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A Daily Study Of The Sleep-Pain Relationship In Fibromyalgia

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A DAILY STUDY OF THE SLEEP-PAIN RELATIONSHIP IN FIBROMYALGIA

by

MAREN E. HYDE-NOLAN

DISSERTATION

Submitted to the Graduate School

of Wayne State University,

Detroit, Michigan

in partial fulfillment of the requirements

for the degree of

DOCTOR OF PHILOSOPHY

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Approved by:

Advisor

Date

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CHAPTER 1

INTRODUCTION

Fibromyalgia (FM) impacts millions of individuals around the world and is characterized by widespread chronic pain and tenderness as well as nonrestorative sleep, fatigue, and stiffness (Wolfe et al., 1990; Wolfe et al., 2010). It is a costly condition, both in terms of financial burden as well as disability and reduced quality of life (Berger et al., 2010; Mease, 2005). In addition, more than 90% of individuals with FM report poor sleep quality, and this is often described as light and unrefreshing sleep (Moldofsky, 2008). The high prevalence of sleep disturbance in FM suggests that this may be a contributing factor to the pain experience.

The relationship of sleep and pain is well established in the literature; however, the direction of this relationship is unclear. Experimental studies have been conducted with healthy, pain-free people in order to test the directionality of the sleep and pain relationship. Studies involving sleep restriction and total sleep deprivation have found increased pain sensitivity the next day compared with no restriction conditions (Haack & Mullington, 2005; Onen, Alloui, Gross, Eschallier, & Dubray, 2001; Roehrs, Hyde, Blaisdell, Greenwald, & Roth, 2006). Research has also found that disrupted sleep continuity, rather than restricted sleep, is a significant predictor of next day's pain (Smith, Edwards, McCann, & Haythornthwaite, 2007). These experimental findings on individuals without chronic pain conditions suggest that disturbed sleep may amplify the pain found in individuals with chronic pain.

Studies have also been conducted on chronic pain populations including rheumatoid arthritis and FM, focusing on how poor sleep results in increased pain.

Ağargün et al. (1999) found that an increase in subjective sleep problems was associated with increased pain sensitivity. A similar study by Kolar et al. (1989) assessed problems with sleeping as well as severity of muscle aching, and found that sleeping difficulties were associated with tenderness and muscular aching that are characteristic of FM. Longitudinal studies have also found that poor sleep at baseline is predictive of increased pain after a year (Bigatti, Hernandez, Cronan, & Rand, 2008).

Research has also focused on the impact of pain on subsequent sleep. Nicassio and Wallston (1992) found that arthritis pain predicted sleep disturbance after 2 years, but prior sleep disturbance did not predict future pain. Another study by Pilowsky, Crettenden, and Townley (1985) found that chronic pain patients who slept poorly reported that they slept fewer hours and also reported significantly higher pain intensity compared with those who slept normally.

The prevalence of sleep difficulties in individuals with chronic pain, and especially FM, is very high. The relationship between sleep and pain is evident based on the literature, although the direction of this relationship remains unclear. Further research is needed to determine whether there is a stronger relationship between sleep difficulties and subsequent pain or between pain and subsequent sleep problems, in people with FM. Research is also needed to explore the factors that predict individual differences in this sleep and pain relationship.

Goals of this Study

There were two primary goals for this study. The first was to determine the direction of the sleep and pain relationship in a large sample of individuals diagnosed with FM. Both objective (actigraphy) and subjective (daily diary) measures of nightly

sleep were analyzed to determine the relationship on next day's pain, as well as assessing the relationship between one day's pain on the next night's objective and subjective sleep variables. The second goal was to assess factors that may account for individual differences found in the sleep and pain relationship. Participants completed self-report measures of depression, negative affect, pain catastrophizing, and age at baseline, and these were analyzed as potential moderators of the relationship between sleep and pain.

CHAPTER 2

LITERATURE REVIEW

Fibromyalgia (FM) is a multifaceted disorder characterized by widespread chronic pain and tenderness (Wolfe et al., 1990). Since this initial definition of FM was offered in 1990, the criteria for FM have evolved to include nonrestorative sleep, fatigue, and stiffness (Wolfe et al., 2010). Fibromyalgia impacts millions of individuals worldwide and has an estimated prevalence of 2% of adults in the United States (Arnold, 2010), with an approximately 8:1 female to male ratio (Berger, Dukes, Martin, Edelsberg, & Oster, 2007; Wolfe, Ross, Anderson, Russell, & Hebert, 1995). The average age of FM onset is between 30 and 50 years, and the incidence of FM increases with age, rising in middle age (50-59 years) and dropping in older age groups (80 years and above; Wolfe et al., 1995).

Fibromyalgia is a costly condition and one of the 100 most common diagnoses made in family medicine (Arnold, 2010). This debilitating syndrome occurs in 5% to 6% of adult patients who present at general medical and family practice clinics, and between 10% and 20% of patients presenting at rheumatology practices (Goldenberg, Simms, Geiger, & Komaroff, 1990; Wolfe et al., 1995). Two studies of large claims databases in the United States reported that healthcare costs for people with FM are 2 to 3 times greater than for individuals without FM due to more frequent doctor's office or emergency room visits and a greater number of prescription medications (Berger et al., 2010; White et al., 2008). In addition to the financial burden of FM, many patients also suffer from disability and reduced quality of life. In a recent review, Mease (2005) indicated that there is a greater negative impact on quality of life with FM than many

other diseases, including chronic obstructive pulmonary disease and arthritis. Jones, Rutledge, Jones, Matallana, and Rooks (2008) conducted a large survey of women with FM about how this condition has impacted their activities of daily living. They reported that 25% of the women surveyed had difficulty bathing and taking care of personal needs, and more than 60% had a difficult time with light housework, lifting or carrying 10 pounds, traveling up or down one flight of stairs, or walking one half mile (Jones et al., 2008).

In addition to chronic widespread pain and tenderness to touch, individuals with FM often have a variety of other symptoms and comorbid conditions. Berger and colleagues (2007) noted in their recent study from a United States health insurance database that, compared with age and sex-matched patients without FM, those with FM were more likely to have comorbidities including circulatory disorders, painful neuropathies, diabetes, gastroesophageal reflux disorder, irritable bowel disorder, anxiety, depression, and sleep disorders.

Poor sleep quality is reported by more than 90% of individuals with FM, and this is often characterized as light and unrefreshing sleep (Moldofsky, 2008). In addition to difficulties falling and staying asleep, a complaint of nonrestorative sleep is common. A recent review of the literature (Moldofsky, 2009) explored the polysomnographic (laboratory sleep study) findings of this population and noted the common disturbances in sleep physiology including delayed sleep onset; reductions in sleep efficiency, slow wave sleep, and REM sleep; and an increase in motor activity. The high prevalence of sleep disturbance in FM suggests that this may be a contributing factor to the pain experience.

Sleep and Pain

Both pain problems and sleep disorders are considered among the most common societal complaints, so it is not surprising that these two conditions often co-occur. What is unclear is the direction of the pain and sleep relationship. A literature review by Moldofsky (2001) indicated that millions of Americans complain that their experience of nighttime pain interferes with falling asleep, staying asleep, and often results in early morning awakenings. Contrariwise, disturbances in sleep have also been found to increase the perception of pain. A micro-longitudinal study by Edwards, Almeida, Klick, Haythornthwaite, and Smith (2008) found that self-reported sleep one night was a significant predictor of the next day's pain, as well as pain frequency predicting sleep duration the following night. Human studies have also found that unrefreshing nocturnal sleep in combination with disturbances to sleep physiology result in increased daytime musculoskeletal pain and fatigue (Moldofsky, 2001). A more recent literature review by Finan, Goodin, and Smith (2013) suggested that impairments in sleep may be a more reliable and stronger predictor of pain than pain is of sleep impairments. I now review this literature on the relation of sleep disturbances and pain.

Experimental Sleep Manipulation and Pain

To test the directionality of the sleep and pain relationship, experimental studies have been conducted with healthy, pain-free people. A study by Onen et al. (2001) sought to determine what effect total sleep deprivation, interruptions in slow wave sleep and rapid eye movement (REM) sleep, and recovery sleep would have on pain threshold as assessed with a pressure dolorimeter. They found that total sleep deprivation reduced pain threshold 8% (i.e., increased pain sensitivity) the following

day. Although neither slow wave sleep nor REM sleep interruptions resulted in a significantly decreased pain threshold, recovery sleep following the slow wave sleep interruption led to a 15% increase in pain thresholds. Haack and Mullington (2005) discovered that two nights of partial sleep restriction (4 hours) resulted in reports of spontaneous bodily pain, and that pain was amplified with subsequent nights of partial sleep restriction. Another study by Roehrs et al. (2006) found that both total sleep deprivation and sleep restriction significantly reduced pain threshold compared with a no sleep reduction condition. These findings provide support for the strong impact that sleep restriction has on the next day's pain.

Research has also examined the role of disrupted sleep continuity and subsequent pain. Taylor and colleagues (2007) examined the comorbidity of medical problems and insomnia and discovered that the most common sleep complaint of individuals with chronic pain is multiple awakenings throughout the night as a result of pain-related arousals. Smith et al. (2007) developed a sleep disruption paradigm that awakens participants pseudorandomly each hour during an 8-hour sleep period in order to mimic the continuity disturbance reported by individuals with chronic pain. They discovered that healthy female participants who had disrupted sleep continuity reported spontaneous pain the following day compared with participants who had an equivalent amount of restricted sleep and healthy controls who had uninterrupted sleep for 8 hours. These findings suggest that the disruption of continuous sleep may be an even stronger predictor of subsequent pain than restriction of sleep.

Other research has examined pain sensitivity of sleepy but healthy individuals. One study by Chhangani and colleagues (2009) compared the pain sensitivity of sleepy

versus alert healthy individuals. The sleepy participants had a reduced pain threshold compared with the alert participants. Another study by Roehrs, Harris, Randall, and Roth (2012) increased the amount of sleep allowed over 4 nights in sleepy but pain-free individuals. This increased time in bed resulted in decreased pain sensitivity compared with controls who maintained their regular sleep schedule. These studies provide support for the pathway that alterations in sleep influence pain perception.

Even participants without chronic pain conditions experience a significant decrease in pain threshold when sleep is reduced, disrupted, or eliminated. Therefore, these experimental findings on people without chronic pain conditions suggest that disturbed sleep quality and duration in individuals with chronic pain, such as rheumatoid arthritis (RA) and FM, may contribute to their increased pain.

Sleep and Pain in Chronic Pain Conditions

Studies have examined how poor sleep predicts increased pain among people with RA. A recent study by Irwin and colleagues (2012) restricted the sleep (4 hours) of both healthy participants and individuals with RA. They found that one night of partial sleep deprivation resulted in increased self-reported fatigue, anxiety, depression, and pain for the participants with RA, but not for the healthy controls. Moldofsky, Lue, and Smythe (1983) studied the impact that disturbed sleep has on the morning symptoms experienced by people with RA. The patients were found to have an alpha electroencephalographic (EEG) sleep anomaly and subsequent arousal state during sleep as well as an increase in peripheral joint tenderness the following morning. A similar study compared two groups of patients with RA: those who complained of morning symptoms and those who were free of such symptoms (Moldofsky, Lue, &

Saskin, 1987). Those patients with morning symptoms had fragmented sleep characterized by periodic leg movements and repetitive electroencephalographic (EEG) arousals compared with those patients without morning symptoms. These studies suggest that a nonrestorative sleep disorder may lead to bodily symptoms upon awakening.

Several studies have also been conducted with FM, focusing on how poor sleep results in increased pain. Ağargün et al. (1999) examined the association between pain threshold, measured with a manual algometer, and subjective sleep quality. They found that an increase in sleep problems was associated with a decreased pain threshold, suggesting that greater sleep disturbance is associated with increased pain sensitivity in FM.

Theadom, Cropley, and Humphrey (2007) explored the effect of sleep and coping on pain in FM. Participants were asked to complete self-report measures on sleep quality, forms of coping, and pain. They found that 99% of participants reported poor sleep quality and that this was significantly related to pain, whereas coping strategies were not related to pain.

Bigatti et al. (2008) conducted a longitudinal study that assessed whether baseline sleep predicted subsequent pain in participants with FM. They had participants complete self-report questionnaires on sleep quality and pain at both baseline and at a 1-year follow-up assessment. The results suggest that poor sleep is predictive of subsequent pain in the FM population, even after a year.

Another study that looked at the sleep and pain relationship had participants with chronic, widespread, unexplained muscular aching—which is characteristic of FM—

assess the severity of their muscle aching as well as any problems with sleeping including falling asleep, frequent nocturnal awakenings, or waking too early (Kolar et al., 1989). The results of this study indicate that sleeping difficulties are associated with the tender points and muscular aching that are characteristic of FM. Similarly, Davies et al. (2008) followed individuals with chronic widespread pain over a period of 15 months and found that those participants who reported good quality sleep at the end of the study had a resolution of their pain symptoms. These results suggest that restorative sleep may improve the long-term prognosis of individuals with chronic pain.

Finally, Tang, Goodchild, Sanborn, Howard, and Salkovskis (2012) examined the temporal link between sleep and pain in individuals with various chronic pain conditions and concomitant insomnia. Participants wore an Actiwatch, a small, watch-like device that measures movement and activity level with an embedded accelerometer, and completed electronic daily diaries with questions about sleep, pain, mood, and arousal, for 7 days. They found that sleep quality was a predictor of pain the next morning, but that the effect of high quality sleep did not extend into the following afternoon. Results also indicate that pre-sleep pain was not a reliable predictor of the subsequent night's sleep. Rather, sleep was significantly predicted by pre-sleep cognitive arousal.

These studies of the effects of experimentally manipulated sleep on pain as well as the studies conducted with chronic pain populations support the hypothesis that poor sleep increases pain. Yet there are another set of studies that have examined the impact of pain on subsequent sleep.

To examine whether the presence of pain predicts subsequent poor sleep, Pilowsky et al. (1985) compared the amount of pain experienced between chronic pain

patients who slept poorly with those who slept comparatively well. The poor sleepers indicated that they slept fewer hours and reported significantly higher pain intensity compared with those patients who stated that they slept normally. Another study looked at how pain predicts poor sleep in a RA sample. Nicassio and Wallston (1992) collected self-report data on sleep disturbance and pain at two different time points within a 2-year period. Longitudinal regression analyses indicated that arthritis pain predicted sleep disturbance after 2 years, but prior sleep disturbance was not found to have an impact on subsequent pain. These studies provide support for the impact of pain on subsequent sleep. A final category of studies, which I will now review, have found a bidirectional relationship between pain and sleep.

Bidirectional Relationship between Pain and Sleep

The first study examining the bidirectional relationship between pain and sleep was conducted by Affleck, Urrows, Tennen, Higgins, and Abeles (1996) who were interested in determining the effect of attention to pain on nightly sleep. Women with a diagnosis of FM used palm-top computers to answer daily self-report questions about sleep, pain, and attention to pain. They discovered that reports of greater pain during the day predicted a worse night's sleep, and that increased attention to pain also predicted poorer sleep. In addition, those individuals who reported sleeping poorly also reported more pain and more attention to pain the following day. This study suggests that the direction of the sleep-pain relationship may not be conclusive and suggests that more research is needed, particularly within FM.

Another study, which has many design features that parallel the current study, evaluated the influence of depression on the bidirectional relationship between sleep

and pain in chronic pain patients (O'Brien et al., 2011). Twenty-two women with various forms of chronic pain including facial pain, back pain, and fibromyalgia, completed self-report measures of sleep and pain at baseline as well as daily assessments of sleep and pain. Participants wore an Actiwatch for 2 weeks and also completed 2 weeks' worth of sleep diaries and pain ratings. Hierarchical linear modeling analyses indicated that there was a bidirectional relationship between subjectively-reported sleep and pain, in that a day of increased pain was followed by a night of disrupted sleep, and a night of disrupted sleep was followed by increased pain the next day. Analyses on objective measures of sleep collected with actigraphy found no significant relationships among the sleep and pain variables. O'Brien and colleagues (2011) suggested that the subjective experience of sleep has a stronger relationship with reports of pain compared with more objective sleep measures.

There is a high prevalence of sleep difficulties in people with chronic pain, and especially FM. Although these studies differ on the directionality of the sleep and pain relationship, it is evident that there is a relationship between these two factors. Further research is needed to determine whether there is a stronger relationship between pain and subsequent sleep difficulties, or between sleep problems and subsequent pain, in people with FM. In addition, research is needed to explore the factors that predict individual differences in this sleep and pain relationship.

