted for 5% of the variance of the relationship ( $kappa = .05$ ). Additionally, the Sobel test was significant ( $p < .05$ ) for UAR as a mediator. This significant mediation suggests that affective responses to unpleasant events accounted for a small portion of the relationship that exists between impaired sleep quality and depressive symptoms. Since our measure of UAR was based on a change score from baseline, we took the additional step of examining whether it remained a mediator when controlling for baseline unpleasant responses. UAR did not remain a significant mediator under this scenario, suggesting the possibility that baseline unpleasant responses may have played a role (CI's include 0).

### **Mediation Model: Testing Aim Two**

In accordance with our second aim, the significant mediator was entered into the model with other mediators as covariates. As UAR was the only significant mediator, UAR was entered into the formula as a mediator with the non-emotion mediators (BMI and WM) entered as covariates. This specific model was utilized to test whether UAR was a significant and, more importantly, unique mediator in the relationship between sleep quality and depression. To serve as a significant mediator in a bootstrapping design, the upper and lower bounds of the confidence intervals must not contain 0, as above.

UAR emerged as a significant mediator, even when controlling for both BMI and WM as covariates, marking it as a unique mediator among this group of variables (see Table 3). The indirect effect for UAR with covariates included was numerically similar to the indirect effect without covariates included, probably due to the nonsignificance of the covariates. Therefore, it can be statistically concluded that affective response to unpleasant stimuli was a unique mediator in the relationship between sleep quality and depression, even after taking into account indices of health (e.g. BMI) and of cognitive functioning (e.g. working memory).

### **Exploratory Analyses of PSQI Subscale Predictors**

In accordance with our third exploratory aim, the significant mediator was entered with a subscale of the sleep quality scale rather than the overall scale. As UAR was the only significant mediator, UAR was entered into the formula as a mediator with three different predictors for three different models tested: sleep duration, sleep efficiency, and sleep disturbances. This specific model was utilized with various indices of sleep as the predictor to explore which aspects of sleep were driving this relationship. In order to serve as a significant mediator for a bootstrapping design, the upper and lower bounds of the confidence intervals must not contain 0, as before. UAR emerged as significant mediator with sleep disturbances as a predictor. However, neither sleep duration nor sleep efficiency was significantly mediated by UAR in relation to depressive symptoms (see Table 4).

*Sleep Disturbances.* The indirect path of UAR was a significant mediator of the relationship between sleep disturbances and depressive symptoms. The specific indirect

effect through UAR accounted for 5% of the variance of the relationship ( $kappa = .05$ ). Additionally, the Sobel test was significant ( $p < .05$ ) for UAR as a mediator. This model of sleep disturbances was nominally similar to the model of overall sleep quality. It is thus plausible that sleep disturbances are responsible for the meditational relationship between sleep quality and depression when UAR is the mediator.



## DISCUSSION

Although the relationships between sleep and emotion (Walker & van der Helm, 2009) and emotion and depression (Vuilleumier & Pourtois, 2007) are well established, the processes by which the two co-occur are not well established. This study was a first step in testing pathways by which disordered sleep leads to increases in depression symptoms. We found expected correlations between sleep, emotion, and depression, and also found that affective responses partially mediated the relationship between poor sleep and depression. Specifically, both poor sleep and increased depression severity was associated with reduced emotional responses to unpleasant stimuli. In our sample, blunted emotional responding to unpleasant images partially accounted for the link seen between sleep and depression. However, when we performed the additional control of controlling for baseline reports of negative emotion, our mediation effect no longer survived, suggesting that baseline emotion reports contribute to this effect.

Understanding the role of sleep in depression is important given the social and economic burdens of sleep loss (Kessler et al., 2011) and alarming increases in depression prevalence (Kessler, 2002). Specifically in our sample, we found a mean disordered sleep score higher than the cutoff for problematic sleep, indicating that college students are reporting considerable disordered sleep. The exploration of pathways by which bad nights of sleep turn into a depressive disorder has been particularly understudied. Sleep may uniquely interfere with emotional experiences, and thus erode various emotional responses (Walker & van der Helm, 2009). It is especially important to

isolate these pathways, given links between depression and other potential mediators (cognitions, physical attributes, etc.). This study specifically examined three potential categories of mediators: cognition, health, and emotion. We found that only emotional reactivity mediated the sleep-depression relationship.

In terms of sleep quality, exploratory analyses revealed interesting trends about which types of sleep are important as predictors. Sleep disturbances, of all the sleep scales, proved a significant independent predictor within the model, while other subscales did not. Though this finding is relatively novel, it has precedence. Emotion research has emphasized the importance of examining the qualitative aspects of sleep (e.g. Soffer-Dudek, 2011). Physiologically, sleep disturbances may be indicative of impaired REM sleep, which have direct consequences for emotional reactivity (Goldstein & Walker, 2014). These findings reinforce the idea that specific sleep characteristics – sleep disturbances, most especially – may have incremental predictive power when looking at certain emotion-related domains.

