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An Examination of Clinical Pain Severity, Sleep Quality, And Neighborhood Disadvantage in a Racially/Ethically Diverse Sample of Adults With Symptomatic Knee Osteoarthritis

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AN EXAMINATION OF CLINICAL PAIN SEVERITY, SLEEP QUALITY, AND NEIGHBORHOOD DISADVANTAGE IN A RACIALLY/ETHNICALLY DIVERSE SAMPLE OF ADULTS WITH SYMPTOMATIC KNEE OSTEOARTHRITIS

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ABSTRACT

Knee osteoarthritis (OA) is often accompanied by insomnia symptoms, characterized by difficulty falling asleep, maintaining sleep, or both. Among individuals with knee OA, up to 31% report significant difficulty falling asleep, 81% have difficulties staying asleep, and up to 77% report any sleep problem. However, the sleep and pain relationship has also rarely been evaluated using objective and subjective sleep measures. Thus, this study attempted to characterize objective and subjective sleep variables in a diverse sample of individuals with symptomatic knee OA. Further, the impact of objective and subjective sleep quality on clinical pain severity, movement-evoked pain, and physical function change across time was explored. Furthermore, this study examined racial/ethnic differences stratified by levels of neighborhood disadvantage with our variables of interest both at baseline and across time. Lastly, we examined the relationship between pain catastrophizing and clinical pain severity and the mediating role of sleep quality. Results showed that individuals living with symptomatic knee OA had reported worse subjective sleep quality and reductions to objective sleep quality compared to healthy controls. Objective and subjective sleep quality were associated with clinical pain severity, movement-evoked pain, and declines in physical function depending on the mode of sleep measurement. Objective/subjective sleep quality was not predictive of change scores of clinical pain, MEP, and physical function measures. Racial/ethnic group differences were

demonstrated in that NHB individuals reported increased clinical pain severity, MEP, and reductions in physical function. Analyses examining neighborhood disadvantages showed that individuals living in areas with more disadvantages had significant reductions in physical function scores. Multimodal sleep disturbance mediated the relationship between pain catastrophizing and clinical pain severity. Moderated mediation was present for NHW individuals in the relationship between pain catastrophizing and clinical pain severity via subjective sleep on the Insomnia Severity Index. These results provide initial support for neighborhood disadvantage as a risk factor for adverse pain outcomes in knee OA. Health providers should assess and target pain catastrophizing and multimodal sleep disturbance as clinical interventions when managing individuals living with symptomatic knee OA.

Keywords: osteoarthritis, sleep, pain, catastrophizing, neighborhood disadvantage

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INTRODUCTION

Knee Osteoarthritis and Clinical Pain

Chronic pain is a multifarious and prominent health concern prevalent in contemporary society, impacting individuals with greater healthcare costs and increasing the burden on all healthcare systems (Domenichiello & Ramsden, 2019). Chronic pain's economic and financial strain on the United States healthcare system is prodigious, and it is estimated that the total costs associated with chronic pain exceed $635 billion (Gaskin & Richard, 2012). Chronic pain is characterized as pain that persists beyond the period of normal tissue healing and is quantitatively described as lasting greater than 3-6 months in duration. (Treede, et al., 2015). Chronic pain can be explained by some, albeit variable, element of central sensitization (Crofford, 2015). Central sensitization is when prolonged heightened reactivity causes spinal and supraspinal processing changes that increase afferent nociceptive input. These changes lead to hypersensitivity to pain and the maintenance of pain even after the initial injury might have healed, leading to the chronicity of these symptoms. However, chronic pain is a complex sensory and emotional experience that varies widely on a person-to-person basis. The experience of pain is relative to the context of how the pain is experienced and the individual's psychological state. For these reasons, chronic pain conditions often lead to a wide range of negative physical, psychological, and social consequences that profoundly reduce overall quality of life (Grichnik & Ferrant 1991).

One of the most common disabling chronic pain conditions and a leading contributor to medical care-seeking in adults is knee osteoarthritis (OA) (Helmick et al., 2008). Knee OA, a degenerative joint disorder impacting the three compartments of the knee joint (medial, lateral, and patellofemoral), is characterized by persistent pain and stiffness (Chu, Millis, & Olson, 2014). Knee OA causes soft tissue structures, such as the synovium, ligaments, and bridging muscle, surrounding the joint to become inflamed or weak. These changes to soft tissue structures cause joint space narrowing, subchondral sclerosis, and osteophyte formation (Courtney, O'Hearn, & Hornby, 2012). Diagnosis of knee OA has traditionally focused on radiographic imaging techniques, given their ability to identify pathoanatomical disease processes, including joint space narrowing, cartilage degradation and sclerosis, and osteophyte formation. Narrowing of the joint space and osteophyte formation seen in knee radiographs are often graded using the Kellgren-Lawrence (KL) classification, which assigns a grade from zero to four, indicating the severity of OA. Greater KL scores indicate the presence of more severe radiographic knee OA. However, the presence of radiographic knee OA findings and corresponding KL grades does not regularly correspond with the presence and severity of knee pain. Radiographic imaging and corresponding KL grades have demonstrated variable predictive validity of self-reported knee pain symptoms, with some studies reporting moderate to strong associations (Duncan et al., 2007; Neogi et al., 2009) and other studies reporting weaker associations (Bedson & Croft, 2008; Finan et al., 2013). In a study by Torres et al. (2006), it was suggested that bone marrow lesions and synovitis, neither captured by radiographic imaging, are more predictive of knee pain symptoms than the traditional KL grades. Altogether, the varied nature of these findings demonstrates the

impreciseness of solely relying on radiographic knee OA imaging as the primary indicator of knee pain presentation and clinical outcomes. The poor alignment between an objective diagnostic measure of knee OA on radiographic imaging and the subjective experience of chronic knee pain suggests an abundance of intersecting biopsychosocial factors beyond pathoanatomy likely contributing to knee OA clinical pain severity.

For example, localized inflammation leads to pain resulting from nociceptors located deep within the knee (peripheral sensitization), followed by central nervous system changes that augment the perception of the pain (central sensitization) (Glover et al., 2015). Plastic changes in the central nervous system (especially the brain) resulting from pain-related central sensitization are thought to be one way that chronic knee pain persists even when localized pathoanatomy of the knee is minimal or significantly improved with treatment (e.g., total knee arthroplasty) (Loggia & Edwards, 2018; Pujol et al., 2017). Evidence for this paradigm is supported by animal research demonstrating that input from afferent nociceptive pathways in damaged knee joints potentiates central sensitization in rats (Martindale et al., 2007). Similarly, experimental human studies have also revealed evidence of central sensitization among those with knee OA using experimental pain stimuli that include mechanical and thermal stimuli (Suokas et al., 2012). Knee OA is often referred to as a progressive disease and has been demonstrated to develop slowly over 10 to 15 years, ultimately interfering with functional mobility and activities of daily living (Lespasio et al., 2017). While localized inflammation and peripheral/central sensitization are thought to contribute to the development and progression of knee OA, it has also been shown that greater load bearing on the knee from obesity or injury tends to initiate or exacerbate cartilage degeneration, increasing

both severity and disability (Deveza et al., 2017). Although disease pathophysiology of knee OA is still actively under investigation, it is currently accepted that there is no consistent and direct relationship between the degree of knee pathophysiology and the symptoms (i.e., pain) of knee OA therein (Bedson & Croft, 2008). This discrepancy suggests that other pain-relevant psychosocial and behavioral factors may contribute to the degree of knee pain experienced by those with/without radiographic evidence of knee OA.

Movement-evoked pain versus pain at rest

Contemporary research suggests that the type of pain assessed is likely to be relevant to understanding how biopsychosocial factors (e.g., sleep, SES, pain catastrophizing) contribute to the progression and severity of chronic knee pain, as well as the frequent discordance between radiographic findings and knee pain symptoms. Given that many knee pain experiences are brought on or exacerbated by movement, an emerging diagnostic measure of clinical knee pain severity is the assessment of movement-evoked pain (MEP) (Ferreira-Gomes & Castro-Lopes, 2008). MEP examines an individual's pain perception in response to functional movement (Wan et al., 2018). Recent studies have demonstrated that MEP is often a significant factor in observed physical performance outcomes, disability and unfavorable psychological characteristics (Cruz-Almeida et al., 2017; Simon et al., 2023). However, most experimental and clinical pain assessments often fail to consider or measure MEP (Corbett et al., 2019). Instead, most clinical pain severity measures in use today typically assess clinical pain at rest (PAR) through reports of spontaneous pain or recall of previous self-reported pain episodes (Booker et al., 2019). Emerging evidence has revealed distinct differences

between PAR on self-report questionnaires and MEP (Fullwood et al., 2021). A key mechanism of movement-evoked pain is the activation of silent nociceptors in response to joint movement or other movement-related stimuli that are generally not painful (McDougall, 2006). These findings suggest that MEP represents a pain-related phenomenon unique and distinct from PAR. Therefore, MEP may be an essential measure of knee OA pain severity in conjunction with PAR measures when comprehensively assessing clinical pain.