Moderators of the Sleep and Pain Relationship in Fibromyalgia

There are several factors that may aid in predicting the direction and the strength of the relationship between sleep and pain in FM. Unfortunately, there is almost no literature to guide the study of predictors, but I proposed to examine age, depression,

and pain catastrophizing. Theory and an occasional study suggest that these factors may predict the direction and strength of the sleep-pain relationship in FM.

A recent longitudinal study by Mork and Nilsen (2012) evaluated the relationship between self-reported sleep difficulties and risk of developing FM. Adult women who did not have a diagnosis of FM or any other chronic pain condition were included in the study and asked to indicate frequency of sleep problems. These same women were assessed approximately 10 years later for the presence of FM. The results indicate that the women who developed FM during the follow-up period reported a greater incidence of sleep difficulty at baseline compared with those who did not develop FM. When the women were stratified into older (≥ 45 years) and younger (20-44 years) age groups, the relative risk of FM development was greater for those women in the older group who reported sleep problems, compared with the women in the younger group (Mork & Nilsen, 2012). This study speaks to both direction and strength of the sleep-pain relationship. It suggests that sleep disturbance may result in FM-related pain, and older individuals with sleep difficulties may be at greater risk of developing FM than younger individuals. Additional studies provide evidence for significant sleep disruptions in older individuals with chronic pain (Lunde, Pallesen, Krangnes, & Nordhus, 2010) as well as associations of daily sleep and pain in older individuals with insomnia (Dzierzewski et al., 2010). These findings suggest that age is an important potential moderator in the sleep-pain relationship.

One of the prevalent comorbidities found with FM is depression, which may impact sleep fragmentation and sleep loss (Berger et al., 2007; Roehrs & Roth, 2005). A recent study by Miró, Martínez, Sánchez, Prados, and Medina (2011) evaluated the role

of sleep problems as a mediator of pain intensity on depression. Women with a FM diagnosis completed several self-report measures including pain, sleep, and depression. Compared with control participants, those with FM had significantly poorer sleep and greater levels of depression. Poor sleep quality was significantly correlated with greater pain intensity and depression, and pain intensity was also significantly correlated with depression levels (Miró et al., 2011). The study described earlier by O'Brien and colleagues (2011) evaluated the influence of depression on the relationship between sleep and pain in patients with chronic pain. In addition to finding a bidirectional relationship between sleep and pain, depressive symptoms also moderated this relationship, with participants who reported higher baseline depression levels having a stronger sleep-pain relationship than those with lower baseline levels of depression. Both of these studies speak to the strength of the sleep and pain relationship, suggesting that individuals with higher levels of self-reported depression symptoms have a stronger relationship between sleep and pain than individuals with lower levels of depression.

Pain catastrophizing impacts how individuals experience pain. Campbell, Edwards, and Quartana (2009) define pain catastrophizing "as a set of exaggerated and negative cognitive and emotional schema brought to bear during actual or anticipated painful stimulation." People who catastrophize tend to do three things: they ruminate about their pain, they magnify the severity of their pain, and they feel helpless to manage their pain (Sullivan, Bishop, & Pivik, 1995). As a result, individuals who catastrophize often attempt to avoid or escape painful experiences (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Pain catastrophizing has been associated with pain

sensitivity in experimental pain testing both with healthy individuals and those with chronic pain conditions (Edwards, Bingham, Bathon, & Haythornthwaite, 2006; Sullivan et al., 2001). A study by Severeijns, Vlaeyen, van den Hout, and Weber (2001) examined the relationship between pain catastrophizing and pain intensity and psychological distress in individuals with chronic pain. This study did not examine the sleep and pain relationship; instead, this study assessed chronic pain patients, who are known to have disturbed sleep, and discovered that those individuals who catastrophized experienced greater pain intensity and more psychological distress including depression. This suggests that individuals with chronic pain, who most likely have disturbed sleep as well, will experience greater pain intensity with catastrophizing compared with those who do not catastrophize about their pain.

Aims of this Study

The review of the literature provides evidence that sleep and pain are related. Experimental sleep manipulation studies resulted in alterations of pain perception, and chronic pain populations indicated that poor sleep often resulted in increased pain as well as the presence of pain resulting in disrupted sleep. The purpose of this study was to determine the direction of the sleep-pain relationship in a FM population as well as to uncover any potential factors that might predict individual differences in the sleep-pain relationship.

The prior literature with chronic pain populations relied almost exclusively on self-reports of sleep to evaluate the relationship between sleep and pain. Very few studies have evaluated daily sleep with an objective measure of actigraphy (O'Brien et al., 2011; Tang et al., 2012), and these have included only heterogeneous pain populations.

The goal of this study was to evaluate the relationship between sleep and pain using a large sample of individuals with FM. Sleep was objectively measured with 2 weeks of actigraphy along with subjective daily sleep diaries and pain ratings. Baseline self-report measures of depression, pain catastrophizing, negative affect and age were also evaluated to determine if any of these factors might explain individual differences in the sleep / pain relationship.

Aim 1. To determine the direction of the sleep-pain relationship for a sample of patients with FM. The current literature is unclear regarding direction of the relationship between sleep and pain. This study utilized both objective (actigraphy) and subjective (daily diary) measures to analyze this relationship. Separate analyses evaluated how one night's subjective and objective sleep variables influenced the next day's self-reported pain, as well as how one day's reported pain influenced the following night's subjective and objective sleep variables. Baseline predictor variables (depression, negative affect, pain, sleep quality, pain catastrophizing, and age) were also analyzed to determine their effect on the objective and subjective sleep variables as well as self-reported daily pain.

Aim 2. To determine what factors predict individual differences in the relationship between sleep and pain. Age, depression, negative affect, and pain catastrophizing were each evaluated as potential moderators of this relationship. Based upon the literature review, it was predicted that older individuals, as well as those who endorse more depression, negative affect, and pain catastrophizing would have a stronger relationship between sleep and pain.

CHAPTER 3

METHOD

Participants

Participants were 90 adults, aged 21 to 74, who were diagnosed with fibromyalgia (FM) and recruited as part of a National Institute of Health randomized clinical trial for FM interventions. Although this was a multi-center trial, including Wayne State University and the University of Michigan, the current data were taken solely from the Wayne State University site. There were 85 women (94.4%) and 5 men (5.6%), and they identified themselves as Caucasian (67.8%), African American (25.5%), or other (6.7%). Participants met the 1990 ACR criteria and/or the modified 2010 ACR FM criteria to be included in the study. Potential participants were excluded from the study if they had co-morbid autoimmune disorders (e.g., rheumatoid arthritis, systemic lupus erythematosus) or any other serious medical condition that could have impaired health status independently of FM including cardiopulmonary disorders (e.g. COPD, CHF), uncontrolled endocrine or allergic disorders, or malignancy within the previous 2 years. Other exclusion criteria included current psychotic disorder, dissociative identity disorder, alcohol or drug dependence in the past 2 years, or active suicide risk. Individuals with cognitive impairment or dementia, who were unable to fluently read or converse in English, or who had pending (or recently received) FM-related litigation, disability, or workman's compensation were also excluded.

Procedure

Screening

Participants recruited for the clinical trial were screened by telephone for FM symptoms, litigation/disability status, and co-morbid autoimmune disorders. Individuals who passed the telephone screening criteria and remained interested in participating were screened in person by the study coordinator.

An in-person screening was conducted with each participant at a convenient location (i.e., Detroit, Farmington Hills, or Macomb). Participants completed the written informed consent document, approved by the Human Investigation Committee of Wayne State University. The study coordinator obtained demographic and medical history information and ensured that the participants met the diagnostic criteria for FM (Wolfe et al., 1990; Wolfe et al., 2010). Tender point counts were assessed with the standard procedure for applying pressure in the Manual Tender Point Survey (MTPS) using the thumb pad of the examiner's dominant hand (Okifuji, Turk, Sinclair, Starz, & Marcus, 1997). The FM Symptom scale (FS) was also assessed by combining the Widespread Pain Index (WPI) and modified Symptom Severity scale (SS) as described in the modified ACR 2010 FM criteria (Wolfe et al., 2011). The participants completed a number of self-report measures not used in this dissertation and received \$50 for the screening visit.

Baseline evaluation

Participants were asked to return for an in-person evaluation session conducted by a research assistant. Each participant was assessed for changes in health, medications, disability claims, and recent stressors since the screening visit. Those

participants who still met study inclusion criteria completed a battery of self-report measures, including the Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977), Pain Catastrophizing Scale (PCS; Sullivan et al., 1995), Brief Pain Inventory (BPI; Cleeland & Ryan, 1994), Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989), Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) and others that were not used in this dissertation. The baseline evaluation also included experimental pain testing and heart rate variability (HRV) recording, neither of which was included in this dissertation. At the conclusion of the evaluation session, each participant was taught how to use an Actiwatch (Mini-Mitter, Respironics, Inc.) and was given one of these devices to wear for the following 2 weeks. In addition to wearing the Actiwatch, participants completed an activity log each day for the 2-week period (Appendix A). The activity log provided context for the movement data that was recorded with actigraphy. Participants were asked to record their average pain level for the entire day prior to bed each night. They also completed a set of morning questions upon awakening that pertained to the previous night's sleep. These questions included what time the participants attempted to fall asleep, how long they took to fall asleep, what time they woke to begin their day, and how refreshed they felt after their previous night's sleep. In addition, participants were requested to write down each time the Actiwatch was removed and for how long the device was off the wrist. The participants received \$100 for the evaluation visit and \$50 for returning the Actiwatch after 2 weeks.

Measures

Prospective Daily Measures

This dissertation assessed daily sleep and pain ratings recorded with the Actiwatch (Mini-Mitter, Respironics, Inc.) and activity log.

Actiwatch. The Actiwatch (Mini-Mitter, Respironics, Inc.) is a lightweight activity and movement monitor that is worn on the non-dominant wrist. It provides an objective behavioral measurement of sleep by recording activity throughout the day and night with an accelerometer. Participants wore this device for a 2-week period, and their activity data was translated into either “wake” or “sleep” based on a standard, validated algorithm that applies correction factors derived from polysomnography (Phillips Respironics, 2009). The Actiware scoring software is both reliable and valid for estimating sleep statistics when compared with traditional laboratory methods of sleep measurement (Cellini, Buman, McDevitt, Ricker, & Mednick, 2013). Data was recorded in 1-minute epochs throughout the study period, and actigraphy data was cleaned and scored with reference to diary data obtained from the activity log. For example, the information written in the activity log regarding what time the participants attempted to fall asleep and what time the participants woke to start their day aided in creating accurate rest and active intervals. The time and duration that the participants wrote down for when the watch was removed also aided in creating accurate exclusion intervals. The cleaned and scored actigraphy data produced several sleep and wake statistics. The variables of interest for this study were time in bed (TIB; the number of minutes in the nighttime rest interval), sleep onset latency (SOL; the number of minutes scored as wake from the beginning of the nighttime rest interval until the initiation of

sleep), total sleep time (TST; the number of minutes during the nighttime rest interval scored as sleep), wake after sleep onset (WASO; the number of minutes within the TST interval scored as wake), and sleep efficiency (SE; the percentage of scored total sleep time to the time in bed interval).

Daily activity log. In addition to wearing the Actiwatch, participants were asked to complete a daily activity log with subjective sleep information and to rate their average daily pain severity. The activity log produced several self-reported sleep and wake statistics including time in bed (SRTIB), sleep onset latency (SRSOL), total sleep time (SRTST), wake after sleep onset (SRWASO), sleep efficiency (SRSE), and refresh score (RS; an indication of how refreshed the participant felt after the previous night's sleep). Refresh score was measured on a scale from 0 to 10 where 0 represents "not at all refreshed" and 10 indicates "completely refreshed." All of the self-reported sleep variables were recorded in the morning upon awakening. Average pain was measured on a scale from 0 to 10 where 0 represents "no pain" and 10 indicates "pain as bad as you can imagine." This daily value was recorded in the activity log as well as in the Actiwatch prior to bed each night.

Baseline Measures

Depression. The Center for Epidemiological Studies Depression Scale (CES-D; Radloff, 1977) contains 20 items that measure depressive symptomatology. Participants were instructed to focus on their depressed mood during the past week when completing the items. Each item was scored from 0 to 3 where 0 indicates "rarely or none of the time" and 3 represents "most or almost all the time." The scale can be analyzed either as a continuous measure of depressive symptoms or as a dichotomous

measure, with scores of 16 or greater indicating symptom levels suggestive of depression. Normative studies of women scoring 16 or above on the CES-D have found rates between 8.7% and 17.4% (Knight, Williams, Mcgee & Olaman, 1997; Myers & Weissman, 1980; Roberts & Vernon, 1983). Several studies have found that the established cutoff of 16 for the CES-D overestimates the prevalence of depression in non-clinical samples (Beekman et al., 1997; Santor, Zuroff, Ramsay, Cervantes, & Palacios, 1995; Roberts, Lewinsohn, & Seeley, 1991). Therefore, a cutoff of 20 was used in this dissertation as an estimate of probable depression. The CES-D has also successfully been used to assess depression symptoms across wide age ranges (Lewinsohn, Seeley, Roberts, & Allen, 1997), an important consideration for the present dissertation. In this study, the CES-D demonstrated good reliability at baseline (Cronbach's alpha = 0.70).

Pain Catastrophizing. The Pain Catastrophizing Scale (PCS; Sullivan et al., 1995) contains 13 statements about pain. Participants were instructed to indicate the degree to which they have the thoughts and feelings listed when they experience pain, from 0 meaning "not at all" to 4 meaning "all the time." The total scale is dichotomous with scores of 30 or greater indicating a clinically relevant level of catastrophizing. For this dissertation, a mean score of the 13 items was used to assess overall pain catastrophizing, and this scale demonstrated excellent reliability at baseline (Cronbach's alpha = 0.93).

Brief Pain Inventory. A modified version of the Brief Pain Inventory (BPI; Cleeland & Ryan, 1994) was used in this dissertation. Participants were asked to answer four questions about their worst, least, and average pain over the past week, as

well as their current level of pain at the time of assessment. Each of these questions was scored on a 0 to 10 scale where 0 represents “no pain” and 10 indicates “pain as bad as you can imagine.” A mean score for all four pain severity items was calculated and utilized in analyses as an index of baseline pain, and this measure demonstrated good internal consistency (Cronbach’s alpha = 0.85).

Pittsburgh Sleep Quality Index. The Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989) contains 19 self-rated questions related to usual sleep habits during the past month. These items were combined to form seven “component” scores (i.e., subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction), all of which were scored on a 0 to 3 scale where 0 indicates “no difficulty” and 3 represents “severe difficulty.” The seven component scores were added to yield a global PSQI score with a range of 0 to 21 points that was utilized in analyses as an index of baseline sleep quality. In this study, global PSQI demonstrated an acceptable level of reliability at baseline (Cronbach’s alpha = 0.64).

Positive and Negative Affect Schedule. The Positive and Negative Affect Schedule (PANAS; Watson et al., 1988) contains 20 words and phrases that describe different feelings and emotions. Participants were instructed to indicate to what extent they felt each descriptor over the past few weeks on a scale from 1 to 5 where 1 represents “very slightly or not at all” and 5 indicates “extremely.” Mean positive affect and negative affect scores were calculated from the 10 words and phrases that loaded onto each construct. In this study, internal consistency was excellent for both positive affect (Cronbach’s alpha = 0.91) and negative affect (Cronbach’s alpha = 0.90). These

variables were utilized in analyses to determine if the depression variable (CES-D) assessed unique variance in addition to negative affect.

Data Analysis

The actigraphy data was cleaned and scored with reference to the daily activity log with subjective sleep information as noted previously. The resultant objective variables of time in bed (TIB), sleep onset latency (SOL), total sleep time (TST), wake after sleep onset (WASO), and sleep efficiency (SE) were used in the analyses. The subjective variables of time in bed (SRTIB), sleep onset latency (SRSOL), total sleep time (SRTST), wake after sleep onset (SRWASO), sleep efficiency (SRSE), and refresh score (RS) were also used in the analyses.

Multilevel modeling was utilized for this dissertation due to the hierarchical structure of the data (14 daily observations nested within persons). This methodology was able to account for within-person variation (a participant's daily deviation from their own 14-day mean) and between-person variation (each participant's deviation from the grand mean). In order to model both within- and between-person variation, each daily predictor variable was represented by two different variables (Hoffman & Stawski, 2009). For each predictor, a within-person variable (Level 1) represented the deviation from that individual's mean on a particular day. For each participant and predictor, a second between-person variable (Level 2) represented that individual's average for the predictor across all days (person mean), and the variable was centered so that 0 was the grand mean.