Although there is no consensus about conventions about the magnitude of mediation effect sizes, UAR's effect size of .11 could be considered a medium effect (.09 for a medium effect; Preacher & Kelley, 2011). Unpleasant responses accounted for 5% of the relationship between sleep and depression within this sample at one time point. The magnitude of this finding is similar to other emotion-relevant mediations within the literature (e.g. emotion regulation as a mediator, Besharat, Nia, & Farahani, 2013). Additionally, pinpointing a significant emotion-related mediator has potential clinical implications in helping define treatment focus. Interventions with a strong focus on affective responses to everyday stimuli, for example, may follow the pattern of focusing

on emotion strategies, which have predicted recovery from depression (Arditte & Joormann, 2011). These data suggest that emotion-related foci would be a more fruitful place to intervene to blunt the harmful effects of poor sleep than memory or health.

It is both noteworthy and puzzling that our facial recognition construct was uncorrelated with measures of sleep and depression (which precluded it playing a role as a mediator); similarly, other mediators of interest were uncorrelated. Although the accuracy rates of facial recognition were consistent with other college samples (Van der Schalk et al., 2011), the software was restricted to static faces at a single intensity. Previous effects of depression have been found when looking at variations in the intensity of facial expressions (e.g. Joormann, Gilbert, & Gotlib, 2010), which was not tested here. Although it is difficult to conclusively interpret null findings, these factors may have contributed.

Likewise, all other mediators (BMI, working memory, and physical activity) were unrelated to key variables. Although both sleep and depression have been associated with all non-emotion related mediators (see above), it is possible that it is easier to detect these effects in a clinical, rather than college sample. The relationship between BMI and sleep, for example, has been seen in clinically depressed but not healthy control groups (Wojnar et al., 2010). Additionally, there are studies that have been unable to find the link between sleep and obesity (i.e., Lytle et al., 2013). Similarly, in regards to executive functioning, many of the findings in this literature have been either small effects or are nonspecific (McIntyre et al., 2013). It is possible that the present study was underpowered to detect effects for these variables.

In the present sample, college students represent a population where environmental and individual sleep disturbances are common. This is most notable in both subscale and overall scores, in which this college sample scored higher than the cutoff for clinically significant sleep problems, suggesting it should not be regarded as a healthy sample. Given the necessity of empirically supported treatments, extending these findings to a clinical population would be a logical next step. It would also be useful to examine these issues in a longitudinal design, which would be needed to clarify the temporal order of effects between sleep problems, emotion processing, and depression symptoms. Longitudinal designs, then, are important for developing stronger arguments about causal pathways.

In this sense, these findings need to be considered in the light of a few limitations. Because the design was cross-sectional in nature, there is no direct evidence for directional causality within the model. Nevertheless, our conclusions are guided by previous research demonstrating that sleep predicts emotional responding and depression (Schwarz et al., 2013; Alvaro, Roberts, & Harris, 2013), as well emotional responses predicting depression (Baune et al., 2010). Finally, our mediation effect was only a medium effect size. Preliminary results such as these raise the question of how large of an effect would be found if emotional reactivity was indexed via other means (e.g., reactivity in everyday life).

## **Conclusions**

Overall, this study represents an important first step in the field to explore the pathways by which impaired sleep quality influences depression. Specifically, our results indicate that affective emotional responding (in this case, to specific cued images) is a

significant pathway, which sleep progresses into depression severity. In order to advance the study of process and pathway-related models within transdisciplinary realms of psychopathology, additional investigation into the role of emotional responding in the sleep-depression relationship would be highly valuable.

## TABLES

Table 1. Means and standard deviations of key variables.

	Mean	Standard Deviation	N
<b>PSQI</b>	6.41	3.19	195
<b>BDI</b>	9.21	8.12	200
<b>Unpleasant AR</b>	3.08	1.42	200
<b>Pleasant AR</b>	.44	1.36	198
<b>Facial Rec</b>	28.41	3.42	200
<b>BMI</b>	23.45	4.90	194
<b>WM</b>	7.10	1.03	196
<b>IPAQ</b>	2782.35	1992.75	198

\* PSQI = Pittsburgh Sleep Quality Index; BDI = Beck Depression Inventory; Unpleasant AR = Unpleasant Affective Response; Pleasant AR = Pleasant Affective Response; Facial Rec = Facial Recognition; BMI = Body Mass Index; WM = Working Memory; IPAQ = International Physical Activity Questionnaire

\*N's varied slightly due to computer problems and computational impossibilities (solely for BMI)

Table 2. Pearson product-moment correlations between the key variables.