Sleep Disturbance and Clinical Pain

Sleep disturbance is one such behavioral factor that is likely to affect the experience of knee pain, given that problems with sleep are one of the most prevalent complaints for individuals with chronic pain conditions. Systems that regulate arousal and pain are neurobiologically intertwined and may share common pathways that undermine the attainment of quality sleep. Sleep disturbance has also been shown to increase the risk of developing pain complaints and amplify persistent post-injury pain (Gupta et at., 2007). Clinical studies using polysomnography have demonstrated that chronic pain patients may show reduced delta sleep and increased alpha sleep leading to sleep disturbance (Smith & Haythornthwaite, 2004). However, the bidirectionality of the sleep and pain relationship is poorly understood (Finan, Goodin, & Smith, 2013). A longitudinal study looking at fibromyalgia patients suggests that a high prevalence of sleep problems preceded increases in clinical pain, whereas pain at baseline was not associated with sleep disturbance at follow-up (Bigatti et al., 2008). Conversely, an observational study evaluating clinical pain and sleep in individuals with chronic physical disability suggested that the effects of pain on sleep were stronger than sleep on pain when included in cross-lagged effects models

(Amtmann et al., 2020). A limitation of these studies and most studies evaluating the sleep and pain relationship is the exclusive use of subjective self-reported sleep disturbance outcomes without considering objective sleep measures. Few studies have evaluated this relationship using objective sleep measures such as actigraphy or polysomnography. One study by Bulls et al. (2017) demonstrated the impact of actigraphy-based sleep efficiency on experimental pain thresholds. Despite a lack of clarity regarding the bidirectionality of the sleep and pain relationship, it is widely acknowledged that chronic pain and sleep disturbance are linked to poor health outcomes, including psychiatric comorbidity and long-term disability (Smith & Haythornthwaite, 2004).

Cross-sectional studies have consistently found that sleep disturbance is positively associated with increased clinical pain severity and negative mood symptomology (Smith & Haythornthwaite, 2004). Specifically for individuals with knee OA, about one-third of knee OA patients report both clinically significant pain and co-morbid insomnia (McCurry et al., 2011). Sleep disturbance in knee OA is characterized by poor sleep quality, sleep fragmentation, and frequent shifts between sleep stages at night (Allen et al., 2008) that are due at least in part to linkages with pain (Lamberg, 1999; Leigh et al., 1988; Wilxcox et al., 2000). With the variety of sleep disturbances that these individuals with knee OA experience at night, there are often impairments throughout the following day, such as significant daytime fatigue, impaired functional mobility, and reduced quality of life (Hawker et al., 2010). Researchers have also endeavored to elucidate potential psychosocial factors that may explain the sleep disturbance and pain relationship. Others have posited that depressive symptoms and overall mental health status may mediate the relationship between sleep and pain leading to unfavorable trajectories in knee OA

(Parmalee, Tighe, & Dautovich, 2015; Song et al., 2020). Another psychological factor that could possibly mediate the relationship between sleep and pain is pain catastrophizing (Tighe et al., 2020). A review conducted by De Baets et al., (2023) summarized that depressive symptoms, pain catastrophizing, and pain self-efficacy as a mechanism explaining the association between insomnia and pain in knee OA populations.

Pain Catastrophizing, Clinical Pain, and Sleep

A psychosocial factor with established relevance to sleep and pain is pain catastrophizing (Campbell & Edwards, 2009). Pain catastrophizing is a persistently negative cognitive-affective style characterized by helplessness, magnification, and ruminative thinking regarding experienced pain symptomology (Edwards et al., 2006). It has been shown that pain catastrophizing is a significant predictor of greater chronic pain severity and related disability in OA populations (Edwards et al., 2011). Pain catastrophizing has also been reliably associated with increased pain sensitivity (Edwards, Campbell, & Fillingim, 2005) and diminished endogenous pain inhibitory controls when evaluated in studies involving healthy participants (Goodin et al., 2011). In recent years, pain catastrophizing has been implicated as a potential underlying factor in the sleep and pain relationship. Campbell & Edwards (2015) examined sleep disturbance in knee OA revealing a combined effect of pain catastrophizing and sleep disturbance that increases pain sensitivity in knee OA. Similarly, Tighe et al. (2020) demonstrated that pain catastrophizing and self-efficacy partially mediated the association between sleep disturbance and OA symptom severity. This finding suggests that insufficient sleep may deplete adaptive resources such as one's sense of self-efficacy, leading to increased maladaptive coping such as pain catastrophizing. These results are consistent with existing

literature linking better sleep quality with improved cognitive and affective functioning (Ong et al., 2017) and higher resilience scores (Wang et al., 2020). Despite this promising research, few studies to date have evaluated this relationship in the context of neighborhood disadvantage. One study by Goodin et al. (2011) suggested that NHB with reduced sleep quality may be at greater risk for pain catastrophizing. This finding suggests that further research is warranted in disentangling the role of pain catastrophizing and its impact on health outcomes in diverse community samples.

Racial/Ethnic Differences in Clinical Pain and Sleep

Chronic pain is known to be a great equalizer and poses the same similar risks to all segments of the population. However, individuals within the U.S. who identify as non-Hispanic Black (NHB) often experience more adverse, frequent, and disabling chronic pain than their counterpart racial group, non-Hispanic Whites (NHW). This trend is particularly true for chronic pain patients diagnosed with knee osteoarthritis (OA) (Cruz-Almeida et al., 2014). While previous research inconclusively alluded to mixed results of racial/ethnic differences in the presence and severity of chronic pain (Edwards et al., 2005; Green et al., 2003), it can now be widely accepted that the burden of chronic pain disproportionately marginalizes racial/ethnic minorities. The first phase of this study (UPLOAD 1) demonstrated that NHBs with knee OA reported significantly greater OA-related pain and disability than their NHW counterparts (Cruz-Almeida et al., 2014). In addition, we observed racial differences in pain-related psychosocial and biological measures, contributing to racial group differences in experimental pain sensitivity. Along with pain severity, NHBs have been found to report less perceived control over their pain and more significant pain-related emotional distress than NHWs (Green et al., 2003). NHB adults

with knee OA often report greater severity of their pain symptoms than NHW adults because NHBs demonstrate more significant endogenous pain modulatory balance alterations. Several pain studies have found significant associations between clinical pain severity and increased sensitivity to pain stimuli, including lower ischemic pain, temporal summation of mechanical pain, and differences in pain inhibition using conditioned pain modulation (Edwards et al., 2001; Goodin et al., 2014).

Emerging literature has reported that there may also be distinct racial/ethnic differences in sleep disturbance (Ruiter et al., 2010, 2011). Specifically, several studies have reported shorter sleep time among NHBs as compared to NHWs across the adult life span and poorer sleep quality on several measures, including overall efficiency, sleep latency, wakefulness after sleep onset (WASO), and sleep fragmentation (Beatty et al., 2012; Carnethon et al., Tomfohr et al., 2012). Bulls et al. (2017) demonstrated that greater depressive symptoms and reduced sleep efficiency enhanced the temporal summation of mechanical pain, particularly for NHBs. One explanation for potential ethnic/racial differences could be environmental and social risk factors, such as those related to low socioeconomic status, which may play a role in exacerbating the perception of both clinical pain severity and sleep disturbance (Fuller-Rowell et al., 2016; Thompson et al., 2019).

A Consideration of Neighborhood Disadvantage

Socioeconomic status (SES) refers to the social standing or class of an individual or group. SES is thought to incorporate a wide range of resources, including money, knowledge, prestige, power, and beneficial social connections that, when present, can serve as a buffer against poor health and functional limitations (Phelan et al., 2010). SES is considered one of the most robust determinants of variations in health outcomes worldwide

(Williams, Priest, & Anderson, 2016). In the context of health research, SES has traditionally represented a composite demographic variable that generally includes a summary score composed of income, occupational status, and educational level (Poleshuck & Green, 2008). Over the years, low SES has been associated consistently with virtually every aspect of poor health, including increased morbidity, decreased life expectancy, and higher infant mortality (Link & Phelan, 1995). Not surprisingly, low SES also is consistently associated with an increased risk for chronic pain (Janevic et al., 2017). SES and pain are linked for patients with an array of pain sites (e.g., musculoskeletal, sciatica, ulcer, neuropathic pain) (Gran, 2003; HeliÖvaara et al., 1991; Levenstein & Kaplan, 1998; Torrance et al., 2006).