The data was checked for accuracy and frequency distributions. Several of the between-person predictor variables were skewed (average sleep onset latency, average

sleep efficiency, average self-report sleep onset latency, and average self-report wake after sleep onset). These variables were winsorized and average sleep onset latency and average self-report wake after sleep onset were $\log_{10}(x+1)$ transformed. Results of correlational analyses did not differ between the original and the winsorized and transformed variables. Therefore, only original variables were used in subsequent analyses.

Preliminary analyses assessed the relationships among the baseline predictor variables of depression, negative affect, pain catastrophizing, sleep quality, pain, and age. Analyses were also conducted to determine the relationships among the average objective and subjective sleep predictor variables.

Sleep/Pain Relationship

Two sets of analyses were conducted with multilevel modeling in order to determine the relationship between daily sleep and pain, each looking at the entire sample of participants. The first set of analyses described the relationship between the objective (actigraphy) and subjective (activity log) nightly sleep variables and the next day's average pain. The second set of analyses described the relationship between average daily pain and the subsequent night's objective and subjective sleep variables. These analyses addressed the hypothesis about the direction of the sleep / pain relationship. Analyses were also conducted with multilevel modeling in order to determine the relationship between the baseline predictor variables and sleep and pain outcomes.

Potential Moderator Analysis

To determine what factors may be driving the sleep and pain correlation across individuals, the potential moderator variables of age, depression, negative affect, and pain catastrophizing were built into the multilevel model to determine if they were significant predictors of the relationship between sleep and pain and between pain and sleep. Each potential moderator was added as a continuous variable and was further described as a dichotomous variable when a significant interaction term resulted (e.g., older vs. younger, high vs. low depression, high vs. low negative affect, and high vs. low pain catastrophizing).

CHAPTER 4

RESULTS

Preliminary Analyses

Individuals were included in the analyses if they completed at least 7 days of actigraphy and daily sleep and pain diaries. Adherence for actigraphy was very high, with 83 individuals completing at least 12 of the 14 days ($M = 13.24$ days, $SD = 1.34$). Daily sleep and pain diary adherence was also very high, with 86 individuals completing at least 12 of the 14 days ($M = 13.28$ days, $SD = 1.07$).

Analyses were conducted to determine the relationships among the six baseline predictors: depression (CES-D; $M = 20.20$, $SD = 10.97$), negative affect (PANAS; $M = 2.01$, $SD = 0.74$), pain catastrophizing (PCS; $M = 1.39$, $SD = 0.84$), sleep quality (PSQI; $M = 12.32$, $SD = 3.99$), pain (BPI; $M = 5.63$, $SD = 1.82$), and age ($M = 50.24$, $SD = 12.77$). Table 1 presents the correlations among the baseline predictors. As shown in this table, depression and negative affect were highly correlated, as expected ($p < .01$). The remainder of the self-reported variables (depression, negative affect, pain catastrophizing, sleep quality, and pain) were all positively correlated, with most correlations ranging from $r = .26$ to $r = .49$. Interestingly, baseline pain was not significantly related to baseline depression or negative affect. Age was inversely correlated with the other five predictors, with correlations ranging from $r = -.16$ to $r = -.30$.

Table 1

Correlations among the Baseline Predictor Variables

	2	3	4	5	6
1. Depression (CESD)	.79**	.49**	.42**	.14	-.16
2. Negative Affect (PANAS)	---	.33**	.26*	.06	-.21
3. Pain Catastrophizing (PCS)		---	.26*	.35**	-.30**
4. Sleep Quality (PSQI)			---	.35**	-.16
5. Pain (BPI)				---	-.19
6. Age					---

Note. All correlations were 2-tailed.

* $p < .05$. ** $p < .01$.

Analyses were also conducted to determine the relationships among the daily objective and subjective sleep outcome variables: time in bed (TIB; $M = 495.71$ min, $SD = 64.10$), sleep onset latency (SOL; $M = 16.81$ min, $SD = 15.80$), wake after sleep onset (WASO; $M = 65.92$ min, $SD = 24.47$), total sleep time (TST; $M = 401.16$ min, $SD = 57.52$), sleep efficiency (SE; $M = 80.47\%$, $SD = 8.85$), refresh score (RS; $M = 4.0$, $SD = 1.78$), self-report time in bed (SRTIB; $M = 500.73$ min, $SD = 65.42$), self-report sleep onset latency (SRSOL; $M = 27.37$ min, $SD = 20.31$), self-report wake after sleep onset (SRWASO; $M = 36.74$ min, $SD = 34.32$), self-report total sleep time (SRTST; $M = 440.05$ min, $SD = 53.85$), and self-report sleep efficiency (SRSE; $M = 88.09\%$, $SD = 7.39$). These descriptive analyses represent the average of each of these daily variables across the 14 days of data collection.

Table 2 presents the correlations among the average daily sleep variables. As shown in this table, the subjective measures were more highly correlated with one another than the objective measures. Importantly, the sleep variables were related to one another in similar patterns (e.g., as time in bed (TIB) increased, total sleep time (TST) also increased). Although not significant for either subjective or objective sleep,

the one exception to the predictable pattern of correlations was found between sleep onset latency (SOL) and TST; a negative relationship for objective sleep, and a positive relationship for subjective sleep.

The objective outcome variables of time in bed (TIB), wake after sleep onset (WASO), and total sleep time (TST), were significantly correlated with their subjective, self-reported counterparts ($p < .01$). In contrast, the objective outcome variables of sleep onset latency (SOL) and sleep efficiency (SE) were not significantly related to their subjective counterparts. Although self-reported refresh score was significantly related to other self-reported sleep variables, how refreshed one feels upon awakening was not significantly correlated with any objective sleep variables.

Table 2

Correlations among the Average Daily Sleep Variables

	2	3	4	5	6	7	8	9	10	11
1. Time in Bed	.17	.45[†]	.80[†]	-.03	-.20	.95[†]	.54[†]	.43[†]	.68[†]	-.42[†]
2. Sleep Onset Latency	---	.19	-.11	-.70[†]	-.03	.23*	.17	.01	.22*	-.05
3. Wake After Sleep Onset	---	---	-.10	-.54[†]	-.12	.50[†]	.29[†]	.46[†]	.25*	-.41[†]
4. Total Sleep Time	---	---	---	.39[†]	-.11	.72[†]	.41[†]	.21*	.57[†]	-.23*
5. Sleep Efficiency	---	---	---	---	.04	-.11	-.01	-.19	-.03	.14
6. Refresh Score	---	---	---	---	---	-.24*	-.28[†]	-.25*	-.04	.32
7. Self-report Time in Bed	---	---	---	---	---	---	.55[†]	.43[†]	.74[†]	-.41[†]
8. Self-report Sleep Onset Latency	---	---	---	---	---	---	---	.37[†]	.07	-.65[†]
9. Self-report Wake After Sleep Onset	---	---	---	---	---	---	---	---	-.18	-.87[†]
10. Self-report Total Sleep Time	---	---	---	---	---	---	---	---	---	.29[†]
11. Self-report Sleep Efficiency	---	---	---	---	---	---	---	---	---	---

Note. All correlations were 2-tailed.

* $p < .05$. [†] $p < .01$.

Each of the Level 1 (within-person) outcome variables of sleep and pain was then tested to determine if they differed significantly across individuals, that is, whether participants differed from one another on the daily sleep and pain variables, which is a prerequisite for further HLM analyses. These were conducted with the intercept-only model in HLM, which is the equivalent to a one-way, random-effects ANOVA model (Table 3).

Table 3

HLM One-Way Random Effects ANOVA Model

Equations	Example
Level 1 Model $Y_{ij} = \beta_{0j} + r_{ij}$	Level 1 Model $PS = \beta_{0j} + r_{ij}$
Level 2 Model $\beta_{0j} = \gamma_{00} + u_{0j}$	Level 2 Model $\beta_{0j} = \gamma_{00} + u_{0j}$
Mixed Model $Y_{ij} = \gamma_{00} + u_{0j} + r_{ij}$	Mixed Model $PS = \gamma_{00} + u_{0j} + r_{ij}$

Note. Equations are from Raudenbush and Bryk (2002); PS = Daily pain score

All of the objective and subjective sleep variables as well as daily pain were significantly different across individuals ($p < .001$). This signaled that further HLM analyses were indicated because there was sufficient variation among participants to warrant an analytic method that would assess daily variation within- and between-individuals. Intraclass correlation coefficients ($\rho = \tau_{00} / (\tau_{00} + \sigma^2)$) were also calculated to determine the percentage of the variance in each outcome variable that was between individuals (Table 4). The variation between individuals for the objective and subjective sleep variables of time in bed, sleep onset latency, wake after sleep onset, total sleep time, and sleep efficiency ranged from 15.3 to 45.1%, indicating that over half of the variance was found within individuals. In contrast, refresh score and daily pain score

had intraclass correlation coefficients that were 58.3 and 64.4% respectively, indicating that there was more variance between- than within-individuals for these variables.

Table 4

Intraclass Correlation Coefficients (ICC) for Each Outcome Variable

	Intraclass Correlation Coefficient (ICC) / % of variance
Objective Variables	
Time in Bed (TIB)	$\rho = 0.306 / 30.6\% *$
Sleep Onset Latency (SOL)	$\rho = 0.153 / 15.3\% *$
Wake After Sleep Onset (WASO)	$\rho = 0.357 / 35.7\% *$
Total Sleep Time (TST)	$\rho = 0.318 / 31.8\% *$
Sleep Efficiency (SE)	$\rho = 0.451 / 45.1\% *$
Subjective Variables	
Self-report Time in Bed (SRTIB)	$\rho = 0.321 / 32.1\% *$
Self-report Sleep Onset Latency (SRSOL)	$\rho = 0.272 / 27.2\% *$
Self-report Wake After Sleep Onset (SRWASO)	$\rho = 0.334 / 33.4\% *$
Self-report Total Sleep Time (SRTST)	$\rho = 0.184 / 18.4\% *$
Self-report Sleep Efficiency (SRSE)	$\rho = 0.267 / 26.7\% *$
Refresh Score (RS)	$\rho = 0.583 / 58.3\% *$
Daily Pain Score (PS)	$\rho = 0.644 / 64.4\% *$

* $p < .001$.

Primary Analyses

The intercept-only models indicated the appropriateness of further HLM analyses. Two sets of primary analyses were conducted. The first analyzed the daily objective and subjective sleep variables as predictors of next day's pain score. The second set of analyses evaluated daily pain on the next night's objective and subjective sleep variables. For each daily predictor variable, two different variables were entered into the model to account for both within- and between-person variation (Hoffman & Stawski, 2009; Table 5).

Table 5

HLM Random Coefficient Model

Equations	Example
Level 1 Model $Y_{ij} = \beta_{0j} + \beta_{1j}(X_{WP}) + r_{ij}$	Level 1 Model $PS = \beta_{0j} + \beta_{1j}(TIB_{WP}) + r_{ij}$
Level 2 Models $\beta_{0j} = \gamma_{00} + \gamma_{01}(X_{BP}) + u_{0j}$ $\beta_{1j} = \gamma_{10} + u_{1j}$	Level 2 Models $\beta_{0j} = \gamma_{00} + \gamma_{01}(TIB_{BP}) + u_{0j}$ $\beta_{1j} = \gamma_{10} + u_{1j}$
Mixed Model $Y_{ij} = \gamma_{00} + \gamma_{01}(X_{BP}) + \gamma_{10}(X_{WP}) + u_{0j} + u_{1j}(X_{WP}) + r_{ij}$	Mixed Model $PS = \gamma_{00} + \gamma_{01}(TIB_{BP}) + \gamma_{10}(TIB_{WP}) + u_{0j} + u_{1j}(TIB_{WP}) + r_{ij}$

Note. Equations are from Raudenbush and Bryk (2002); WP = within-person; BP = between-person; PS = Daily pain score; TIB = Time in Bed

Sleep Variables Predicting Next Day's Pain

Each objective (actigraphy) and subjective (activity log) sleep predictor variable was entered into the model individually. This included a within-person variable (Level 1) that indicated the deviation from that individual's mean on a particular day, and a between-person variable (Level 2) that represented that individual's average for the predictor across all days.

As shown in Table 6, objective and subjective sleep did not impact next day's pain in general (between-person), except for wake after sleep onset (WASO). In other words, there was not a significant difference between individuals for the relationship of average objective and subjective sleep variables (Level 2) on daily pain (Level 1), except for average WASO. Individuals who had more WASO on average experienced increased daily pain. Similarly, each participant's daily objective and subjective sleep did not impact their experience of pain the following day (within-person), except for self-reported refresh score (RS). Participants who reported less than their average refresh score upon awakening experienced more pain the next day.

Table 6

Relationship of Objective and Subjective Sleep Predictor Variables on Next Day Pain Ratings

	Daily Pain Outcome	
	Between-Person (Level 2)	Within-Person (Level 1)
Objective Predictor Variables		
Time in Bed (TIB)	-0.0008 (0.0026)	0.0002 (0.0005)
Sleep Onset Latency (SOL)	0.0057 (0.0092)	0.0008 (0.0014)
Wake After Sleep Onset (WASO)	0.0140 (0.0061)*	0.0001 (0.0015)
Total Sleep Time (TST)	-0.0046 (0.0030)	0.0002 (0.0005)
Sleep Efficiency (SE)	-0.0288 (0.0186)	-0.0008 (0.0048)
Subjective Predictor Variables		
Self-report Time in Bed (SRTIB)	-0.0006 (0.0026)	-0.0000 (0.0004)
Self-report Sleep Onset Latency (SRSOL)	-0.0066 (0.0083)	0.0015 (0.0012)
Self-report Wake After Sleep Onset (SRWASO)	0.0065 (0.0057)	0.0007 (0.0010)
Self-report Total Sleep Time (SRTST)	-0.0020 (0.0038)	-0.0002 (0.0004)
Self-report Sleep Efficiency (SRSE)	-0.0134 (0.0254)	-0.0024 (0.0034)
Refresh Score (RS)	-0.0884 (0.0996)	-0.1326 (0.0364)**

Note. Parameter estimates are unstandardized beta coefficients, with SE in parentheses.

** $p = .001$, * $p < .05$.

Daily Pain Predicting Next Night's Sleep

Each objective (actigraphy) and subjective (activity log) sleep variable was entered into the model individually as the outcome variable. Daily pain was then added as the predictor variable, including a within-person variable (Level 1) that indicated the deviation from that individual's mean pain score on a particular day, and a between-person variable (Level 2) that represented that individual's average pain score across all days.

As shown in Tables 7 and 8, average daily pain did not impact the next night's objective and subjective sleep in general (between-person), except for wake after sleep onset (WASO). Individuals who reported greater daily pain on average experienced

increased nightly WASO. Similarly, each participant's daily pain did not impact their objective and subjective sleep the following night (within-person), except for self-reported sleep onset latency (SRSOL). Participants who reported more than their average pain one day experienced a greater latency to sleep the following night.

Table 7

Relationship of Daily Pain Score Predictor on Objective Sleep Outcome Variables

	Time in Bed (TIB)	Sleep Onset Latency (SOL)	Wake After Sleep Onset (WASO)	Total Sleep Time (TST)	Sleep Efficiency (SE)
Daily Pain Score _{BP}	-1.56 (3.83)	0.32 (0.87)	2.99 (1.29)*	-6.03 (3.81)	-0.79 (0.45)
Daily Pain Score _{WP}	-0.24 (2.25)	0.38 (0.75)	0.62 (0.81)	0.03 (2.09)	-0.01 (0.21)

Note. Parameter estimates are unstandardized beta coefficients, with *SE* in parentheses. BP = Between-person (Level 2); WP = Within-person (Level 1)

* $p < .05$.

Table 8

Relationship of Daily Pain Score Predictor on Subjective Sleep Outcome Variables

	Self-Report Time in Bed (SRTIB)	Self-Report Sleep Onset Latency (SRSOL)	Self-Report Wake After Sleep Onset (SRWASO)	Self-Report Total Sleep Time (SRTST)	Self-Report Sleep Efficiency (SRSE)	Refresh Score (RS)
Daily Pain Score _{BP}	-1.28 (3.99)	-0.68 (1.35)	3.06 (2.20)	-1.98 (3.51)	-0.24 (0.49)	-0.09 (0.11)
Daily Pain Score _{WP}	0.05 (2.70)	1.35 (0.68)*	-0.01 (1.08)	-0.94 (2.83)	-0.18 (0.25)	-0.03 (0.04)

Note. Parameter estimates are unstandardized beta coefficients, with *SE* in parentheses. BP = Between-person (Level 2); WP = Within-person (Level 1)

* $p < .05$.