Scale	<b>PSQI</b>	<b>BDI</b>	<b>UAR</b>	<b>PAR</b>	<b>FR</b>	<b>BMI</b>	<b>WM</b>	<b>IPAQ</b>
<b>PSQI</b>	1	.58***	-.21***	.06	-.01	.02	-.02	.12
<b>BDI</b>	.58***	1	-.32***	.17**	-.05	.03	-.03	.07
<b>UAR</b>	-.21***	-.32***	1	-.08	.15**	.03	-.10	.15**
<b>PAR</b>	.06	.17**	-.08	1	.02	.04	.04	.00
<b>FR</b>	-.01	-.05	.15**	.02	1	-.07	.05	.11
<b>BMI</b>	.02	.03	.03	.04	-.07	1	.07	.06
<b>WM</b>	-.02	-.03	-.10	.04	.05	.07	1	-.02
<b>IPAQ</b>	.12	.07	.15**	.00	.11	.06	-.02	1

\* PSQI = Pittsburgh Sleep Quality Index; BDI = Beck Depression Inventory; UAR = Unpleasant Affective Response; PAR = Pleasant Affective Response; FR = Facial Recognition; BMI = Body Mass Index; WM = Working Memory; IPAQ = International Physical Activity Questionnaire

\*\* p< .05 significance level, \*\*\* p< .01 significance level

Table 3. Bootstrapping mediation effects with sleep quality as the predictor.

	<i>Effect</i>	<i>SE</i>	<i>CI (lower)</i>	<i>CI (upper)</i>
<b>UAR</b>	.11**	.06	.03	.26
<b>PAR</b>	.02	.03	-.01	.10
<b>FR</b>	.00	.01	-.01	.03
<b>BMI</b>	-.00	.01	-.02	.02
<b>WM</b>	.00	.01	-.02	.03
<b>IPAQ</b>	-.01	.03	-.08	.03
<b>UAR<sup>1</sup></b>	.11**	.06	.02	.28

\*UAR = Unpleasant Affective Response; PAR = Pleasant Affective Response; FR = Facial Recognition; BMI = Body Mass Index; WM = Working Memory; IPAQ = International Physical Activity Questionnaire; UAR<sup>1</sup> = Unpleasant Affective Response with other non-emotion mediators as covariates

\*\*If the confidence intervals do not contain 0 = significance



Table 4. Bootstrapping mediation effects with various sleep subscales as predictors.

		<i>Effect</i>	<i>SE</i>	<i>CI (lower)</i>	<i>CI (upper)</i>
<b>Duration</b>	UAR	.29	.23	-.08	.83
<b>Efficiency</b>	UAR	.03	.24	-.44	.51
<b>Disturbances</b>	UAR	.59**	.35	.08	1.51

\*UAR = Unpleasant Affective Response; Duration = Sleep Duration subscale; Efficiency = Sleep Efficiency subscale; Disturbances = Sleep Disturbances subscale

\*\*If the confidence intervals do not contain 0,  $p < .05$  significance

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## APPENDICES

### Appendix I: IRB Approval.

The Institutional Review Board (IRB) at the University of South Florida (USF) reviewed and approved this study (IRB#: Pro00017863).



RESEARCH INTEGRITY AND COMPLIANCE  
Institutional Review Boards, FWA No. 00001669  
12901 Bruce B. Downs Blvd., MDC035 • Tampa, FL 33612-4799  
(813) 974-5638 • FAX(813)974-7091

7/9/2014

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

RE: **Expedited Approval for Initial Review**  
IRB#: Pro00017863  
Title: The emotional processing system: mediators for the link between disordered sleep and depression

**Study Approval Period: 7/7/2014 to 7/7/2015**

Dear Ms. O'Leary:

On 7/7/2014, the Institutional Review Board (IRB) reviewed and **APPROVED** the above application and all documents outlined below.

**Approved Item(s):**  
**Protocol Document(s):**  
[IRB Protocol OLeary](#)

**Consent/Assent Document(s)\*:**  
[Thesis Informed Consent.doc.pdf](#)

\*Please use only the official IRB stamped informed consent/assent document(s) found under the "Attachments" tab. Please note, these consent/assent document(s) are only valid during the approval period indicated at the top of the form(s).

It was the determination of the IRB that your study qualified for expedited review which includes activities that (1) present no more than minimal risk to human subjects, and (2) involve only procedures listed in one or more of the categories outlined below. The IRB may review research through the expedited review procedure authorized by 45CFR46.110 and 21 CFR 56.110. The research proposed in this study is categorized under the following expedited review category:

(7) Research on individual or group characteristics or behavior (including, but not limited to, research on perception, cognition, motivation, identity, language, communication, cultural beliefs or practices, and social behavior) or research employing survey, interview, oral history, focus group, program evaluation, human factors evaluation, or quality assurance methodologies.

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 by an amendment.

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,

A handwritten signature in black ink that reads "John A. Schinka, Ph.D." The signature is written in a cursive style with a large initial 'J' and 'S'.

John Schinka, Ph.D., Chairperson  
USF Institutional Review Board