Socioeconomic disadvantage (i.e., low SES) and its contribution to individuals' chronic pain experience with knee OA are of growing interest and relevance in the field. SES has been shown to differentially affect the clinical symptoms experienced by individuals with knee OA. To illustrate, individuals with lower educational attainment, non-managerial jobs, and live in impoverished neighborhoods reported the highest levels of pain and functional limitations on a self-reported measure of knee OA (Cleveland et al., 2013). Additionally, environmental neighborhood characteristics, such as safety and social cohesion, are related to physical and mental health outcomes in individuals with knee OA (Luong et al., 2012). Similarly, neighborhood disadvantage has been linked to reduced sleep duration, poor sleep efficiency, and increased wake time after sleep onset (WASO) (Troxel et al., 2018). While SES is often considered a robust predictor of health outcomes, it is paramount to consider the combined implications of SES and neighborhood

characteristics on health outcomes to get a complete picture of risks and protective factors associated with knee OA clinical symptoms.

A recent novel way to quantify components of geography-specific socioeconomic disadvantage that incorporates neighborhood characteristics is the National Area Deprivation Index (NADI) v.3.0 from Neighborhood Atlas (Kind & Buckingham, 2018). NADI provides a composite measurement of overall neighborhood deprivation in the form of a national decile comprised of 17 socioeconomic variables in the domains of education, income, employment, and housing quality. A composite such as NADI better reflects the multidimensional nature of a community's impact by capturing potential stressors (i.e., safety, social cohesion) that may alter both pain severity (Fuentes et al., 2007) and sleep (Troxel et al., 2018). A recent study by Jackson et al. (2022) revealed that NADI is associated with pain severity outcomes in lower back pain. Another study by Rumble et al. (2021) demonstrated that neighborhood-level SES (NADI) significantly impacted objective sleep quality in a population of chronic low back pain compared to pain-free controls.

Rationale for the Current Study

Despite the prevalence of sleep disturbance in individuals with symptomatic knee OA, minimal research has examined the bi-directional relationship between sleep and pain (Parmelee, Tighe, & Dautovich, 2015). Further little analysis has been conducted on the longitudinal trajectory of sleep and its association with clinical pain and poor physical function. Thus, the relationship between sleep quality, pain catastrophizing, and clinical pain outcomes in knee OA warrants further examination. Increased understanding of the mechanisms that underlie the sleep and pain relationship is crucial due to the high

modifiability of sleep and subsequent reduction of pain catastrophizing, which can potentially alter pain outcomes (Lerman et al., 2017). Therefore, the present study aimed to characterize objective and subjective sleep variables among a racially/ethnically diverse sample of adults with knee OA across time. Secondly, this study longitudinally examined the associations of our sleep quality variables with our clinical pain outcomes, including physical function. Thirdly, this study examined racial/ethnic and neighborhood disadvantage differences with our outcomes at baseline and across time. Lastly, this study investigated the mediating role of sleep quality in the relationship between pain catastrophizing and clinical pain severity at baseline. The present study addressed these questions with the following aims and tested the related hypotheses.

SPECIFIC AIMS AND HYPOTHESES

Specific Aim 1: To characterize group differences in subjective and objective sleep between individuals with and without symptomatic knee OA pain across a two-year observational period.

***Hypothesis 1a*:** At baseline, individuals with symptomatic knee OA will have significantly worse subjective and objective sleep compared to controls (i.e., without knee pain). Worse subjective sleep is self-reported as greater scores on the Insomnia Severity Index (ISI), while worse objective sleep is reflected as lower actigraphic sleep efficiency.

***Hypothesis 1b*:** Over the two-year observational period, individuals with symptomatic knee OA will show a greater worsening of subjective/objective sleep compared to controls.

Specific Aim 2: To evaluate the associations of objective/subjective sleep quality with clinical knee pain severity and physical function over two years among adults with symptomatic knee OA.

Hypothesis 2a: At baseline, poor objective/subjective sleep quality will be significantly associated with greater clinical pain severity and poorer physical function in individuals with symptomatic knee OA pain, including (a) greater clinical pain severity at rest, (b) greater movement-evoked pain (MEP) severity, and (c) worse lower extremity physical function.

Hypothesis 2b: Poor objective/subjective sleep quality at baseline will be associated with greater worsening of clinical pain severity, MEP severity, and physical function over the two-year observational period for individuals with symptomatic knee OA.

Specific Aim 3: To examine the influence of race/ethnicity and neighborhood disadvantage on objective/subjective sleep quality, clinical pain severity, movement-evoked pain, and physical function among individuals with symptomatic knee OA.

Hypothesis 3a: At baseline, non-Hispanic Black (NHB) participants and participants living in areas with high neighborhood disadvantage will report (a) poorer objective/subjective sleep quality, (b) greater clinical pain severity quality, and (c) reduced physical function in comparison to their non-Hispanic White (NHW) counterparts and those living in areas with low neighborhood disadvantage. For this aim, neighborhood disadvantage will be measured using the 2019 National Area Deprivation Index (NADI) v.3.0. from the Neighborhood Atlas, which assesses neighborhood-level socioeconomic disadvantage.

Hypothesis 3b: Poor objective/subjective sleep quality at baseline and over the two-year observational period will be associated with greater worsening of clinical pain severity, MEP severity, and physical function in NHB individuals compared to NHW individuals and High NADI individuals compared to low NADI individuals with symptomatic knee OA.

Specific Aim 4: To examine the associations of pain catastrophizing with objective/subjective sleep quality and clinical pain severity, and to determine whether the strength of these associations significantly differs according to

race/ethnicity and neighborhood disadvantage among individuals with symptomatic knee OA.

Hypothesis 4a: Baseline pain catastrophizing will be significantly associated with worse sleep quality and clinical pain severity at baseline. Further, sleep quality will mediate the association between pain catastrophizing and clinical pain severity, such that greater baseline pain catastrophizing will be associated with poorer objective/subjective sleep quality, which will, in turn, be associated with greater clinical pain severity.

Hypothesis 4b: Race/ethnicity and neighborhood disadvantage will moderate the mediating effect that sleep quality has on the pain catastrophizing and clinical pain severity relationship. Specifically, the strength of mediation will be greatest for NHBs (compared to NHWs) and those experiencing the most neighborhood disadvantage.

METHODS

Study Design Overview

The study represents a secondary analysis of data collected as part of a larger parent project known as the Understanding Pain and Limitations in Osteoarthritic Disease (UPLOAD) study; data were collected between 2014 and 2019. The UPLOAD study was a multi-site investigation conducted in collaboration between the University of Florida (UF) and the University of Alabama at Birmingham (UAB). The study sought to elucidate ethnic/racial differences in knee OA-related pain severity and limitations in NHB and NHW middle-aged to older adults over a two-year observational timeframe. As such, some participants included in this study were recruited at UF, while the remaining participants were recruited from UAB. The measures and procedures described below are limited to those involved in the current study. For community-based recruitment, participants were eligible to participate in the study if they were between 45 and 85 years of age, self-reported their ethnicity as non-Hispanic and their race as either African American/Black or Caucasian/White, and had unilateral or bilateral symptomatic knee OA based upon American College of Rheumatology clinical criteria (Altman et al., 1986). Patients were recruited with an emphasis on equalizing the number of NHB and NHW adults for this study. We used several strategies to optimize enrollment. First, we advertised around our local institutions and throughout the local communities, including advertisements in local retail establishments, at bus stops and on buses, and in local print and electronic media.

Second, we participated in community health fairs and education programs sponsored by entities within our universities. Third, to ensure adequate minority enrollment, we adopted targeted outreach programs such as attending local minority events, partnering with minority organizations, and partnering with local churches. These approaches have been successfully utilized to enhance recruitment of under-represented minorities in past studies. In addition to these community-based approaches, we recruited from Internal Medicine and Rheumatology Clinics at University-affiliated hospitals. Given the documented sex differences in the prevalence rates of knee OA populations (O'Connor, 2007), we recruited more women (60-65%) than men (35-40%). However, recruitment was monitored to ensure relatively equal ratios of women to men were recruited within each racial group. All procedures were reviewed and approved by the institutional review boards at the University of Florida and the University of Alabama at Birmingham. Participants provided written consent and were compensated for their participation.