Level 2 Baseline Measures Predicting Daily Pain Outcome across Individuals

In addition to evaluating the impact of sleep variables on next day's pain, Level 2 baseline measures of depression, negative affect, sleep quality, age, and pain

catastrophizing, were each built into the intercepts- and slopes-as-outcomes model individually to determine their effect on daily pain across individuals (Table 9).

Table 9

HLM Intercepts- and Slopes-as-Outcomes Model with a Level 2 Predictor Only

Equations	Example
Level 1 Model $Y_{ij} = \beta_{0j} + r_{ij}$	Level 1 Model $PS = \beta_{0j} + r_{ij}$
Level 2 Model $\beta_{0j} = \gamma_{00} + \gamma_{01}(W_j) + u_{0j}$	Level 2 Model $\beta_{0j} = \gamma_{00} + \gamma_{01}(CESD) + u_{0j}$
Mixed Model $Y_{ij} = \gamma_{00} + \gamma_{01}(W_j) + u_{0j} + r_{ij}$	Mixed Model $PS = \gamma_{00} + \gamma_{01}(CESD) + u_{0j} + r_{ij}$

Note. Equations are from Raudenbush and Bryk (2002); PS = Daily pain score; CESD = Depression total score

As shown in Table 10, baseline depression, negative affect, and sleep quality did not significantly predict daily pain across individuals. Age was a significant predictor of daily pain, such that individuals who were older reported less pain, on average, than those who were younger. Baseline pain catastrophizing was also a significant predictor of daily pain, such that individuals who reported greater levels of catastrophizing about their pain reported more pain, on average, than those individuals who reported less catastrophizing.

Table 10

Relationship of Baseline Predictors on Daily Pain Outcome across Individuals

	Daily Pain Score
Depression (CESD)	0.02 (0.02)
Negative Affect (PANAS)	0.15 (0.28)
Sleep Quality (PSQI)	0.09 (0.04)
Age	-0.05 (0.01)**
Pain Catastrophizing (PCS)	0.74 (0.18)***

Note. Parameter estimates are unstandardized beta coefficients, with *SE* in parentheses.

*** $p < .001$, ** $p = .001$

*Level 2 Baseline Measures Predicting Objective and Subjective Sleep Outcome**Variables across Individuals*

In addition to evaluating the impact of daily pain scores on the next night's sleep variables, Level 2 baseline measures of depression, negative affect, pain catastrophizing, pain, and age were each built into the intercepts- and slopes-as-outcomes model individually to determine their effect on objective and subjective sleep variables across individuals.

As shown in Tables 11 and 12, baseline negative affect did not significantly predict objective or subjective sleep outcome variables across individuals. Depression was a significant predictor of objective time in bed (TIB) as well as most of the subjective sleep variables (i.e., self-reported time in bed, self-reported sleep onset latency, self-reported wake after sleep onset, self-reported sleep efficiency, and refresh score). Individuals who reported higher levels of baseline depression spent more time in bed objectively, and reported that they spent more time in bed, took longer to fall asleep, spent more time awake during the night, and had a lower sleep efficiency than

those individuals who reported lower levels of depression. In addition, the individuals who reported higher levels of baseline depression reported feeling less refreshed upon awakening than those who reported lower levels of depression.

Baseline pain was a significant predictor of refresh score (RS), such that individuals who reported greater baseline pain also reported feeling less refreshed upon awakening than individuals who reported lower levels of baseline pain. Age was a significant predictor of objective sleep onset latency (SOL), such that individuals who were older took longer to fall asleep than those who were younger. Lastly, baseline pain catastrophizing was a significant predictor of objective wake after sleep onset (WASO) and subjective time in bed (SRTIB). Individuals who reported greater levels of catastrophizing about their pain spent more time awake during the night and reported spending more time in bed than those individuals who reported lower levels of pain catastrophizing.

Table 11

Relationship of Baseline Predictors on Objective Sleep Variables across Individuals

	Time in Bed (TIB)	Sleep Onset Latency (SOL)	Wake After Sleep Onset (WASO)	Total Sleep Time (TST)	Sleep Efficiency (SE)
Depression (CESD)	1.57 (0.69)*	-0.10 (0.11)	0.41 (0.24)	1.31 (0.67)	0.01 (0.07)
Negative Affect (PANAS)	7.48 (10.77)	-3.37 (2.08)	-0.48 (3.71)	13.95 (9.41)	1.74 (0.98)
Pain (BPI)	1.35 (3.42)	0.44 (0.82)	2.29 (1.33)	-2.41 (3.84)	-0.56 (0.48)
Age	0.27 (0.48)	0.30 (0.11)[†]	-0.07 (0.20)	0.12 (0.47)	-0.04 (0.06)
Pain Catastrophizing (PCS)	8.25 (5.46)	-0.93 (1.67)	6.09 (2.97)*	2.54 (6.03)	-0.90 (0.84)

Note. Parameter estimates are unstandardized beta coefficients, with *SE* in parentheses.

* $p < .05$, [†] $p < .01$.

Table 12

Relationship of Baseline Predictors on Subjective Sleep Variables across Individuals

	Self-Report Time in Bed (SRTIB)	Self-Report Sleep Onset Latency (SRSOL)	Self-Report Wake After Sleep Onset (SRWASO)	Self- Report Total Sleep Time (SRTST)	Self-Report Sleep Efficiency (SRSE)	Refresh Score (RS)
Depression (CESD)	1.69 (0.73)*	0.63 (0.27)*	0.84 (0.42)*	0.39 (0.57)	-0.19 (0.09)*	-0.03 (0.02)*
Negative Affect (PANAS)	7.10 (11.32)	5.67 (3.74)	3.04 (5.98)	0.57 (8.94)	-0.82 (1.23)	-0.22 (0.24)
Pain (BPI)	2.26 (3.39)	0.67 (1.07)	3.78 (1.95)	-1.33 (3.32)	-0.74 (0.45)	-0.25 (0.10)*
Age	0.30 (0.48)	0.03 (0.14)	-0.26 (0.30)	0.47 (0.48)	0.03 (0.07)	0.02 (0.02)
Pain Catastrophizing (PCS)	11.82 (5.66)*	1.65 (1.92)	3.62 (4.80)	6.81 (6.88)	-0.68 (1.07)	-0.31 (0.26)

Note. Parameter estimates are unstandardized beta coefficients, with *SE* in parentheses.

* $p < .05$.

Moderator Analyses

Two sets of moderator analyses were conducted. The first analyzed the potential moderator variables of age, depression, negative affect, and pain catastrophizing to determine if each individually predicted the relationship between daily objective and subjective sleep variables and next day's pain. The second set of analyses evaluated the same potential moderator variables to determine if each individually predicted the relationship between daily pain and the next night's objective and subjective sleep variables. Each potential moderator was individually added to the intercepts- and slopes-as-outcomes model as a continuous variable, and was further described as a dichotomous variable when a significant interaction term resulted (Preacher, Curran, &

Bauer, 2006; Table 13). Only those moderator analyses that resulted in a significant interaction are presented below.

Table 13

HLM Intercepts- and Slopes-as-Outcomes Model

Equations	Example
Level 1 Model $Y_{ij} = \beta_{0j} + \beta_{1j}(X_{WP}) + r_{ij}$	Level 1 Model $PS = \beta_{0j} + \beta_{1j}(TIB_{WP}) + r_{ij}$
Level 2 Models $\beta_{0j} = \gamma_{00} + \gamma_{01}(X_{BP}) + \gamma_{02}(W_j) + u_{0j}$ $\beta_{1j} = \gamma_{10} + \gamma_{11}(W_j) + u_{1j}$	Level 2 Models $\beta_{0j} = \gamma_{00} + \gamma_{01}(TIB_{BP}) + \gamma_{02}(Age) + u_{0j}$ $\beta_{1j} = \gamma_{10} + \gamma_{11}(Age) + u_{1j}$
Mixed Model $Y_{ij} = \gamma_{00} + \gamma_{01}(X_{BP}) + \gamma_{02}(W_j) + \gamma_{10}(X_{WP}) + \gamma_{11}(W_j)(X_{WP}) + u_{0j} + u_{1j}(X_{WP}) + r_{ij}$	Mixed Model $PS = \gamma_{00} + \gamma_{01}(TIB_{BP}) + \gamma_{02}(Age) + \gamma_{10}(TIB_{WP}) + \gamma_{11}(Age)(TIB_{WP}) + u_{0j} + u_{1j}(TIB_{WP}) + r_{ij}$

Note. Equations are from Raudenbush and Bryk (2002); WP = within-person; BP = between-person; PS = Daily pain score; TIB = Time in Bed

Moderators of the Relationship between Sleep and Next Day's Pain

Age was a significant moderator of the relationship between subjective sleep onset latency (SOL) and the next day's pain. Figure 1 indicates that there is a stronger, positive relationship between subjective SOL and next day's pain for individuals who are older ($\beta = 0.004$ (0.002), $p=0.04$), compared with the negative relationship between subjective SOL and next day's pain for younger individuals ($\beta = -0.002$ (0.002), $p=0.52$). In addition to the fact (noted above in Table 10), that older individuals have significantly lower daily pain than younger people, the interaction indicates that this finding is more pronounced for individuals with shorter subjective sleep onset latency the previous night compared with those who report longer sleep onset latency.

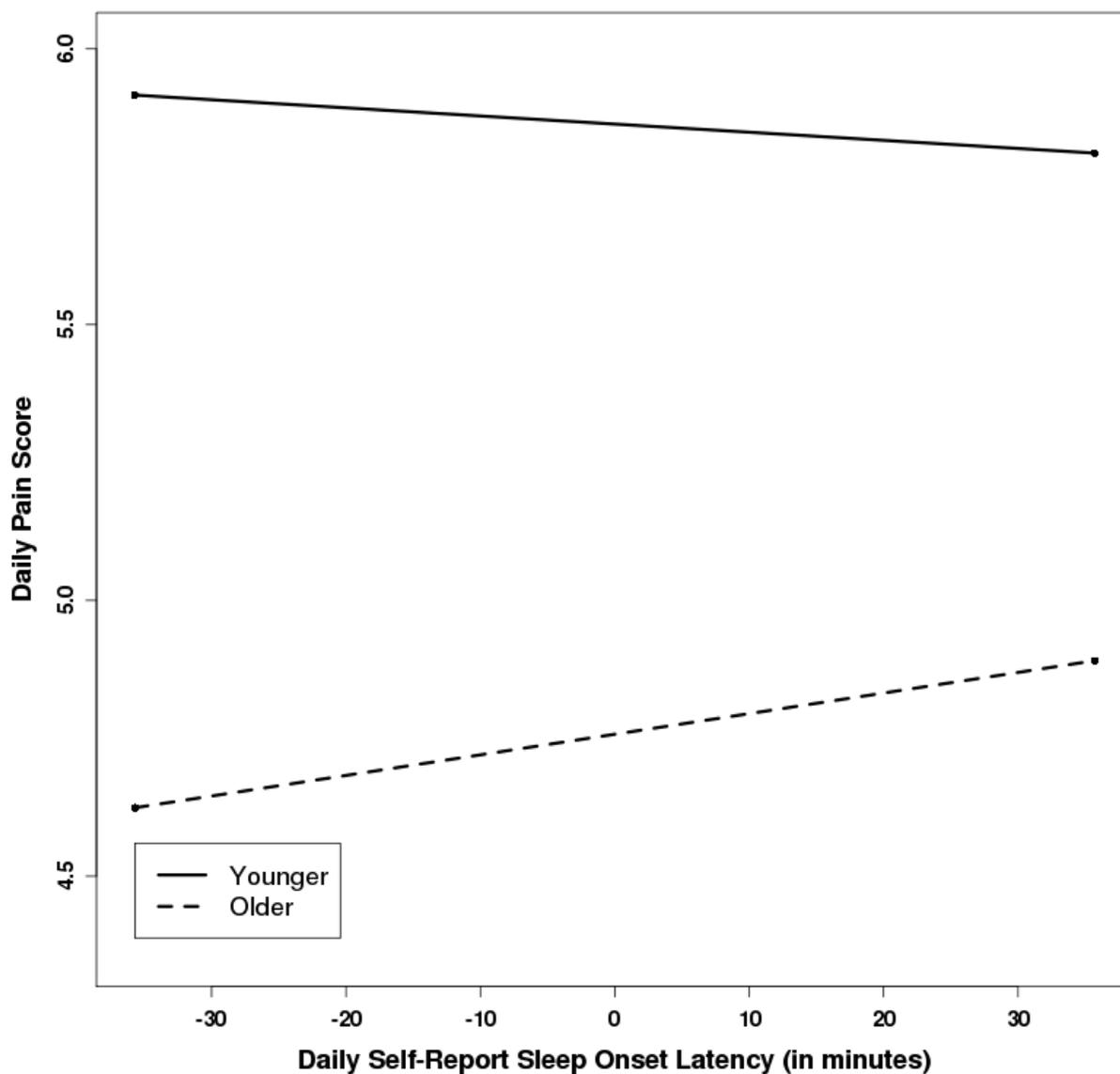


Figure 1. Interaction of daily self-report sleep onset latency (SOL) and age on the next day's pain score. Younger and older participants were -1SD and +1SD of the mean respectively. Daily self-report sleep onset latency was centered around a mean of zero.

Depression was a significant moderator of the relationship between refresh score and next day's pain. Figure 2 indicates that there is a stronger, negative relationship between self-reported refreshing quality of sleep and next day's pain for individuals who reported higher levels of baseline depression ($\beta = -0.23$ (0.05), $p=0.0$) than for those

who reported lower levels of baseline depression ($\beta = -0.04 (0.05), p=0.50$). In addition to the fact (noted above in Table 10), that individuals who reported higher levels of depression have greater daily pain than those who reported lower levels of depression, the interaction indicates that this finding is more pronounced for individuals who report less refreshing quality of sleep from the previous night compared with those who report more refreshing quality of sleep.

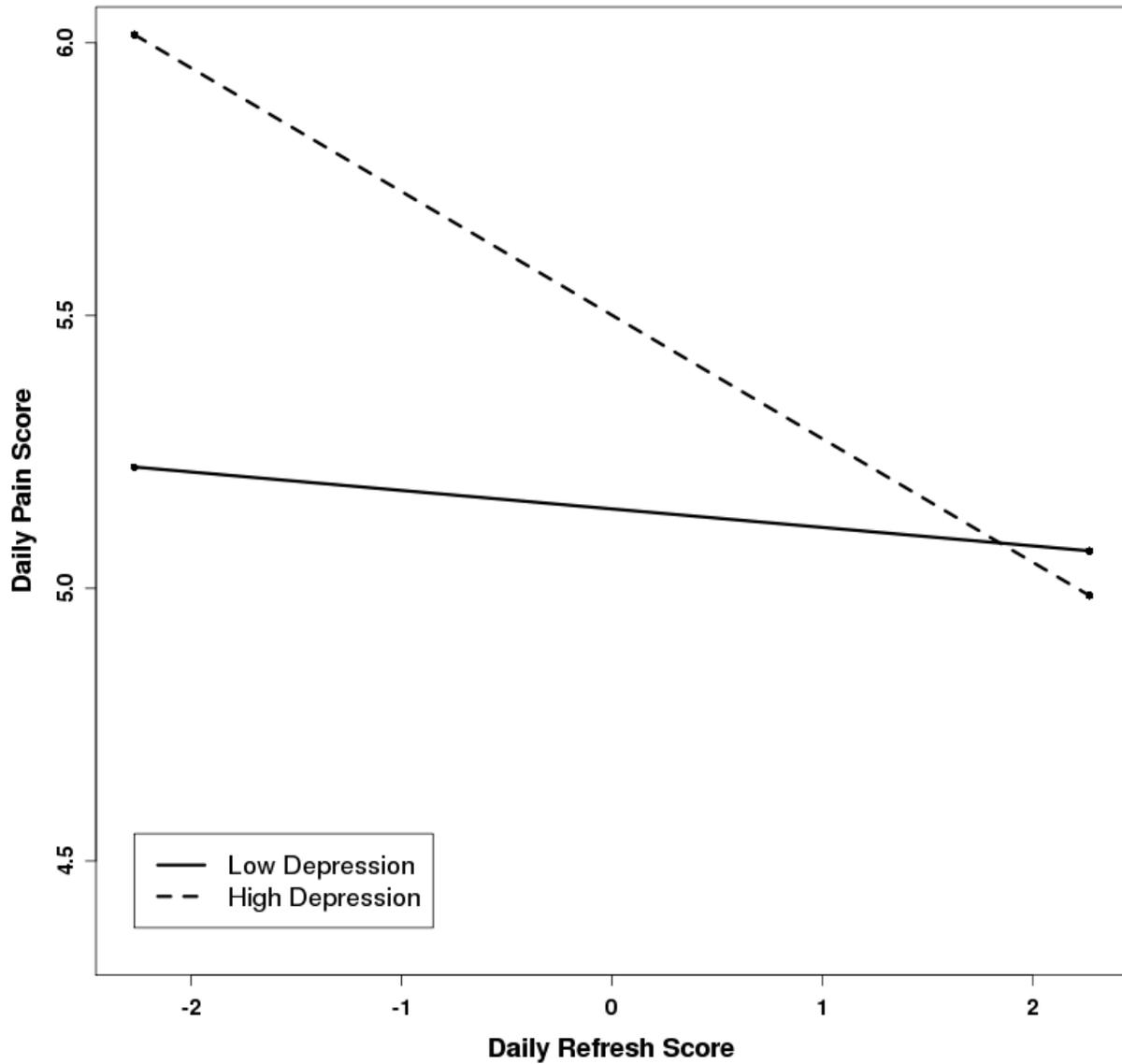


Figure 2. Interaction of daily refresh score and baseline depression on the next day's pain score. Low and high depression were -1SD and +1SD of the mean respectively. Daily refresh score was centered around a mean of zero.