Procedure Overview

Individuals with symptomatic knee OA interested in being included in this study were screened for eligibility during an initial telephone screening and a subsequent Health Assessment Session. The first visit, or Health Assessment Session, lasted approximately 2.5 hours, during which participants were evaluated by a study-affiliated nurse or rheumatologist to 1) confirm the clinical diagnosis of knee OA, 2) further verify eligibility criteria, and 3) collect baseline information. Participants provided information regarding their racial background and socioeconomic status in this exam portion. As part of the Health Assessment Session, participants self-reported their clinical symptoms of knee OA including pain and physical function, using validated questionnaires. They also completed

an objective measure of lower extremity function and movement-evoked pain (i.e., SPPB), a radiographic exam of the affected knee(s), and a self-reported sleep assessment. Radiographic imaging of the affected knee joint was scored for the severity of radiographic OA using the KL grading system (score range 0–4) (Kellgren & Lawrence, 1957). Finally, participants were given an actigraph (Actiwatch2, Philips Respironics) for objective sleep monitoring and provided sleep diaries to complete before returning for follow-up.

Approximately 1-2 weeks following the Health Assessment Session, those participants who were still eligible to continue in the study returned. Actiwatches and sleep diaries were retrieved at the beginning of this visit. They then completed a Quantitative Sensory Testing Session (QST) that lasted approximately 2.5 hours and included different assessments of endogenous pain modulatory processes. The results of the QST session are not a focus of this study and will not be addressed unless necessary. All study procedures were carried out at the Center for Clinical and Translational Science-affiliated Clinical Research Units (CRU) at UAB and UF. This study was intended to be longitudinal in design. Participants completed the entire procedure at baseline and returned at two years to repeat identical procedures. A flow diagram depicting matriculation through the proposed study can be seen in **Figure 1**. Study procedures and measures are further described below.

Telephone Screening

All of the study's participants completed a screening via telephone to determine initial eligibility for study inclusion. The following demographic and physical health data were obtained as part of the screening: self-reported ethnicity and race, sex, and age, as well as a health history of painful experiences related to knee OA. Individuals were asked

about current or past health conditions that may exclude them from being admitted into the study. Further exclusion criteria included: a) age under 45 years, b) past or present neurological disease, c) past or present cardiovascular disease, d) severe psychiatric disorder requiring hospitalization within the past 12 months or characterized by active suicidal ideation, e) diminished cognitive function determined to increase the risk of study participation, f) current pregnancy, g) contradictions to MRI scanning, and h) past or present systemic rheumatic disease/conditions. Individuals who endorse current knee pain and meet initial study inclusion criteria were asked to present approximately one to two weeks later for the Health Assessment Session.

Health Assessment Session

Additional health information was collected during the Health Assessment Session to confirm ongoing study eligibility. For example, we collected three consecutive resting blood pressure measurements. Participants were excluded from the remainder of the study if there was any evidence of uncontrolled hypertension (>150/95). Participants with continued eligibility completed a bilateral knee joint evaluation by an experienced examiner (i.e., the study rheumatologist or nurse practitioner) to confirm their diagnosis of osteoarthritis using the American College of Rheumatology clinical criteria for knee OA (Altman et al., 1986). Based on the physical exam and the participant's self-report, the affected knee was used for participants with unilateral knee OA and the most symptomatic knee was selected for those with bilateral knee OA. The chosen knee was designated as the index knee for QST purposes. Height and weight were measured for the calculation of body mass index. Participants completed several measures of clinical pain and knee OA symptoms, including the Insomnia Severity Index (ISI) and the Western Ontario McMaster

University Osteoarthritis Index (WOMAC); both described in greater detail below. Also, as part of the Health Assessment Session, participants reported demographics, their annual household income, the number of occupants residing in the household, their educational attainment, and their current employment status. Additional information collected included the presence of health insurance, alcohol/nicotine habits, and current medications (if any).

Measures

Neighborhood disadvantage – National Area Deprivation Index. Geographically focused socioeconomic status was captured for this study using the 2019 Area Deprivation Index v.3.0. (Kind & Buckingham, 2018), which is calculated from 17 socioeconomic variables in the domains of education, income, employment, and housing quality. Each participant was assigned a National Area Deprivation Index (NADI) value according to the census block group (9-digit zip code) they resided in during study participation. NADI values range from 1 to 100, with higher scores indicating greater neighborhood disadvantage. "Neighborhood" is defined as a Census Block Group. Two groups will be developed using NADI using a median split. Individuals below the median were characterized as the low NADI group (<71), and individuals above the median were characterized as the high NADI group (>71).

Patient-Reported Outcomes Measurement Information Systems Depression Scale. Depressive symptoms were assessed using an 8-item scale developed by the Patient-Reported Outcomes Measurement Information System (PROMIS). PROMIS is a National Institutes of Health Roadmap initiative aiming to provide precise, valid, reliable, and standardized questionnaires measuring patient-reported outcomes across physical, mental, and social health domains (Cella et al., 2010). The PROMIS depression scale was

developed using item response theory to promote greater precision and decrease the respondent burden. Specifically, the PROMIS depression scale has been correlated and validated with other commonly used depression instruments, including the Center for Epidemiological Studies Depression Scale (CES-D), the Beck Depression Inventory (BDI-II), and the Patient Health Questionnaire (PHQ-9). (Choi et al., 2014). The 8-item PROMIS depression scale asked participants how frequently in the past seven days they had experienced depression, including feeling hopeless, worthless, helpless, or depressed. These items were scored on a 5-point Likert scale ranging from 1 to 5, corresponding to responses of "Never," "Rarely," "Sometimes," "Often," and "Always." Thus, the total possible raw score was between 4 and 20. In the current study, Cronbach's alpha for PROMIS depression was .85. T scores for PROMIS instruments are standardized and based on the United States general population with a population mean of 50 and SD of 10.

Pain Catastrophizing. Pain catastrophizing was assessed using the Coping Strategies Questionnaire-Revised (CSQ-R). The CSQ-R is a 27-item assessment utilized to assess participants' use of cognitive strategies to cope with pain and maladaptive strategies such as pain catastrophizing (Rosenstiel & Keefe, 1983). The CSQ-R includes the following subscales representing six cognitive domains: distraction (five items), ignoring pain sensations (five items), distancing oneself from pain (four items), coping self-statements (four items), praying/hoping (three items), and catastrophizing (six-items). Each item is scored from 0 (never do that) to 6 (always do that) to indicate how frequently the strategy is engaged in response to pain. Each subscale is scored separately; higher scores indicate greater engagement in that respective cognitive domain. Only the pain catastrophizing

subscale was used for this study. The CSQ-R in this study had adequate internal consistency (Cronbach's $\alpha = 0.86$).

Clinical Pain Severity and Physical Function - Western Ontario McMasters University OA Index. Clinical pain severity was assessed using the Western Ontario McMasters University OA Pain Index (WOMAC). The WOMAC is frequently used in clinical and research settings to assess an individuals' retrospective self-report of clinical knee OA symptoms over the preceding 48 hours (Bellamy et al., 1988). The WOMAC includes 24 items and can be divided into pain, stiffness, and physical function subscales. The WOMAC pain and physical function subscale scores specifically were used in this study, with higher scores indicating increased knee OA pain severity/pain severity in relation to physical function. High-construct validity and test-retest reliability have been found in paper and computerized versions of the WOMAC for the overall measure and its respective subscales (Bellamy et al., 1988; Theiler et al., 2002). Cronbach's alpha for WOMAC pain and function scales was .93 and .98, respectively.

Movement-Evoked Pain and Physical Function - Short Physical Performance Battery: SPPB. The SPPB assesses lower extremity function with balance, chair, and walking tests (Guralnik et al., 1994). Specifically, participants were asked to: (1) stand with their feet together in the side-by-side, semi-tandem, and tandem positions for up to one minute; (2) rise from a seated position in a chair and return to a seated position five times; and (3) walk a four-meter course twice. If the participant did not feel it was safe to perform the activity, they received a score reflecting non-participation. For each category, based on their performance, they received a score of 0 to 4 (total score of 0 to 12). A lower score indicates worse function and a greater likelihood of disability (Guralnik et al., 1995). Additionally,

after each test, a pain severity rating was obtained on a scale from 0 (no pain) to 100 (most intense pain imaginable), indicating how painful it was for the participant to complete each test. This pain rating represented our movement-evoked pain (MEP) rating. The SPPB has been standardized and is widely used in older populations to measure lower extremity function (Guralnik et al., 2000).