Negative affect was also a significant moderator of the relationship between refresh score and next day's pain. Similar to the findings with depression, Figure 3 indicates that there is a stronger, negative relationship between self-reported refreshing quality of sleep and next day's pain for individuals who reported higher levels of

baseline negative affect ($\beta = -0.22$ (0.05), $p=0.0$) than for those who reported lower levels of baseline negative affect ($\beta = -0.04$ (0.05), $p=0.41$). In addition to the fact (noted above in Table 10), that individuals who reported higher levels of negative affect have greater daily pain than those who reported lower levels of negative affect, the interaction indicates that this finding is more pronounced for individuals who report less refreshing quality of sleep from the previous night compared with those who report more refreshing quality of sleep.

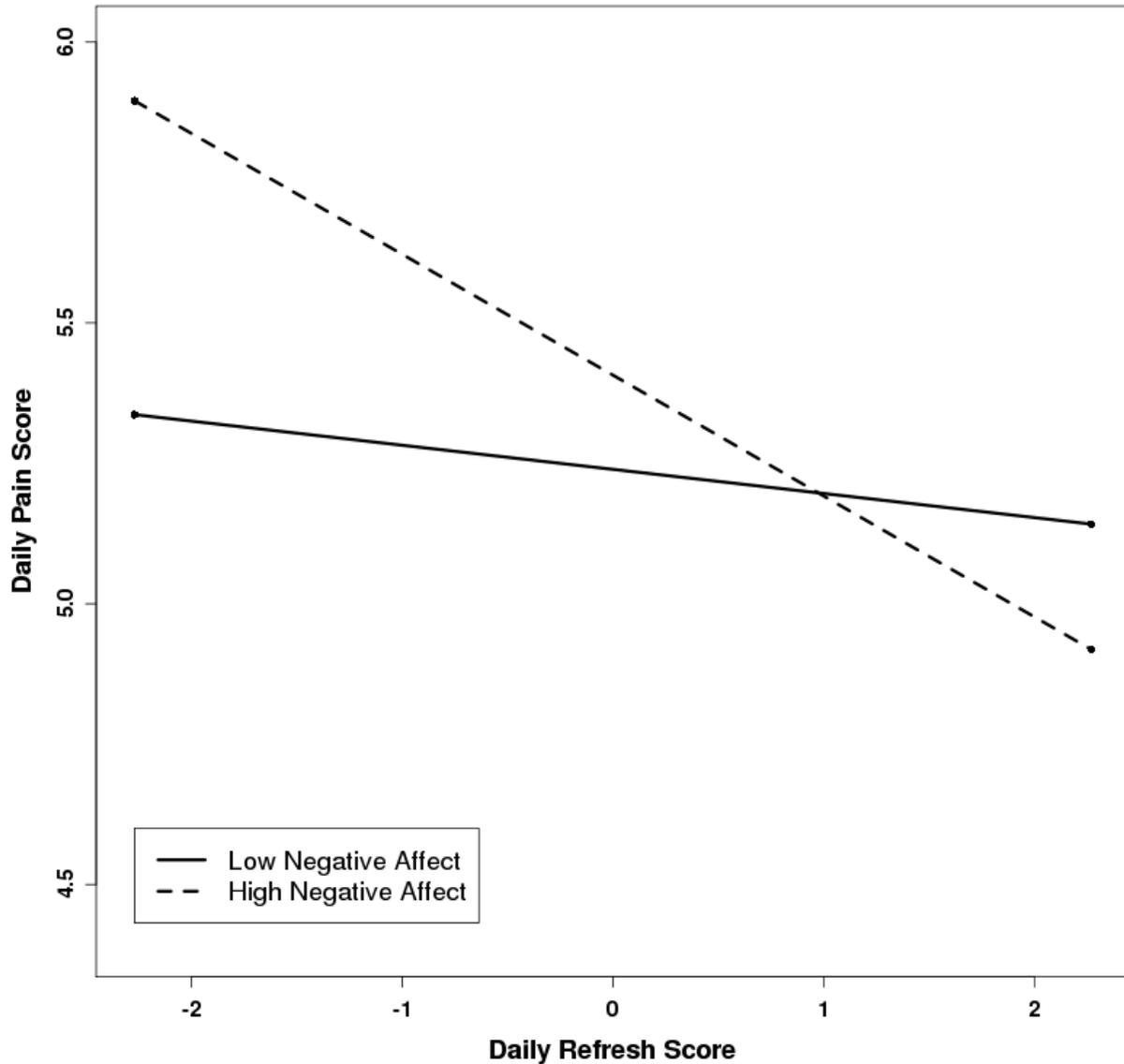


Figure 3. Interaction of daily refresh score and baseline negative affect on the next day's pain score. Low and high negative affect were -1SD and +1SD of the mean respectively. Daily refresh score was centered around a mean of zero.

Negative affect was a significant moderator of the relationship between objective total sleep time (TST) and next day's pain. Figure 4 indicates that there is a small, negative relationship between objective TST and next day's pain for individuals who reported higher levels of baseline negative affect ($\beta = -0.0007$ (0.0007), $p=0.30$),

compared with a small, positive relationship between objective TST and next day's pain for those who reported lower levels of baseline negative affect ($\beta = 0.001$ (0.0008), $p=0.13$). In addition to the fact (noted above in Table 10), that individuals who reported higher levels of negative affect have greater daily pain than those who reported lower levels of negative affect, the interaction indicates that this finding is more pronounced for individuals who report less total sleep time the previous night compared with those who report more total sleep time.

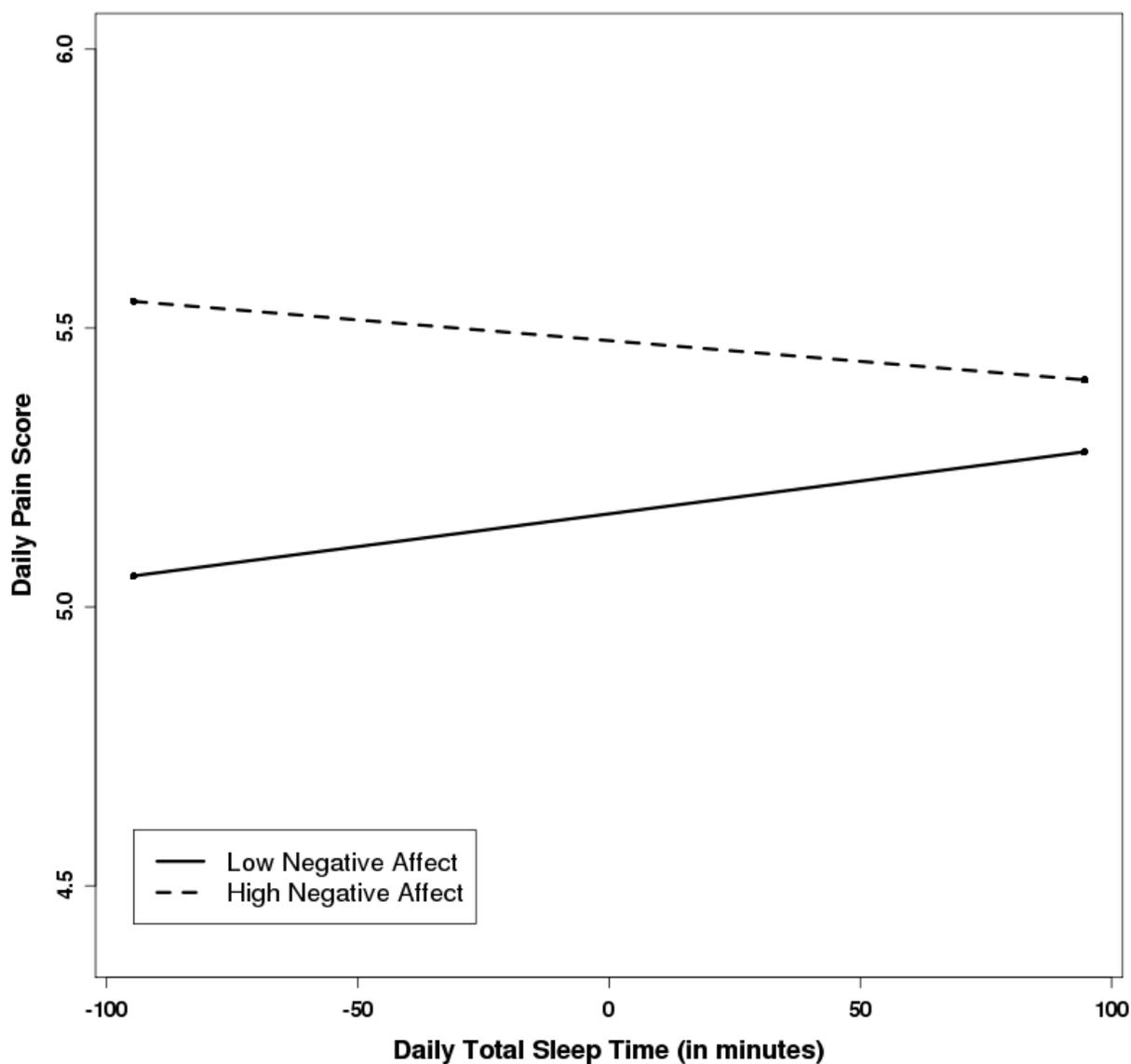


Figure 4. Interaction of objective daily total sleep time (TST) and baseline negative affect on the next day's pain score. Low and high negative affect were -1SD and +1SD of the mean respectively. Daily total sleep time was centered around a mean of zero.

Pain catastrophizing was found to be a significant moderator of the relationship between objective sleep efficiency (SE) and next day's pain. Figure 5 indicates that there is a small, negative relationship between objective SE and next day's pain for individuals who reported higher levels of baseline pain catastrophizing ($\beta = -0.0099$

(0.007), $p=0.16$), and an almost equal positive relationship between objective SE and next day's pain for those who reported lower levels of baseline pain catastrophizing ($\beta = 0.0097$ (0.008), $p=0.24$). In addition to the fact (noted above in Table 10), that individuals who reported higher levels of pain catastrophizing have greater daily pain, the interaction indicates that this finding is more pronounced for individuals with less objective sleep efficiency the previous night compared with those who have greater objective sleep efficiency.

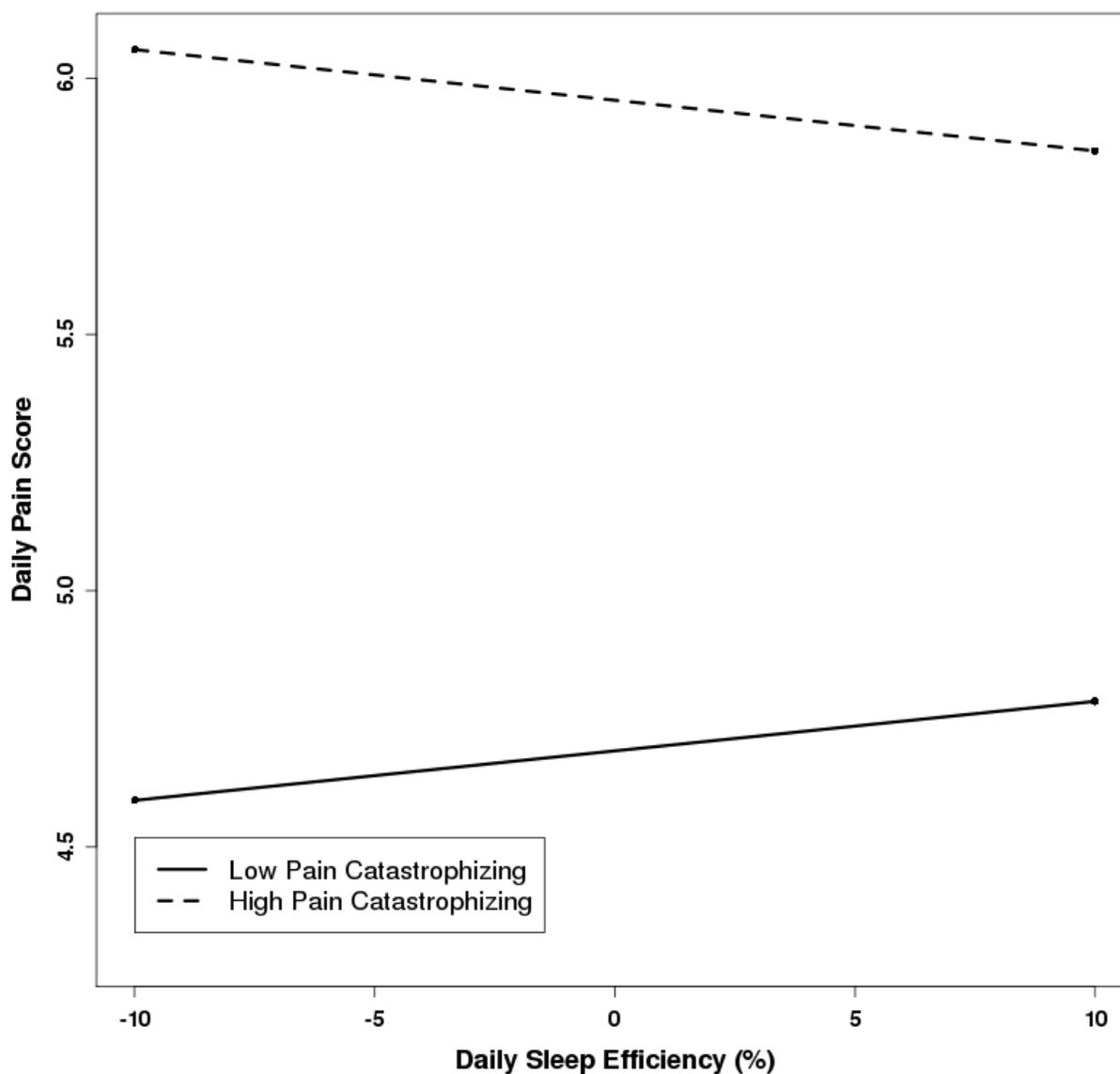


Figure 5. Interaction of objective daily sleep efficiency (SE) and baseline pain catastrophizing on the next day's pain score. Low and high pain catastrophizing were -1SD and +1SD of the mean respectively. Daily sleep efficiency was centered around a mean of zero.

Moderators of the Relationship between Daily Pain and the Next Night's Sleep

Age was a significant moderator of the relationship between daily pain and the next night's subjective sleep onset latency (SOL). Figure 6 indicates that there is a

stronger, positive relationship between daily pain and the next night's subjective SOL for individuals who are older ($\beta = 2.49$ (1.10), $p=0.03$) compared with those who are younger ($\beta = 0.09$ (1.16), $p=0.94$). In addition to the fact (noted above in Table 12), that older individuals have greater self-reported sleep onset latency than younger people, the interaction indicates that this finding is more pronounced for individuals who report higher levels of pain the previous day compared with those who report lower levels of pain.