Objective Sleep Quality – Actigraphic Sleep Efficiency. After completion of the HAS, the participants were briefed on the concept of actigraphy, and the experimenter reviewed actigraphy instructions, including the instructions for completing the sleep diary. Objective sleep data was acquired using the Actiwatch2 (Respironics, Bend, OR), a wrist-worn, watch-like actigraph. The Actiwatch2 is a solid-state accelerometer, or movement detector, designed to measure ambulatory activity. It was used in the scope of this study to measure daily sleep-wake patterns and record body movement. The Actiwatch2 has good reliability and criterion validity (Gironda et al., 2007; Wood et al., 2008)

The Actiwatch2 was placed on the participant's nondominant wrist, and the following instructions were provided to the participant. Actiwatches should be worn 24 hours per day, including in the bath or shower. The optimal length of wear for the watch was one week (7 days) prior to the QST session. In special circumstances, the actiwatch may be worn for up to 15 days but no less than five days before the QST. Study participants were instructed to press the "event marker" button on the Actiwatch2 at bedtime and upon waking in the morning. These events were also compared to the corresponding sleep diaries that participants completed daily. While the broader scope of sleep diaries was not the primary focus of this study, the aid of these diaries and event markers helped researchers outline a protocol for actigraphy scoring and set sleep periods. Sleep-wake patterns were

extracted from the actigraphy data using the Actiware Sleep software, which bases its algorithm on the amplitude and frequency of detected movements, which were scored in 30-s epochs.

The following measures were derived from the actigraphy data: total sleep time, sleep onset latency, wake after sleep onset time, and sleep efficiency. Total sleep time was scored as sleep in minutes from sleep onset to sleep offset. Sleep onset latency represents the time in minutes it took to transition from fully awake to asleep. Wake after sleep onset was calculated by adding the number of minutes in which participants were awake from sleep onset to final awakening. Sleep efficiency is a percentage calculated as the ratio of estimated total sleep time divided by total time spent in bed multiplied by 100, with values closer to 100% meaning the most efficient sleep. This study will focus on sleep efficiency as it is the most representative index of overall objective sleep quality and the sleep parameter most reliable and consistent to polysomnography in comparison studies (Kushida et al., 2001).

Subjective Sleep Quality – Insomnia Severity Index: (ISI). The Insomnia Severity Index (ISI) measures outcomes of perceived sleep difficulties and insomnia severity over the past two weeks. The ISI has seven items, each measured on a Likert scale from 0 to 4 (range: 0-28). The validity and reliability of the ISI have been demonstrated, and it has been used effectively across multiple populations with co-morbid diseases (Morin et al., 2011). Interpretation of scores for this measure includes the following: 0-7 = no clinically significant Insomnia; 8-14 = subthreshold insomnia; 15-21 = moderate insomnia; 22-28 = severe insomnia. However, this study will analyze the ISI as a continuous variable. Cronbach's alpha for the ISI was .84.

DATA ANALYSIS

Data Inspection

Prior to analysis, data were examined for missingness, normality, and outliers. Prior to testing hypotheses, the distribution of each variable was inspected to assess for normality, identify outliers, skewness, or other abnormalities in the distributions, and determine the need for transformation. Only actigraphic sleep at timepoint 2 met the study variables criteria for severe skewness (i.e., skewness, when divided by the standard error, is greater than 2.5). Thus, log transformation was used on this variable. Further, each variable was visually inspected to check for outliers (i.e., values ≥ 3 standard deviations above/below the mean), and it was determined that there was no need to omit outliers from data analysis. No other abnormalities in the data structure were identified.

Inferential Statistics

Aim 1: General linear models (GLM) were used for baseline data to examine differences in the individual outcome measures according to case vs. control status. For each GLM proposed, assumptions including linearity, normality, homogeneity of variances, and no cases with outliers ±3 standard deviations were met, and transformations were employed for sleep actigraphy at time point 2. Each outcome measure (e.g., actigraphic sleep efficiency, insomnia symptoms on ISI) is the dependent variable, and symptomatic knee OA status (i.e., case vs. control) is the independent variable, adjusting for identified

covariates (age, race, BMI, depressive symptoms, and site). The selection of covariates was decided based on theory and previous research showing these variables to be associated with alterations in sleep quality. For the two-year follow-up data, we incorporated repeated measures GLMs. The primary outcome variables for these longitudinal analyses are comparing baseline scores to scores at timepoint two while adjusting for identified covariates.

Aim 2: The analytic strategy for this aim focused on those participants with symptomatic knee OA to determine if actigraphic sleep efficiency and insomnia severity on the ISI are significantly associated with clinical knee pain severity, MEP, and SPPB physical function. Pearson correlations were conducted to examine the associations between these variables.

Hierarchical linear regression analyses were used to assess the ability of actigraphic sleep efficiency/insomnia symptoms on the ISI to predict change scores for pain and physical function outcomes: WOMAC clinical pain severity, WOMAC physical function, SPPB MEP, SPPB physical function after controlling for the influence of age, BMI, KL index, PROMIS depressive symptoms, and study site. Analyses were carried out in two steps. Age, BMI, KL index, PROMIS depressive symptoms, and study site were entered into the regression model as control variables (Block 1). Secondly, actigraphic sleep efficiency/insomnia symptoms on the ISI in respective models were added to the model (Block 2). There was linearity as assessed by partial regression plots and a plot of studentized residuals against the predicted values. There was the independence of residuals, as assessed by a Durbin-Watson statistic < 4. There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals

versus unstandardized predicted values. There was no evidence of multicollinearity, as assessed by tolerance values and VIF. There were no studentized deleted residuals greater than ±3 standard deviations, no leverage values greater than 0.2, and values for Cook's distance above 1. The assumption of normality was met, as assessed by Q-Q Plot, as the sample size was > 50.

Aim 3: For hypothesis 3a, six 2x2 ANCOVAs assessed main effects and interaction effects of racial/ethnic group and NADI group on sleep measures: actigraphic sleep efficiency/insomnia symptoms on the ISI as well as pain/physical function measures: WOMAC clinical pain severity, WOMAC physical function, SPPB MEP, SPPB physical function using baseline data. NADI was dichotomized into high and low groups using a median split. All models adjusted for identified covariates: age, BMI, KL index, PROMIS depressive symptoms, and study site. For each ANCOVA proposed, assumptions including linearity, normality, homogeneity of variances, and no cases with outliers ±3 standard deviations were met.

For hypothesis 3b, racial/ethnic group and NADI group were examined as effect moderators in the regression models described in hypothesis 2b above by including interaction terms (e.g., racial/ethnic group by actigraphic sleep efficiency, NADI group by Insomnia symptoms on the ISI) into each model to determine if the relationship of each predictor of interest with each outcome significantly varies between NHBs and NHWs as well as high and low NADI. Analyses were carried out in two steps, age, racial/ethnic group, or NADI group were entered into the model in Block 3. Secondly, the interaction term of either racial/ethnic group or NADI group by sleep measure (actigraphic sleep efficiency/insomnia symptoms on the ISI) in Block 4. A simple slope

analysis would be conducted for any significant interaction terms. There was linearity as assessed by partial regression plots and a plot of studentized residuals against the predicted values. There was the independence of residuals, as assessed by a Durbin-Watson statistic < 4. There was homoscedasticity, as assessed by visual inspection of a plot of studentized residuals versus unstandardized predicted values. There was no evidence of multicollinearity, as assessed by tolerance values and VIF. There were no studentized deleted residuals greater than ±3 standard deviations, no leverage values greater than 0.2, and values for Cook's distance above 1. The assumption of normality was met, as assessed by Q-Q Plot, as the sample size was > 50.

Aim 4: The analytic strategy for this aim focused on participants with symptomatic knee OA to determine if pain catastrophizing is significantly associated with actigraphic sleep efficiency, insomnia symptoms on the ISI, and WOMAC clinical pain severity at baseline. Pearson correlations were conducted to examine the associations of these variables.

The PROCESS macro, created and described by Hayes (Hayes & Rockwood, 2017), uses a bootstrapped 95% confidence interval with 5,000 resamples to test the total mediating effect (model 4). Bootstrapping is a nonparametric resampling procedure that is a viable alternative to normal-theory tests of the intervening mediator between the independent and dependent variables (Preacher & Hayes, 2008). Two simple mediations were conducted to test hypothesis 4a. One model tested the mediating effect of actigraphic sleep efficiency on the relationship between pain catastrophizing and WOMAC clinical pain severity. The other mediation examined the mediating effect of insomnia symptoms on the ISI on the relationship between pain catastrophizing and

WOMAC clinical pain severity. Mediation analysis indicates whether the total effect (path c) of pain catastrophizing on WOMAC clinical pain severity is comprised of a significant mediated effect (a × b). Path a denotes the effect of pain catastrophizing on sleep quality measure, whereas path b is the effect of sleep quality measure on WOMAC clinical pain severity. Study variables that emerged as significantly associated with pain catastrophizing and/or pain severity were included in the mediation model as statistical covariates. These variables included age, BMI, KL index, PROMIS depression symptoms, and site.