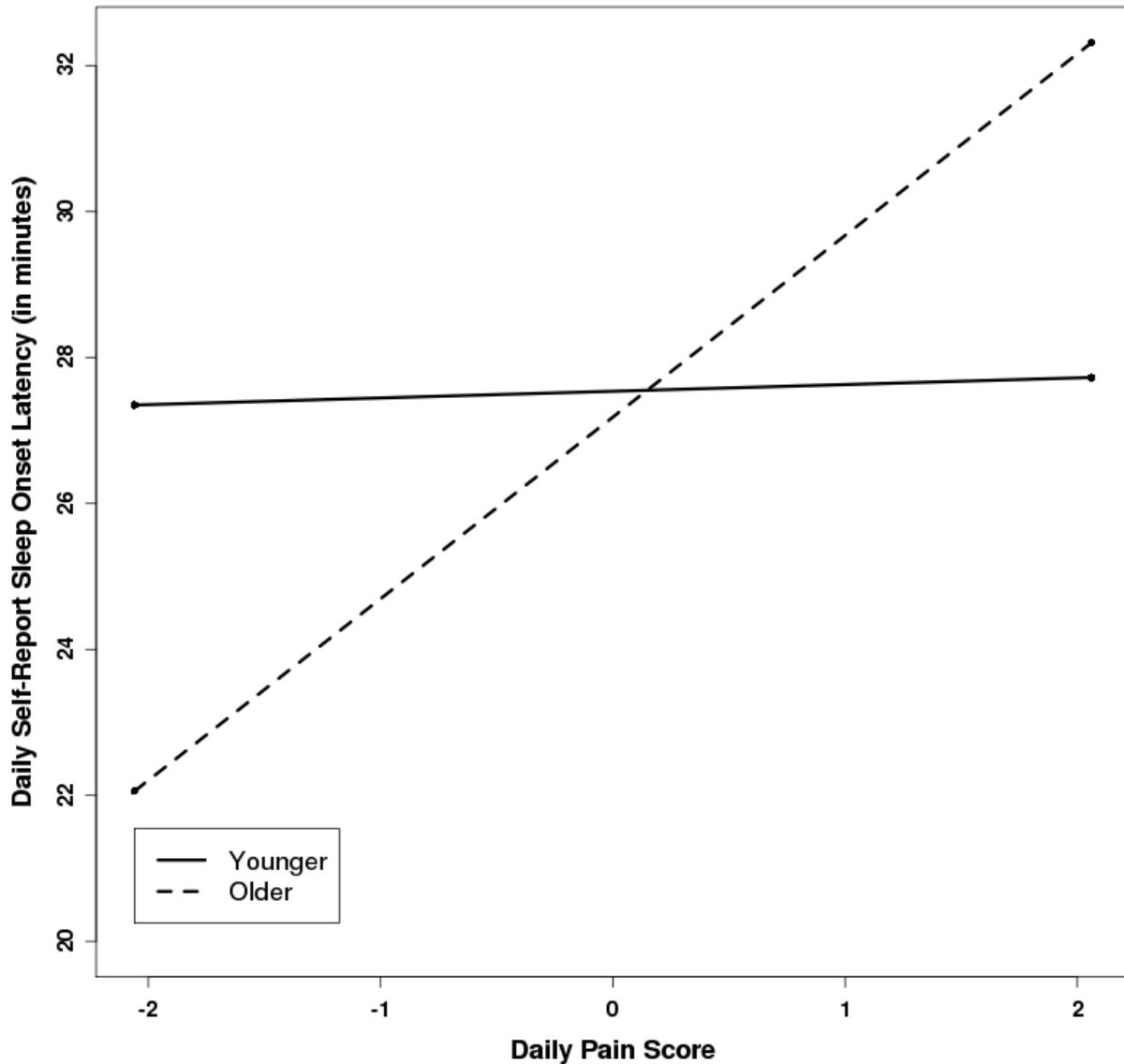


Figure 6. Interaction of daily pain score and age on the next night's self-report sleep onset latency (SOL). Younger and older participants were $-1SD$ and $+1SD$ of the mean respectively. Daily pain score was centered around a mean of zero.

Depression was a significant moderator of the relationship between daily pain and refresh score upon awakening the next morning. Figure 7 indicates that there is a stronger, negative relationship between daily pain and the next night's refreshing quality of sleep for individuals who reported higher levels of baseline depression ($\beta = -0.13$

(0.06), $p=0.03$), compared with the positive relationship between daily pain and next night's refreshing quality of sleep for individuals who reported lower levels of baseline depression ($\beta = 0.07$ (0.06), $p=0.24$). In addition to the fact (noted above in Table 12), that individuals who reported higher levels of depression also reported significantly lower refreshing quality of sleep than those who reported lower levels of depression, the interaction indicates that this finding is more pronounced for individuals who report higher levels of pain the previous day compared with those who report lower levels of pain.

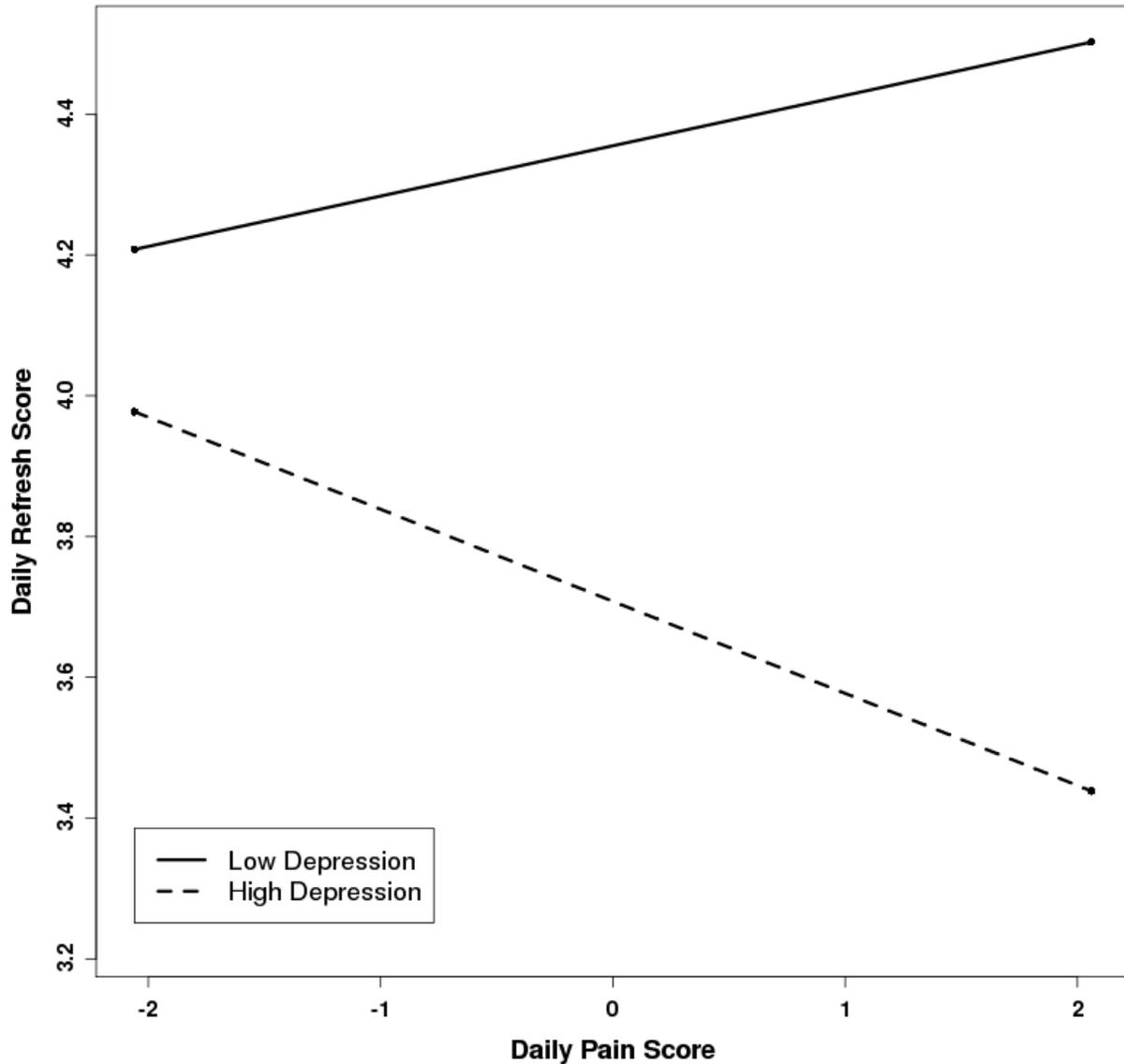


Figure 7. Interaction of daily pain score and baseline depression on the next day's refresh score. Low and high depression were -1SD and +1SD of the mean respectively. Daily pain score was centered around a mean of zero.

Lastly, pain catastrophizing was found to be a significant moderator of the relationship between daily pain and the next night's objective sleep efficiency (SE). Figure 8 indicates that there is a negative relationship between daily pain and objective SE for individuals who reported higher levels of baseline pain catastrophizing ($\beta = -0.45$

(0.28), $p=0.11$), compared with the positive relationship between daily pain and objective SE for individuals who reported lower levels of baseline catastrophizing ($\beta = 0.42$ (0.29), $p=0.15$). In addition to the fact (noted above in Table 11), that individuals who reported higher levels of pain catastrophizing have lower objective sleep efficiency than those who reported decreased levels of pain catastrophizing, the interaction indicates that this finding is more pronounced for individuals who report higher levels of pain the previous day compared with those who report lower levels of pain.

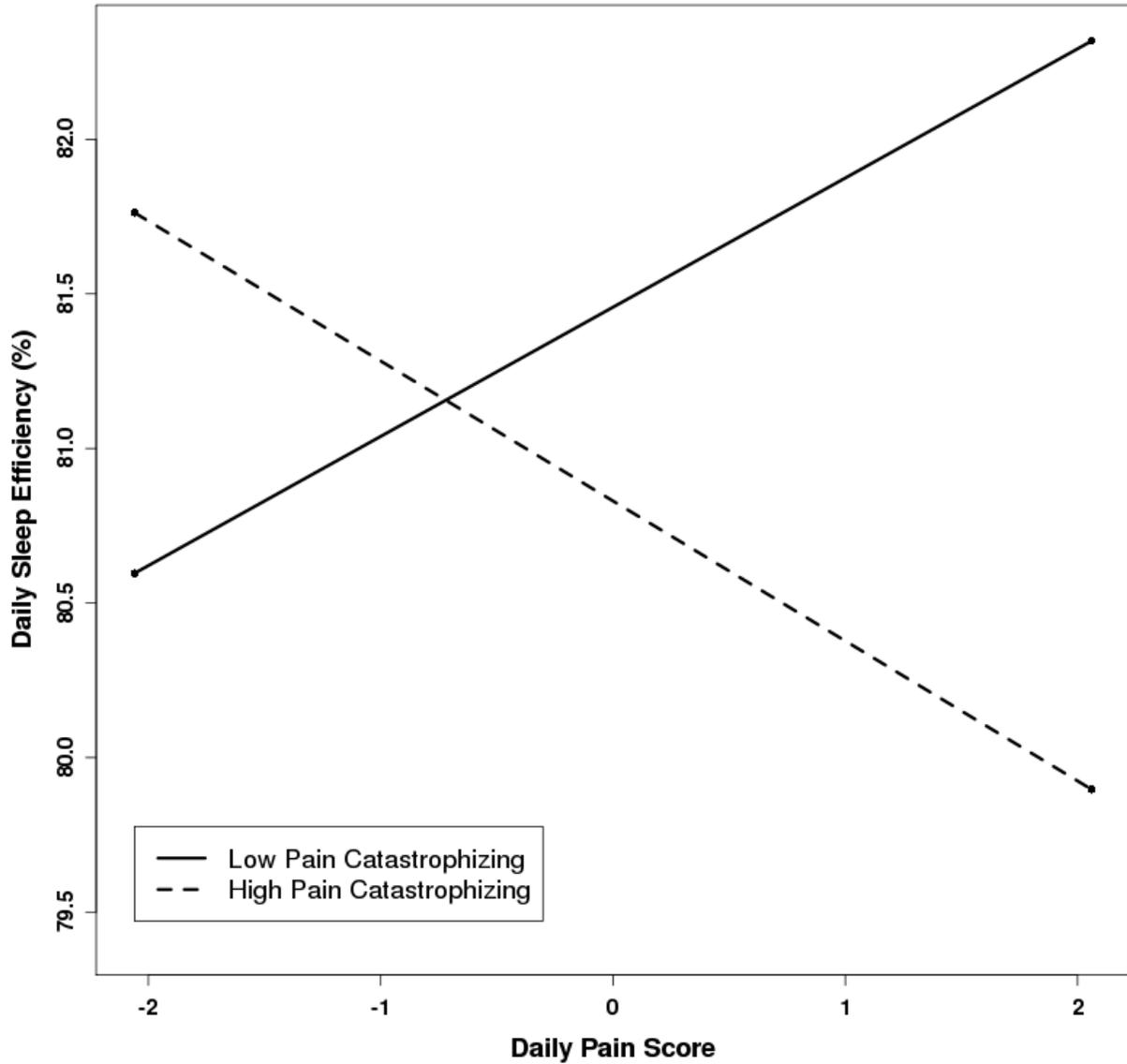


Figure 8. Interaction of daily pain score and baseline pain catastrophizing on the next night's objective sleep efficiency (SE). Low and high pain catastrophizing were -1SD and +1SD of the mean respectively. Daily pain score was centered around a mean of zero.

CHAPTER 5

DISCUSSION

This dissertation sought to examine the daily relationship between sleep and pain in a large population of chronic pain patients with fibromyalgia (FM), as well as the factors that may explain individual differences in this relationship. This study utilized actigraphy, an objective measurement of sleep, in addition to self-report daily sleep diaries. Few studies that have examined the daily sleep and pain relationship have included an objective measurement of sleep (O'Brien et al., 2011; Tang et al., 2012). In addition, these previous studies have failed to show a clear relationship between pain and objectively measured sleep, which may, in part, be due to evaluating populations that are very heterogeneous with respect to type of pain condition. In this study, participants with FM wore an actiwatch for two weeks while also completing daily diaries about their sleep and pain. The constructs of depression, negative affect, pain, sleep disturbance, and pain catastrophizing were assessed at baseline and tested as moderators of the daily sleep-pain relationship.

As mentioned previously, poor sleep quality is highly prevalent among individuals with FM, and is often characterized as light and unrefreshing (Moldofsky, 2008). An extensive literature of polysomnographic sleep studies has identified common sleep disturbances in this population including delayed sleep onset (Branco, Atalaia, & Paiva, 1994; Horne & Shackell, 1991), increased arousals (Jennum, Drewes, Andreasen, & Nielsen, 1993), and lower total sleep time (Harding & Lee-Chiong, 2006) compared with healthy controls. Self-report is the most common methodology of studying sleep in FM through the use of sleep diaries and questionnaires such as the PSQI (Buysse et al.,

1989). Similar to the study by O'Brien and colleagues (2011), the FM population in this dissertation reported longer sleep onset latency and shorter wake after sleep onset than was objectively measured with actigraphy. Total sleep time and sleep efficiency in the current study were similar to prior FM samples as well as healthy controls, suggesting that the nonrestorative aspect of sleep in the FM population may be due to perceived quality of sleep rather than amount of actual sleep obtained (Okifuji & Hare, 2011). The current FM sample also responded similarly to previous FM populations on the PSQI (Bigatti et al., 2008; Hamilton et al., 2012; O'Brien et al., 2010), providing further support for the representativeness of the current sample.

The Daily Sleep and Pain Relationship

The first aim of this study was to determine the direction of the sleep and pain relationship among a sample of participants with FM. Recent reviews of the literature have established the connection between sleep and pain, although the direction of this relationship remains unclear (Finan et al., 2013; Moldofsky, 2001). In addition, FM is characterized by chronic widespread pain, and over 90% of individuals with this condition report poor sleep quality (Moldofsky, 2008; Wolfe et al., 1990). Therefore, this study sought to evaluate the direction of the daily sleep and pain relationship within this population.