Four moderated mediation analyses were conducted using PROCESS macro (model 7) to test the moderating effects of racial/ethnic group and NADI group for hypothesis 4b. The initial two models tested the moderating effect of racial/ethnic group on the two simple mediations from hypothesis 4a, examining the mediating role of either actigraphic sleep efficiency or insomnia symptoms on the ISI. The following two models tested the moderating effect of NADI group on the two simple mediations from hypothesis 4a, examining the mediating role of either actigraphic sleep efficiency or insomnia symptoms on the ISI. Study variables that emerged as significantly associated with pain catastrophizing and/or pain severity were included in the mediation model as statistical covariates. These variables included age, BMI, KL index, PROMIS depression symptoms, and site.

RESULTS

Participant Characteristics

The descriptive characteristics of the 253 study participants are presented in **Table 1**. There were 65 healthy controls in the sample and 188 individuals living with symptomatic knee OA. The sample was racially diverse between non-Hispanic white (NHW) and non-Hispanic Black (NHB) participants. NHWs compromised 48% and NHBs compromised 52% of the symptomatic knee OA sample, while the healthy control group comprised 62% NHW and 38% NHB. There was no significant group difference in age between healthy controls, 57.25 years (SD = 8.3), and individuals living with symptomatic knee OA, 57.96 (SD = 7.76). There were more females than males for both healthy control and symptomatic knee OA groups, 65% and 63%, respectively. There was a significant group difference in the National Area of Deprivation Index (NADI) between groups revealing that individuals living with symptomatic knee OA in this sample live in more disadvantaged areas than healthy controls ($p = .004$). Individuals living with symptomatic knee OA reported increased PROMIS depressive symptoms ($p < .001$) and Kellgren-Lawrence (KL) index score ($p < .001$) than healthy counterparts. There was no significant group difference in body mass index (BMI) between healthy controls and individuals living with knee OA ($p = .305$).

For aim 3, Group differences between NHB and NHW partriciapnts are presented in **Table 2.** There was a significant group difference NADI between groups revealing that

NHBs in this sample live in more disadvantaged areas than NHWs ($p < .004$). NHBs are reporting significantly greater pain catastrophizing ($p <.001$), WOMAC clinical pain severity ($p < .001$) and MEP ($p <.001$). NHBs are reporting significantly greater WOMAC physical function related pain ($p <.001$) and reductions to SPPB physical function score ($p = .014$). NHBs are reporting significantly greater reductions to actigraphic sleep efficiency ($p = .025$) than NHWs but no significant group differences on insomnia symptoms on the ISI ($p = .820$).

Group differences between NADI group (low or high) on outcome variables of interest were presented in **Table 3.** Results revealed that those living in the high NADI group are reporting significantly greater WOMAC clinical pain severity ($p < .028$) and WOMAC physical function related pain ($p = .008$). Individuals living in areas with high NADI are reporting significantly greater reductions ot SPPB physical function score ($p = .001$). There were no sinificant group differences between NADI groups on actigraphic sleep efficiency, insomnia symptoms on the ISI, and SPPB MEP.

Specific Aim 1

Hypothesis 1a: After controlling for age, race, BMI, depressive symptoms, and site, an adjusted model revealed that individuals with knee OA reported significantly greater insomnia symptoms on ISI compared to controls, $F (1, 235) = 17.02)$, $p < .001$ partial $\eta 2 = .07$. In our second adjusted model, it was revealed that individuals with knee OA reported significantly greater reductions to actigraphic sleep efficiency compared to controls, $F (1, 198) = 5.19$, $p < .05$, partial $\eta 2 = .03$). Individuals with symptomatic knee OA also had significantly greater wake after sleep onset ($p < .05$) but did not have significantly different total sleep time ($p = .35$) than controls. These findings shown in

Table 1 were consistent with the hypothesis that individuals with knee OA would have reductions in actigraphic sleep efficiency and increased insomnia symptoms on ISI compared to controls.

Hypothesis 1b: Our adjusted model (**Figure 2**) adjusting for covariates demonstrated that there was not a statistically significant within-subject effect at different time points for insomnia symptoms on ISI for either group, $F (1, 165) = 1.36$, $p = .246$, partial $\eta 2 = .001$. There was an overall between-group effect of insomnia symptoms over the two years, $F (1, 165) = 9.934$, $p = .002$, partial $\eta 2 = .06$. There was also no significant within-subject effect of group by time for insomnia symptoms on ISI, $F (1, 165) = 2.182$, $p = .142$, partial $\eta 2 = .01$. Thus, individuals with symptomatic knee OA had increased insomnia symptoms on ISI at both time points but minimal change over time occurred.

The second adjusted model (**Figure 3**) revealed that mean actigraphic sleep efficiency statistically differed between baseline and time point two, $F (1, 107) = 9.672$, $p = .002$, partial $\eta 2 = .08$. There was also an overall between-group effect of actigraphic sleep efficiency over the two years, $F (1, 107) = 4.41$, $p = .038$, partial $\eta 2 = .04$. However, there was no significant within-subject difference of group by time, $F (1, 107) = .738$, $p = .392$, partial $\eta 2 = .07$. Thus, individuals with symptomatic knee OA had greater reductions to actigraphic sleep efficiency compared to controls at both time points; however, the rate of sleep quality changes at two years was similar between groups.

Specific Aim 2

Hypothesis 2a: Pearson correlations were conducted for individuals living with symptomatic knee OA to determine associations of actigraphic sleep efficiency, insomnia symptoms on the ISI, WOMAC clinical pain severity, WOMAC physical function, SPPB movement-evoked pain (MEP), and SPPB physical function, which can be found in **Table 4**. Better actigraphic sleep efficiency was found to be significantly correlated with less WOMAC clinical pain severity ($r = -.31$, $p < .01$), less WOMAC physical function ($r = -.31$, $p < .01$), and significantly correlated with increasing SPPB physical function ($r = .32.$, $p < .01$). Actigraphic sleep efficiency was not significantly correlated with SPPB MEP ($r = -.11$, $p = .19$). Greater severity of insomnia symptoms on the ISI was found to be significantly correlated with greater WOMAC clinical pain severity ($r = .41$, $p < .01$), greater WOMAC physical function ($r = .37$, $p < .01$), greater SPPB MEP ($r = .37$, $p < .01$), and reductions to SPPB physical function ($r = -.15.$, $p < .05$).

Hypothesis 2b: *Actigraphic Sleep Actigraphy, WOMAC Clinical Pain Severity Change, and WOMAC Physical Function Change.* In our first regression model, age, BMI, KL index, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 0 % of the variance in WOMAC clinical pain severity change (**Table 5**). After adjusting for control variables, introducing actigraphic sleep efficiency at Block 2 explained an additional 2 % of the variance in WOMAC clinical pain severity change. When all six independent variables were included in Block 2 of the regression model, only actigraphic sleep efficiency was a statistically significant predictor of WOMAC clinical pain severity change. However, the overall regression model was insignificant $F (6, 100) = 1.494$, $p = .188$.

In our second regression model, age BMI, KL index, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 0% of the variance in WOMAC physical function change (**Table 5**). After adjusting for control variables, introducing actigraphic sleep efficiency at Block 2 explained an additional 2 % of the variance in WOMAC physical function change. Only actigraphic sleep efficiency was a statistically significant predictor of WOMAC physical function change when all six independent variables were included in Block 2 of the regression model. However, the overall regression model was insignificant $F(6, 99) = 1.364$, $p = .236$.

Actigraphic Sleep Efficiency, SPPB Movement-Evoked Pain Change, and SPPB Physical Function Change. In our third regression model, BMI, KL index, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 2% of the variance in SPPB MEP change (**Table 5**). After adjusting for control variables, introducing actigraphic sleep efficiency at Block 2 explained an additional 1 % of the variance in SPPB MEP change. When all six independent variables were included in Block 2 of the regression model, only the study site was a statistically significant predictor of SPPB MEP change. However, the overall regression model was insignificant $F (6, 99) = 1.495$, $p = .188$.

In our fourth regression model, age, BMI, KL index, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 0% of the variance in SPPB physical function change (**Table 5**). After adjusting for control variables, introducing actigraphic sleep efficiency at Block 2 explained an additional 0 % of the variance in SPPB physical function change. When all six independent variables were included in Block 2 of the regression model, no variables were a statistically significant predictor of

SPPB physical function change, and the overall regression model was insignificant F (6, 100) = 1.121, p = .355.

Insomnia Severity Index, WOMAC Clinical Pain Severity Change, and WOMAC Physical Function Change. In our fifth regression model, Age, BMI, race, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 0 % of the variance in WOMAC clinical pain severity change (**Table 6**). After adjusting for control variables, introducing insomnia symptoms on the ISI at Block 2 explained an additional 2% of the variance in WOMAC clinical pain severity change. When all six independent variables were included in Block 2 of the regression model, no variables were a statistically significant predictor of WOMAC clinical pain severity change, and the overall regression model was insignificant F (6, 119) = .871, p = .519.

In our sixth regression model, age, BMI, KL index, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 0% of the variance in WOMAC physical function change (**Table 6**). After adjusting for control variables, introducing insomnia symptoms on the ISI at Block 2 explained an additional 2% of the variance in WOMAC physical function change. When all six independent variables were included in Block 2 of the regression model, no variables were a statistically significant predictor of WOMAC physical function. However, the overall regression model was insignificant F (6, 118) = 1.127, p = .351.

Insomnia Severity Index, SPPB Movement-Evoked Pain Change, and SPPB Physical Function Change. In our seventh regression model, age, BMI, KL index, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 3% of the variance in SPPB MEP change (**Table 6**). Introducing insomnia symptoms on the ISI at

Block 2, explained an additional 1 % of the variance in SPPB MEP change after adjusting for control variables. When all six independent variables were included in Block 2 of the regression model, only the study site was a statistically significant predictor of SPPB MEP change. However, the overall regression model was insignificant F (6, 117) = 1.707, p = .125.

In our eighth regression model, age, BMI, KL index, PROMIS depressive symptoms, and study site were entered at Block 1, explaining 1% of the variance in SPPB physical function change (**Table 6**). After adjusting for control variables, introducing insomnia symptoms on the ISI at Block 2 explained an additional 0 % of the variance in SPPB physical function change. When all six independent variables were included in Block 2 of the regression model, no variables were a statistically significant predictor of SPPB physical function change, and the overall regression model was insignificant F (6, 119) = 1.084, p = .376.

Specific Aim 3

Hypothesis 3a: Six two-way ANCOVAs were conducted to examine the effects of racial/ethnic group and NADI group on our six outcomes of interest: actigraphic sleep efficiency, insomnia symptoms on the ISI, WOMAC clinical pain severity, SPPB MEP, WOMAC physical function and SPPB physical function. All ANCOVAs controlled for age, BMI, KL index, PROMIS depressive symptoms, and study site.

Racial/ethnic group by NADI group on Actigraphic Sleep Efficiency/Insomnia Symptoms on ISI. Our first ANCOVA (**Figure 4**) was conducted to examine racial /ethnic groups by NADI groups on actigraphic sleep efficiency after controlling for covariates. There was no statistically significant two-way interaction between racial/ethnic groups and NADI

groups on actigraphic sleep efficiency, F (8, 141) = .721, p = .397, partial η2 = .005. Therefore, an analysis of the main effects for racial/ethnic groups and NADI groups was performed. The main effect of racial/ethnic group did not show a statistically significant difference in adjusted marginal mean actigraphic sleep efficiency for NHB (76.933) versus NHW (80.09), 95% CI [-6.701, .383], p = .08. Adjusted marginal mean actigraphic sleep efficiency in the high NADI NHB group (76.889) was not statistically lower than the high NADI NHW (78.480), 95% CI [-6.787, 3.606], p = .546. Adjusted marginal mean actigraphic sleep efficiency in the low NADI NHB group (76.976) was not statistically lower than the low NADI NHW (81.704), 95% CI [-9.707, .251], p = .063. The main effect of the NADI group did not show a statistically significant difference in adjusted marginal mean actigraphic sleep efficiency for) high NADI (77.684) versus low NADI (79.340, 95% CI [-1.875, 5.186], p = .355. Adjusted marginal mean actigraphic sleep efficiency in the high NADI NHB group (76.889) was not statistically lower than the low NADI NHB (76.976), 95% CI [-4.996, 4.822], p = .972. Adjusted marginal mean actigraphic sleep efficiency in the high NADI NHW group (81.704) was not statistically lower than the low NADI NHW (78.480), 95% CI [-8.471, 2.023], p = .972.

Our second ANCOVA (**Figure 5**) was conducted to examine racial /ethnic groups and NADI groups on insomnia symptoms on the ISI after controlling for covariates. There was no statistically significant two-way interaction between the racial/ethnic groups and NADI groups on insomnia symptoms on the ISI, F (8, 166) = 2.487, p = .117, partial η2 = .015. Therefore, an analysis of the main effects for racial/ethnic groups and NADI groups was performed. The main effect of racial/ethnic group did not show a

statistically significant difference in adjusted marginal mean insomnia symptoms on the ISI for NHB (8.994) versus NHW (9.696), 95% CI [-2.555, 1.150], p = .455. Adjusted marginal mean insomnia symptoms on the ISI in the high NADI NHB group (9.298) was not statistically higher than the high NADI NHW (8.515), 95% CI [-1.890, 3.456], p = .564. Adjusted marginal mean insomnia symptoms in the low NADI NHB group (8.690) were not statistically lower than the low NADI NHW (10.877), 95% CI [-4.764, .388], p = .095. The main effect of the NADI group did not show a statistically significant difference in adjusted marginal mean insomnia symptoms on the ISI for high NADI (8.906) versus low NADI (9.784), 95% CI [-2.754, 0.999], p = .357. Adjusted marginal mean insomnia symptoms on the ISI in the high NADI NHB group (9.298) was not statistically higher than the low NADI NHB (8.690), 95% CI [-1.900, 3.115], p = .633. Adjusted marginal mean insomnia symptoms on the ISI in the high NADI NHW group (10.877) was not statistically higher than the low NADI NHW (8.515), 95% CI [-5.132, .407], p = .094.

Racial/ethnic group by NADI group on WOMAC Clinical Pain Severity/SPPB MEP. Our third ANCOVA (**Figure 6**) was conducted to examine racial /ethnic groups and NADI groups on WOMAC clinical pain severity after controlling for covariates. There was no statistically significant two-way interaction between racial/ethnic groups and NADI groups on WOMAC clinical pain severity, F (8, 177) = .358, p = .550, partial η2 = .002. Therefore, an analysis of the main effects for racial/ethnic groups and NADI groups was performed. The main effect of racial/ethnic group did show a statistically significant difference in adjusted marginal mean WOMAC clinical pain severity for NHB (8.771) versus NHW (6.797), 95% CI [.781, 3.165], p = .001. Adjusted marginal mean WOMAC

clinical pain severity in the high NADI NHB group (8.942) was statistically higher than the high NADI NHW (6.607), 95% CI [6.25, 4.046], p = .008. Adjusted marginal mean WOMAC clinical pain severity in the low NADI NHB group (8.599) was not statistically higher than the low NADI NHW (6.988), 95% CI [-0.53, 3.275], p = .058.The main effect of the NADI group did not show a statistically significant difference in adjusted marginal mean WOMAC clinical pain severity for high NADI (7.774) versus low NADI (7.794), 95% CI [-1.227, 1.190], p = .975. Adjusted marginal mean WOMAC clinical pain severity in the high NADI NHB group (8.942) was not statistically higher than the low NADI NHB (8.599), 95% CI [-1.276, 1.962], p = .676. Adjusted marginal mean WOMAC clinical pain severity in the high NADI NHW group (6.607) was not statistically higher than the low NADI NHW (6.988), 95% CI [-2.159, 1.396], p = .672.

Our fourth ANCOVA (**Figure 7**) was conducted to examine racial /ethnic groups and NADI groups on SPPB MEP after controlling for covariates. There was no statistically significant two-way interaction between the racial/ethnic groups and NADI groups on SPPB MEP, F (8, 178) = .418, p = .519, partial η2 = .002. Therefore, an analysis of the main effects for racial/ethnic groups and NADI groups was performed. The main effect of racial/ethnic group did show a statistically significant difference in adjusted marginal mean SPPB MEP for NHB (30.700) versus NHW (17.047), 95% CI [6.131, 21.174], p = <.001. Adjusted marginal mean SPPB MEP in the high NADI NHB group (32.563) was statistically higher than the high NADI NHW (16.443), 95% CI [5.309, 26.930], p = .004. Adjusted marginal mean SPPB MEP in the low NADI NHB group (28.836) was statistically higher than the low NADI NHW (17.651), 95% CI [.708, 21.663], p = .037. The main effect of the NADI group did not show a statistically

significant difference in adjusted marginal mean SPPB MEP for high NADI (24.503) versus low NADI (23.244), 95% CI [-6.370, 8.888], p = .745. Adjusted marginal mean SPPB MEP in the high NADI NHB group (32.563) was not statistically higher than the low NADI NHB (28.836), 95% CI [-6.501, 13.954], p = .473. Adjusted marginal mean SPPB MEP in the high NADI NHW group (16.443) was not statistically higher than the low NADI NHW (17.651), 95% CI [-12.405, 9.986], p = .832.