In order to examine the intraindividual variability in daily sleep and pain among participants, hierarchical linear modeling (HLM) was utilized to analyze the daily objective and subjective sleep variables as predictors of next day's pain, as well as daily pain on the next night's objective and subjective sleep variables. This methodology accounted for between-person variation (each participant's deviation from the grand

mean) and within-person variation (a participant's daily deviation from their own 14-day mean), a process that reduced bias and more accurately reflected the relationship between daily measures and individual differences (Hoffman & Stawski, 2009). I will begin by discussing the average, between-person analyses of sleep and pain, as well as how the baseline predictors of depression, negative affect, sleep quality, pain, age, and pain catastrophizing predicted the daily sleep and pain outcome variables. I will then transition to the within-person analyses and discuss how these speak to the direction of the sleep and pain relationship.

Between-Person Sleep and Pain

For each participant and sleep predictor, a between-person variable representing that individual's average for the predictor across all days (person mean) was analyzed to determine if there was an impact on daily pain. Individuals who had more objectively-measured wake after sleep onset (WASO), averaged across days, experienced increased daily pain. Average pain across all 14 days was also analyzed to determine if there was an impact on the nightly objective and subjective sleep variables. Individuals who reported greater daily pain, averaged across days, experienced increased nightly objective WASO. These results provide further support for the relationship between sleep and pain and are also consistent with the literature that the most common sleep complaint of individuals with chronic pain is multiple awakenings (Taylor et al., 2007), and disruption of continuous sleep is more predictive of next day pain (Smith et al., 2007). However, average, between-person analyses do not speak to the direction of the sleep and pain relationship. Therefore, within-person, daily analyses are needed, which

will be addressed after discussing the between-person analyses of baseline predictors on sleep and pain.

Between-Person Baseline Predictors on Sleep and Pain

In addition to the established literature linking sleep and pain, several other variables have been shown to significantly relate to pain, sleep, or both (Edwards et al., 2006, Goodin et al., 2011, Lunde et al., 2010, Miró et al., 2011). Therefore, the baseline predictors of depression, negative affect, sleep quality, pain, age, and pain catastrophizing were each analyzed to determine if they significantly predicted the daily sleep and pain outcome variables. Age was found to be a significant predictor of daily pain, such that older individuals reported less pain, on average, than those who were younger. This is consistent with a recent correlational study that evaluated the association between aging and pain complaints among emergency room patients (Marco, Nagel, Klink, & Baehren, 2012). One explanation for this finding is that older individuals may perceive pain as part of the aging process and therefore underreport their pain (Klinger & Spaulding, 1998). Age was also a significant predictor of objective (actigraphy) sleep onset latency (SOL), such that individuals who were older took longer to fall asleep than those who were younger. This finding is consistent with a much larger body of literature on the relationship of sleep and aging. Research with both objective and self-report measures has found that older individuals take longer to fall asleep, spend more time awake during the night, and have decreased sleep efficiency (Lunde et al., 2010). In addition, the sleep of older individuals has been characterized as lighter and less refreshing than those who are younger, a description that has also been used to describe the sleep of individuals with FM (Crowley, 2011; Moldofsky, 2008).

Baseline pain catastrophizing was found to be a significant predictor of daily pain, such that individuals who reported greater levels of catastrophizing about their pain reported more daily pain, on average, than those who reported less catastrophizing. This finding is also consistent with the significant positive correlation between baseline pain catastrophizing and baseline pain, suggesting that those individuals who report more catastrophizing about their pain are more likely to report greater levels of pain both retrospectively and prospectively. Indeed, these findings support the substantial literature linking pain catastrophizing with a heightened pain experience. Diverse patient groups have displayed this relationship between pain catastrophizing and pain, including mixed chronic pain, rheumatoid arthritis, low back pain, and fibromyalgia (Edwards et al., 2006; Flor, Behle, & Birbaumer, 1993; Severeijns et al., 2001; Sullivan et al., 2001). Research has also shown that catastrophizing accounts for up to 31% of the variance in pain ratings (Sullivan et al., 2001). Of even greater importance is the theory behind how catastrophizing is thought to augment the pain experience. Similar to the concept of an irrationally negative outlook on the future that is associated with the catastrophizing found in anxiety and depression, pain catastrophizing is the tendency to exaggerate the level of pain threat coupled with pain-related worry and fear (Chaves & Brown, 1987; Spanos, Radtke-Bodorik, Ferguson, & Jones, 1979). Pain catastrophizing, then, appears to augment the experience and reporting of daily pain in this FM sample.

Greater levels of baseline pain catastrophizing were also found to predict increased objective wake after sleep onset (WASO) and increased subjective time in bed (SRTIB). These findings suggest that individuals who catastrophize about their pain, in addition to reporting more daily pain, also objectively experience and

subjectively report poorer sleep. Although there is significantly less evidence in the literature of a link between pain catastrophizing and sleep, the studies that have evaluated this relationship have found an association between higher pain catastrophizing and poorer sleep with both experimental pain testing and chronic pain populations (Goodin et al., 2011; Lee et al., 2013).

Patients who reported greater baseline pain also reported feeling less refreshed upon awakening than individuals who reported lower levels of baseline pain. It is surprising that baseline pain significantly predicted less refreshing sleep while daily pain did not. One explanation for this may be the way that pain was assessed at baseline. Participants were asked to report their worst, least, and average pain over the past week, as well as their current level of pain at the time of assessment. The interpretation of this retrospective measure may have actually assessed how participants had been “feeling” in the past week, including their fatigue, sleepiness, etc, in addition to their pain. Daily pain assessments, on the other hand, were not retrospective and may have captured a different aspect of the participants’ pain.

Baseline depression was a predictor of objective time in bed (TIB) as well as most of the subjective sleep variables. Individuals who reported higher levels of baseline depression spent more time in bed—and this was confirmed with actigraphy—and also reported that they took longer to fall asleep, spent more time awake during the night, had a lower sleep efficiency, and reported feeling less refreshed upon awakening than those who reported lower levels of depression. These findings are consistent with the literature on sleep and depression in FM (Miró et al., 2011; Munguia-Izquierdo & Legaz-Arrese, 2012). A recent study by Roehrs et al. (2013) found that individuals with

FM reported greater subjective sleepiness and fatigue than individuals with rheumatoid arthritis or healthy controls, but had the least objective daytime sleepiness as assessed by the Multiple Sleep Latency Test. It was concluded that this increased latency to sleep in FM is due to a state of hyperarousal, and this may be particularly true for individuals with more depression. In addition, one of the diagnostic criteria for Major Depressive Disorder is sleep disturbance (American Psychiatric Association, 2013). Similar to our findings, the classic insomnia-type sleep problems in depression are characterized by reports of difficulty falling asleep, waking intermittently throughout the night, and feeling unrefreshed upon awakening (Armitage, 2006). The fact that self-reported depression in our study is more predictive of self-reported poor sleep than of objectively-measured sleep quality suggests that individuals who are more likely to report high levels of depression are also more likely to report poor sleep regardless of the sleep they objectively obtain. This is similar to a study by Edinger et al. (2000) that found that psychological factors, including depression and anxiety, were related to subjectively-reported insomnia, but were not related to objectively-recorded sleep.

One aspect of depression is the experience of negative emotion, or negative affectivity. Surprisingly, baseline negative affect was not predictive of any subjective or objective sleep variables, suggesting that this construct is at least somewhat distinct from depression. Whereas high baseline depression was predictive of poorer nightly sleep, the lack of relationship between negative affect and sleep provides further support that the construct of depression contains unique aspects beyond negative affect. Perhaps it is the low positive affect / low energy component of depression that is more related to sleep difficulties than the negative affect component of depression.

The finding that baseline depression and negative affect did not significantly predict daily pain across individuals was surprising given previous literature that individuals with FM have higher levels of depression than those without a FM diagnosis and that pain intensity is positively associated with depression (Miró et al., 2011). Similarly, baseline depression and negative affect were not correlated with baseline pain, which again is surprising. One possible explanation is that both pain and depression were elevated in this population and the relationship between these factors was eliminated due to the narrowed range of scores.

Within-Person Sleep and Pain

As mentioned previously, average, between-person analyses do not speak to the direction of the sleep and pain relationship, necessitating within-person, daily analyses. Therefore, the nightly objective and subjective sleep variables were analyzed as predictors of next day's pain. Despite the examination of several sleep variables over the 14-day assessment period, there were relatively few findings. Results showed that individuals who reported feeling relatively unrefreshed upon awakening experienced more self-reported pain the rest of the day. This finding is consistent with the established literature that has found poor self-reported sleep quality to be a predictor of increased pain (Ağargün et al., 1999; Bigatti et al., 2008; Tang et al., 2012; Theadom et al., 2007).

Daily pain was also analyzed as a predictor of the next night's objective and subjective sleep variables. Only one sleep variable was predicted by daily pain. Individuals who reported more than their average pain one day experienced a greater self-reported latency to sleep (SOL) the following night. This suggests that pain does

not interfere with sleep once it has been initiated, but that it does prevent the onset of sleep. Increased latency may also represent the hyperarousal state that has been shown in FM and mentioned previously (Roehrs et al., 2013).

These within-person findings suggest that while there does appear to be a bidirectional relationship between sleep and pain, this only applies to a few sleep variables. The strongest relationship was found between daily self-reported refreshing quality of sleep and next day pain; a finding that is consistent with the literature (Finan et al., 2013). In the other direction, daily pain was predictive of self-reported sleep onset latency (SOL) the next night. One thing that is consistent when looking at the day-to-day relationship between sleep and pain is that this relationship is stronger with subjective sleep variables than with sleep variables measured with actigraphy. One possible explanation for this finding is that perception of sleep one night has more influence on self-reported next-day pain than objectively-measured sleep, and that perception of daily pain impacts reports of the next night's sleep more than sleep measured with actigraphy. Other possible explanations, which will be addressed in the limitations section, are shared methods variance of self-report predicting self-report for the sleep and pain variables, as well as a lack of validity in the sleep variables assessed with actigraphy.

Moderators of the Sleep and Pain Relationship

The second major aim of this dissertation was to determine what individual difference factors moderate the daily sleep / pain relationship. The baseline measures of depression, negative affect, age, and pain catastrophizing were analyzed to determine if each individually predicted the relationship between daily objective and

subjective sleep variables and next day's pain as well as the relationship between one day's pain and the next night's objective and subjective sleep variables.

Age was found to significantly moderate the relationship between one night's subjective sleep onset latency (SOL) and the next day's pain, such that older individuals had a stronger, positive relationship between these variables compared with younger individuals, who had no relationship between subjective SOL and next day's pain. As noted above, older individuals experienced significantly lower daily pain than younger individuals, but this difference was more pronounced for those individuals who reported shorter subjective SOL the previous night compared with those who reported longer SOL. Interestingly, age was also a significant moderator of the relationship between daily pain and the next night's subjective SOL, such that there was a stronger, positive relationship between one day's pain and the next night's subjective SOL for individuals who were older compared with no relationship for those who were younger. Older individuals had greater self-reported SOL than younger people, and this finding was more pronounced for individuals who reported higher levels of pain the previous day compared with those who reported lower levels of pain.

Thus, in older individuals, daily subjective SOL and daily pain are positively correlated in both directions, and possibly influence each other. In contrast, younger individuals do not appear to have any relationship between subjective SOL and pain, but they have higher levels of pain and shorter subjective SOL than individuals who are older. Perhaps younger individuals have unique characteristics that explain these findings, such as obtaining less sleep due to work or family obligations. Although not statistically significant, younger individuals tended to have less time in bed, less total

sleep time, and more wake after sleep onset than older individuals, suggesting that the younger participants may have been sleepier, and therefore, experienced more pain. Another explanation is that as individuals age, there may be adaptation to both affect and FM, resulting in the condition being less driven by affective dysregulation. Older people may become more regulated with age, or the FM pain processes may become less dependent on state factors such as mood and sleep.

Baseline depression was also examined as a potential moderator. Depression significantly moderated the relationship between refreshing sleep and next day's pain, such that more refreshing sleep predicted less pain subsequently, among those participants who were more depressed at baseline, compared with no relationship for those who were less depressed. This finding suggests that the perception of sleep quality has more impact on next day's pain for those individuals who reported high levels of baseline depression than those who reported lower levels of depression. Depression also significantly moderated the relationship between daily pain and refresh score upon awakening the next morning, such that there was a stronger, negative relationship between daily pain and the next night's refreshing quality of sleep for individuals who reported higher levels of baseline depression compared with the non-significant relationship between daily pain and next night's refreshing quality of sleep for individuals who reported lower levels of baseline depression. This finding suggests that there is less impact of baseline depression on refreshing quality of sleep when an individual experiences less pain the previous day.

Thus, in combination, these two moderator findings indicate that individuals who report higher levels of baseline depression have a negative relationship between pain

and refreshing quality of sleep in both directions, with these variables likely reciprocally influencing one another. That is, among the relatively depressed patients, refreshing sleep one night leads to less pain the next day, which leads to more refreshing sleep the following night. In contrast, those who reported lower levels of baseline depression did not exhibit the expected negative relationship between pain and refreshing quality of sleep. These findings suggest that the link between pain and sleep exists among those with depression because higher levels of depression are associated with increased reports of pain (Miró et al., 2011) as well as feeling unrefreshed upon awakening (Armitage, 2006). In addition, the relationship between sleep and pain is strengthened by the presence of depression. The negative sleep and pain relationship for individuals high in depression may be due to the presence of “subtypes” of FM. One subtype may be affectively dysregulated, in which a number of systems become disturbed including sleep, pain, and affect, allowing them to covary more tightly. A contrasting subtype of FM may have less affect dysregulation and system disruption, so the sleep, pain, and affect variables do not covary. This is consistent with the subtype model of FM offered by Turk, Okifuji, Sinclair, and Starz (1996), who proposed that there are “dysregulated,” “interpersonally distressed,” and “adaptive copers” types of FM patients.

Very similar results were also found with baseline negative affect, such that there was a stronger, negative relationship between self-reported refreshing quality of sleep and next day's pain for individuals who reported higher levels of baseline negative affect, compared with no relationship for those reporting lower levels of baseline negative affect. This finding suggests that the construct of negative affect moderates the relationship of one night's refreshing quality of sleep on next day's pain in the same way

that was found with depression. This may be due to the negative expression of emotion contained in both depression and negative affect, or may speak to the relationship between one night's refreshing quality of sleep on next day's pain. Individuals who report low refreshing quality of sleep may also express negative affectivity in general, suggesting why both high negative affect and high depression would respond similarly.

Negative affect significantly moderated the relationship between objective total sleep time (TST) and next day's pain, such that there was a small, negative relationship between objective TST and next day's pain for individuals who reported higher levels of baseline negative affect, compared with a small, positive relationship between objective TST and next day's pain for those who reported lower levels of baseline negative affect. Interestingly, as total sleep time increased, the effect of negative affect on daily self-reported pain attenuated, suggesting that negative affect has a greater impact on daily pain when an individual has less sleep the previous night.

Pain catastrophizing significantly moderated the relationship between objective sleep efficiency (SE) and next day's pain, such that there was a small, negative relationship between objective SE and next day's pain for individuals who reported higher levels of baseline pain catastrophizing and a small, positive relationship between objective SE and next day's pain for those who reported lower levels of baseline pain catastrophizing. This finding suggests that as sleep efficiency increases, baseline pain catastrophizing has less of an impact on next day's pain. Pain catastrophizing was also found to significantly moderate the relationship between daily pain and the next night's objective SE, such that there was a negative relationship between daily pain and objective SE for individuals who reported higher levels of baseline pain catastrophizing

compared with the almost equally positive relationship between daily pain and objective SE for individuals who reported lower levels of baseline catastrophizing. This finding suggests that the interpretation of the experience of pain has a significant impact on the way that one day's pain is related to the next night's sleep efficiency.

Thus, individuals who report higher levels of baseline pain catastrophizing have a negative relationship between pain and objective sleep efficiency in both directions, with these variables potentially influencing one another. In contrast, those who reported lower levels of baseline pain catastrophizing did not exhibit the expected negative relationship between pain and refreshing quality of sleep. More importantly, the findings seem to be consistent with the other predictors of baseline depression and age; individuals who report higher baseline catastrophizing and depression, as well as those who are older, show the expected "poor sleep and higher pain" relationship. The similar relationships among these moderator variables suggest a consistent effect. This is not surprising for baseline pain catastrophizing and baseline depression since these variables were positively correlated with each other and there are consistent findings in the literature that individuals who catastrophize about their pain and report symptoms of depression have poor sleep and increased pain compared with those who are lower in catastrophizing and depression (Goodin et al., 2011, Miró et al., 2011, Sullivan et al., 2001). On the other hand, it is surprising that older individuals demonstrated the expected "poor sleep and higher pain" relationship, especially since they reported lower levels of depression and pain catastrophizing. One possible explanation for this finding may be that older individuals had more variability in their daily pain and sleep onset

latency (SOL) than younger individuals, allowing for a relationship between sleep and pain.

Limitations of the Study

Although this study has many strengths, such as the use of a relatively large and homogeneous chronic pain population of individuals with FM, and the utilization of both objective and subjective measures of sleep, there are several limitations that I will now address. One limitation is that all of the participants were individuals who sought participation in a treatment study for stress. Thus, this self-selected sample may have unique characteristics, such as having high rates of affect disorders, trauma histories, etc. that are not representative of the larger FM population. Results should therefore be interpreted with caution. Additionally, the study sample consisted of only individuals with a diagnosis of FM; therefore, the findings may not be generalizable to other chronic pain populations, particularly conditions with younger individuals, with more men, and that have pain with less affective dysregulation.

Another limitation is the use of paper diaries for collecting daily subjective sleep and pain variables. Although the participants were taught how to complete the diaries and when to answer the various questions about their sleep and pain, there was no independent validation of when the diary was completed. Similarly, the use of a once-daily pain rating collected on paper is a limitation of the study. Assessing pain at multiple points throughout the day and utilizing electronic diaries for sleep and pain variables would ensure timely completion of these temporal variables and allow for analyses of daily pain fluctuation. All of the diary data were also based on self-report

measures of sleep and pain. Therefore, the relationships found among the variables may be accounted for, at least in part, by shared method variance.

The study is also correlational in nature, rather than experimental. Therefore, no definitive causal interpretations can be made about the sleep and pain relationship. Although the data suggest a potential bidirectional relationship, there were no good estimates for determining which direction of the effect is stronger. Another limitation of the study is that the lag was only one day, assessing one night's sleep on next day's pain and one day's pain on the next night's sleep. It is possible that effects took longer to manifest and longer lag periods should have been tested in the analyses. Additionally, increasing the study period beyond 14 days may have also resulted in more reliable estimates.

The utilization of actigraphy, although a strength for collecting objective sleep data, may also be a limitation of this study. Actiwatches are an excellent method for assessing participant movement, but there is some question about the validity of sleep variables collected with actigraphy. Although assessment studies have shown a high level of agreement for sleep scored from polysomnography and actigraph algorithms ($r = 0.85$), this is only for normal individuals (Acebo, 2006). Accuracy of sleep and wake measured with actigraphy tends to decrease when sleep is disturbed, as is often the case in chronic pain populations (Kushida et al., 2001). There is also little evidence for the validity of several sleep variables collected with actigraphy, including sleep onset latency and wake after sleep onset (Acebo, 2006). In addition, actigraphy was scored with the aid of self-reported diary variables that include limitations stated above. For example, many participants had periods of "inactivity" as measured with actigraphy that

did not correspond to the times that were recorded in the diary for sleep. Therefore, it was unclear if the participants were asleep, sitting very still, or had removed the actiwatch during these periods. Ambiguous periods of time were eliminated from analyses, which may have underestimated the total sleep time for some participants.

Another limitation of the study is that it would have been ideal to assess more measures during the daily recording period. Negative affect, depressive symptoms, and pain catastrophizing were assessed as trait-type measures, asking participants to respond based on how they typically feel over longer periods of time. These measures could also have been assessed from day to day, allowing me to test how they influenced the sleep / pain relationship. Another measure that would have been ideal to assess daily is pain interference. The long version of the Brief Pain Inventory (BPI; Cleeland & Ryan, 1994) consists of a pain interference component in addition the pain intensity component assessed at baseline in this study. Daily pain interference may be a more important outcome measure than pain intensity for determining the impact of poor sleep the previous night.

The final limitation is the analysis of so many objective and subjective sleep variables in the analyses. Sleep efficiency was predicted to be the sleep variable of interest for this study, but it was not significantly related to daily pain in unmoderated relationships. Other sleep variables were then analyzed to determine their relationship with daily pain, and very few of the analyzed variables resulted in significant findings. Therefore, there is concern regarding Type 1 error for these sleep variables, and the results should be interpreted with caution.

Implications and Future Directions

This study suggests that there is a bidirectional relationship between sleep and pain, with daily refreshing quality of sleep predicting next day's pain, and one day's pain predicting the next night's subjective sleep onset latency. Average objective wake after sleep onset also predicted daily pain, and daily pain predicted average objective wake after sleep onset. In addition to the daily sleep and pain findings, several factors that explained individual differences in the sleep and pain relationship including depression, negative affect, age, and pain catastrophizing were assessed as moderators. Age, depression, and pain catastrophizing all exhibited bidirectional moderation of the sleep and pain relationship; age moderated the self-report sleep onset latency and pain relationship, depression moderated the refreshing quality of sleep and pain relationship, and pain catastrophizing moderated the objective sleep efficiency and pain relationship. In addition, individuals who were older as well as those who reported higher levels of baseline depression and pain catastrophizing demonstrated the expected results of poorer sleep being associated with increased pain. The similar relationships among these moderator variables suggest a consistent effect. These findings provide more support for the temporal relationship of daily sleep and pain and indicate that there are individual factors that should be considered in evaluating this relationship.

There are many questions that remain unanswered, however. Although there was a relationship between sleep and pain for a few select sleep variables, it is unclear as to why other objective and subjective sleep variables were not associated with daily pain. It is also unclear why depression and negative affect were not related to baseline pain or daily pain. This finding is not consistent with the literature, and future studies

should evaluate the relationship of depression and negative affect with pain to determine if this finding can be replicated. Future studies should also explore increasing the length of sleep and pain data collection as well as other methodologies for collecting diary data that increase the reliability of temporal self-report measures.

In conclusion, this dissertation adds to the established literature on the relationship between sleep and pain, utilizing objective and subjective measures of sleep within a large chronic pain population of individuals with FM. Future research should focus on individual differences in the sleep and pain relationship in order to determine if there are more significant moderation relationships among these variables.

APPENDIX A: ACTIWATCH LOG

INSTRUCTIONS

Thank you for agreeing to take part in our study and wearing the *Actiwatch*.

This watch-like device records your daily movement and will help us better understand sleep-wake cycles, activity levels, and daily pain ratings. The information you provide in this workbook will help the researchers analyze the data captured on your *Actiwatch*.

Fill out the questions in this book each day you are asked to wear the *Actiwatch*.

Morning questions are about the previous night's sleep.

At the end of each day, we ask you to record your average pain rating for the day in this logbook.

Some reminders:

- Wear the watch on your non-dominant wrist.
- There is no need to turn the watch on, it will begin recording by itself. The watch does not appear to be doing anything, but it is recording your movement.
- Please note in your logbook anytime during the day you take the watch off (to shower, etc).
- Bring your watch and logbook with you to your next study visit.

Please contact the study coordinator if you have any questions or concerns about wearing this device.

Day 1

Mon / Tue / Wed / Thu / Fri / Sat / Sun

Today's Date: ___ / ___ / _____

Morning Questions1. What time did you turn off the light and try to go to sleep last night?: AM / PM2. How many minutes did it take you to fall asleep last night? minutes3. How many times did you wake up last night before you woke up to start your day? times4. How many total minutes were you awake last night from these awakenings? minutes5. What time did you wake up this morning to start your day?: AM / PM6. Using the scale below, please rate how refreshed you feel after last night's sleep.

0	1	2	3	4	5	6	7	8	9	10
Not at all refreshed										Completely refreshed

Evening Questions7. Did you take off the watch today (to shower, swim, etc.)? If so, please record:

Time that I took off the watch	Approx length of time watch was off
<input type="text"/> <input type="text"/> : <input type="text"/> <input type="text"/> AM / PM	<input type="text"/> <input type="text"/> <input type="text"/> minutes
<input type="text"/> <input type="text"/> : <input type="text"/> <input type="text"/> AM / PM	<input type="text"/> <input type="text"/> <input type="text"/> minutes
<input type="text"/> <input type="text"/> : <input type="text"/> <input type="text"/> AM / PM	<input type="text"/> <input type="text"/> <input type="text"/> minutes
<input type="text"/> <input type="text"/> : <input type="text"/> <input type="text"/> AM / PM	<input type="text"/> <input type="text"/> <input type="text"/> minutes

8. Using the scale below, please rate your average pain today.

0	1	2	3	4	5	6	7	8	9	10
No Pain										Pain as bad as you can imagine

- End of questions for today-

APPENDIX B: BASELINE MEASURES

CES-D

Below is a list of ways you might have felt or behaved. Please indicate how often you have felt this way during the PAST WEEK by placing a check in the box below your response.

	Rarely or none of the time (less than 1 day)	Some or a little of the time (1 to 2 days)	Occasionally or a moderate amount of the time (3 to 4 days)	Most or all of the time (5 to 7 days)
In the past week:				
1. I was bothered by things that usually don't bother me.				
2. I did not feel like eating; my appetite was poor.				
3. I felt that I could not shake off the blues even with help from my family or friends.				
4. I felt that I was just as good as other people.				
5. I had trouble keeping my mind on what I was doing.				
6. I felt depressed.				
7. I felt that everything that I did as an effort.				
8. I felt hopeful about the future.				
9. I thought that my life had been a failure.				
10. I felt fearful.				
11. My sleep was restless.				
12. I was happy.				
13. I talked less than usual.				
14. I felt lonely				
15. People were unfriendly.				
16. I enjoyed life.				
17. I had crying spells.				
18. I felt sad.				
19. I felt that people dislike me.				
20. I could not get going				

PCS

Everyone experiences painful situations at some point in their lives. Such experiences may include headaches, tooth pain, joint or muscle pain. People are often exposed to situations that may cause pain such as illness, injury, dental procedures or surgery.

Instructions:

We are interested in the types of thoughts and feelings that you have when you are in pain. Listed below are thirteen statements describing different thoughts and feelings that may be associated with pain. Using the following scale, please indicate the degree to which you have these thoughts and feelings when you are experiencing pain.

RATING	0	1	2	3	4
MEANING	Not at all	To a slight degree	To a moderate degree	To a great degree	All the time

When I'm in pain ...

Number	Statement	Rating
1	I worry all the time about whether the pain will end.	
2	I feel I can't go on.	
3	It's terrible and I think it's never going to get any better	
4	It's awful and I feel that it overwhelms me.	
5	I feel I can't stand it anymore	
6	I become afraid that the pain will get worse.	
7	I keep thinking of other painful events	
8	I anxiously want the pain to go away	
9	I can't seem to keep it out of my mind	
10	I keep thinking about how much it hurts.	
11	I keep thinking about how badly I want the pain to stop	
12	There's nothing I can do to reduce the intensity of the pain	
13	I wonder whether something serious may happen.	

BPI

1. Please rate your pain by circling the one number that best describes your pain at its **worst** in the last week.

0 1 2 3 4 5 6 7 8 9 10

No pain

Pain as bad as
you can imagine

2. Please rate your pain by circling the one number that best describes your pain at its **least** in the last week.

0 1 2 3 4 5 6 7 8 9 10

No pain

Pain as bad as
you can imagine

3. Please rate your pain by circling the one number that best describes your pain on the **average** for the last week.

0 1 2 3 4 5 6 7 8 9 10

No pain

Pain as bad as
you can imagine

4. Please rate your pain by circling the one number that tell how much pain you have **right now**.

0 1 2 3 4 5 6 7 8 9 10

No pain

Pain as bad as
you can imagine

PSQI

Instructions: The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month. Please answer all questions.

During the past month,

1. What **TIME** have you usually gone to bed? _____
2. How long (in minutes) has it taken you to fall asleep each night? _____
3. What **TIME** have you usually gotten up in the morning? _____
4. How many hours of actual sleep do you get at night? (This may be different than the number of hours you spend in bed) _____

5.	During the past month, how often have you had trouble sleeping because you...	Not during the past month (0)	Less than once a week (1)	Once or twice a week (2)	Three or more times a week (3)
a.	Cannot get to sleep within 30 minutes				
b.	Wake up in the middle of the night or early morning				
c.	Have to get up to use the bathroom				
d.	Cannot breathe comfortably				
e.	Cough or snore loudly				
f.	Feel too cold				
g.	Feel too hot				
h.	Have bad dreams				
i.	Have pain				
j.	Other reason(s), please describe, including how often you have trouble sleeping because of this reason(s):				
6.	During the past month, how often do you take medicine (prescribed or "over the counter") to help you sleep?				
7.	During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?				
8.	During the past month, how much of a problem has it been for you to keep up enthusiasm to get things done?				
		Very Good (0)	Fairly Good (1)	Fairly Bad (2)	Very Bad (3)
9.	During the past month, how would you rate your sleep quality overall?				

PANAS

This scale consists of a number of words and phrases that describe different feelings and emotions. Read each item and then circle the number that corresponds to the appropriate answer. Indicate **to what extent you have felt this way during the past few weeks**. Use the following scale to record your answers:

	Very slightly or not at all	A little	Moderately	Quite a bit	Extremely
1. interested					
2. irritable					
3. distressed					
4. alert					
5. excited					
6. ashamed					
7. upset					
8. inspired					
9. strong					
10. nervous					
11. guilty					
12. determined					
13. scared					
14. attentive					
15. hostile					
16. jittery					
17. enthusiastic					
18. active					
19. proud					
20. afraid					

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ABSTRACT**A DAILY STUDY OF THE SLEEP-PAIN RELATIONSHIP IN FIBROMYALGIA**

by

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Fibromyalgia (FM) impacts millions of individuals around the world and is characterized by widespread chronic pain and tenderness as well as nonrestorative sleep, fatigue, and stiffness (Wolfe et al., 1990; Wolfe et al., 2010). Poor sleep quality is reported by more than 90% of individuals with FM, suggesting that sleep disturbance may be a contributing factor to the pain experience (Moldofsky, 2008). Recent reviews of the literature have established the connection between sleep and pain, although the direction of this relationship remains unclear (Finan et al., 2013; Moldofsky, 2001). This dissertation sought to examine the daily relationship between sleep and pain in a large population of chronic pain patients with fibromyalgia (FM), as well as the factors that may explain individual differences in this relationship. Ninety adults with FM completed baseline self-report measures of depression, negative affect, pain, sleep disturbance, and pain catastrophizing. Participants also wore an actiwatch, an objective measurement of sleep, for two weeks while completing daily diaries about their sleep and pain. Hierarchical linear modeling (HLM) was utilized to examine the intraindividual

variability in daily sleep and pain among participants, as well as the baseline factors that explain individual differences in this relationship.

Results of this study suggest that there is a bidirectional relationship between sleep and pain, with daily refreshing quality of sleep predicting next day's pain, and one day's pain predicting the next night's self-reported sleep onset latency. Average objective wake after sleep onset also predicted daily pain, and daily pain predicted average objective wake after sleep onset. In addition to the daily sleep and pain findings, several factors that explained individual differences in the sleep and pain relationship including depression, negative affect, age, and pain catastrophizing were assessed as moderators. Age, depression, and pain catastrophizing all exhibited bidirectional moderation of the sleep and pain relationship, and individuals who were older as well as those who reported higher levels of baseline depression and pain catastrophizing demonstrated the expected results of poorer sleep being associated with increased pain. The similar relationships among these moderator variables suggest a consistent effect. These findings provide more support for the temporal relationship of daily sleep and pain and indicate that there are individual factors that should be considered in evaluating this relationship.

AUTOBIOGRAPHICAL STATEMENT

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Maren Hyde-Nolan earned her undergraduate degrees in Biology and Psychology from Hope College in 2003. She is completing her PhD in Clinical Psychology at Wayne State University.

Maren's career interests are in the area of health psychology, particularly the relationships of chronic pain, sleep, and coping. Her work and graduate training have provided her an opportunity to participate as an active member of the WSU Health Psychology Research Lab and at the Sleep Disorders and Research Center at Henry Ford Hospital. In addition to the current project, she completed a Master's thesis on the impact of pre-surgical sleep on post-surgical pain, and worked on a study that evaluated the outcomes of a novel, emotional awareness chronic pain treatment program. She has also taught several courses at Wayne State University, including Developmental Psychology, Personality, Health Psychology, Abnormal Psychology, Human Sexuality, and Elements of Psychology.

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