Racial/ethnic group by NADI group on WOMAC Physical Function/SPPB Physical Function. Our fifth ANCOVA (**Figure 8**) was conducted to examine racial /ethnic groups and NADI groups on WOMAC physical function after controlling for covariates. There was not a statistically significant two-way interaction between the racial/ethnic groups and NADI groups on WOMAC physical function, F (8, 178) = .014, p = .905, partial η2 = .000. Therefore, an analysis of the main effects for racial/ethnic group and NADI group was performed. The main effect of racial/ethnic group did show a statistically significant difference in adjusted marginal mean WOMAC physical function for NHB (29.112) versus NHW (21.069), 95% CI [4.227, 11.860], p = <.001. Adjusted marginal mean WOMAC physical function in the high NADI NHB group (29.465) was statistically higher than the high NADI NHW (21.191), 95% CI [2.789, 13.760], p = .003. Adjusted marginal mean WOMAC physical function in the low NADI NHB group (28.759) was statistically higher than the low NADI NHW (20.946), 95% CI [2.496, 13.129], p = .004.The main effect of the NADI group did not show a statistically significant difference in adjusted marginal mean WOMAC physical function for high NADI (25.328) versus low NADI (24.853), 95% CI [-3.395, 4.347], p = .975. Adjusted marginal mean WOMAC physical function in the high NADI NHB group (29.465) was not

statistically higher than the low NADI NHB (28.759), 95% CI [-4.483, 5.897], p = .788. Adjusted marginal mean WOMAC physical function in the high NADI NHW group (21.191) was not statistically higher than the low NADI NHW (20.946), 95% CI [-5.435, 5.925], p = .932.

Our sixth ANCOVA (**Figure 9**) was conducted to examine racial /ethnic groups and NADI groups on SPPB physical function after controlling for covariates. There was not a statistically significant two-way interaction between the racial/ethnic groups and NADI groups on SPPB physical function, F (8, 178) = .001, p = .979, partial η2 = .000. Therefore, an analysis of the main effects for racial/ethnic group and NADI group was performed. The main effect of racial/ethnic group did not show a statistically significant difference in adjusted marginal mean SPPB physical function for NHB (9.130) versus NHW (9.598), 95% CI [-.939, .003], p = .051. Adjusted marginal mean SPPB physical function in the high NADI NHB group (8.875) was not statistically lower than the high NADI NHW (9.336), 95% CI [-1.138, .215], p = .180. Adjusted marginal mean SPPB physical function in the low NADI NHB group (9.386) was not statistically lower than the low NADI NHW (9.860), 95% CI [-1.130, .181], p = .155. The main effect of the NADI group did show a statistically significant difference in adjusted marginal mean SPPB physical function for high NADI (9.105) versus low NADI (9.623), 95% CI [-.995, -.040], p = .034. Adjusted marginal mean SPPB physical function in the high NADI NHB group (8.875) was not statistically lower than the low NADI NHB (9.386), 95% CI [-1.151, .129], p = .117. Adjusted marginal mean SPPB physical function in the high NADI NHW group (9.336) was not statistically lower than the low NADI NHW (9.860), 95% CI [-1.225, .177], p = .142.

Hypothesis 3b: *Actigraphic Sleep Efficiency/Insomnia Symptoms on the ISI by Racial/Ethnic Group.* The impact of racial/ethnic group and its interaction with our two sleep measures (either actigraphic sleep efficiency or insomnia symptoms on the ISI) was evaluated by adding two additional blocks in Models 1-8 (shown in **Tables 5/6**). In Block 3, racial/ethnic group was added for all models predicting our four pain/physical function outcomes: WOMAC clinical pain severity change, WOMAC physical function change, SPPB MEP change, and SPPB physical function change. For Models 1-4, the interaction of actigraphy sleep efficiency by racial/ethnic group was added in Block 4. For Models 5-8, the interaction of insomnia symptoms on ISI by racial/ethnic group was added in Block 4. Introducing racial/ethnic groups in Block 3 did not explain a significant amount of the variance change for Models 1-7. Similarly, introducing the interaction between sleep measure (either actigraphic sleep efficiency or insomnia symptoms on the ISI) and racial/ethnic group in Block 4 did not explain a significant amount of the variance change for Models 1-7.

In Model 8, Introducing racial/ethnic groups at Block 3 explained an additional 4 % of the variance and significantly predicted SPPB physical function change. However, the overall regression model remained insignificant at Block 3. Introducing the interaction of insomnia symptoms on the ISI by racial/ethnic group at Block 4 explained an additional 5% of the variance and significantly predicted SPPB physical function change. When all eight independent variables were included in Block 4 of the regression model, racial/ethnic group and the interaction of insomnia symptoms on the ISI by racial/ethnic group remained statistically significant predictors of SPPB physical change, and the overall regression model was significant $F(8, 117) = 2.445$, $p = 0.018$. Simple

slopes analysis **(Figure 10)** revealed that there was a statistically significant positive linear relationship (b = 0.129, SE = 0.048) between insomnia symptoms on ISI and SPPB physical function change in NHW individuals, p =.033, function but not in NHB individuals (b = 0.051, SE = 0.038) p = .175. This finding indicates that increased insomnia symptoms on the ISI at baseline significantly predicted increased SPPB physical function for NHW individuals.

Actigraphic Sleep Efficiency/Insomnia Symptoms on the ISI by NADI group. The impact of NADI and its interaction with our two sleep measures (either actigraphic sleep efficiency or insomnia symptoms on the ISI) were evaluated by adding two additional blocks in Models 1-8 (shown in **Tables 7/8).** In Block 3, the NADI group was added for all models predicting our four pain/physical function outcomes: WOMAC clinical pain severity change, WOMAC physical function change, SPPB MEP change, and SPPB physical function change. For Models 1-4, the interaction of actigraphy sleep efficiency by the NADI group was added in Block 4. For Models 5-8, the interaction of insomnia symptoms on ISI by the NADI group was added in Block 4. Introducing the NADI group in Block 3 did not explain a significant amount of the variance change for Models 1-8. Introducing the interaction between sleep measure (either actigraphic sleep efficiency or insomnia symptoms on the ISI) and the NADI group in Block 4 did not explain a significant amount of the variance change for Models 1-7.

In Model 8, Introducing the NADI group at Block 3 explained an additional 1 % of the variance but did not significantly predict SPPB physical function change. Introducing the interaction of insomnia symptoms on the ISI by the NADI group at Block 4 explained an additional 4% of the variance and significantly predicted SPPB physical

function change. When all eight independent variables were included in Block 4 of the regression model, only the interaction of insomnia symptoms on the ISI by the NADI group was a statistically significant predictor of SPPB physical function change; however, the overall regression model remained insignificant $F(8, 117) = 1.752$, $p = .094$.

Specific Aim 4

Hypothesis 4a: Pearson correlations were conducted for individuals living with symptomatic knee OA at baseline to determine associations of pain catastrophizing with actigraphic sleep efficiency, insomnia symptoms on the ISI, and WOMAC clinical pain severity, which can be found in **Table 4**. Greater pain catastrophizing was significantly associated with reductions in actigraphic sleep efficiency ($r = -.24$, $p < .01$), increased severity of insomnia symptoms on the ISI ($r = .35$, $p < .01$), and increased WOMAC clinical pain severity ($r = .55$, $p < .01$). Pain catastrophizing was significantly associated with age ($r = -.32$, $p < .01$), PROMIS depressive symptoms ($r = .52$, $p < .01$), BMI ($r = .22$, $p < .01$), and site ($r = -.32$, $p < .01$). Pain catastrophizing was not significantly associated with KL index ($r = .14$, $p > .05$). These clinical and demographic variables were included as covariates in further analyses.

Pain Catastrophizing, Actigraphic Sleep Efficiency, and WOMAC Clinical Pain Severity. The overall mediation model adjusted for covariates shown in **Figure 11** accounted for 39% of the total variance in WOMAC clinical pain severity ($p < .001$). The mediation effect (path $a \times b$) of pain catastrophizing on WOMAC clinical pain severity through actigraphic sleep efficiency had a point estimate of .158 and a 95% confidence interval of .009 to .353. This confidence interval suggests that, even after statistically controlling

for covariates, the mediated effect represented by a × b is significantly different from zero (i.e., the null effect) at $p < 0.05$. The directions of paths a ($t = -2.547$, $p = 0.012$) and b ($t = -2.562$, $p = .012$) are consistent with the interpretation that increased pain catastrophizing is associated with reductions to actigraphic sleep efficiency, which in turn, is associated with increased WOMAC clinical pain severity. There was also a direct effect of pain catastrophizing on WOMAC clinical pain severity, with a point estimate of 1.363 and a 95% confidence interval of .797 to 1.930. Thus, actigraphic sleep efficiency was a significant partial mediator of the association between pain catastrophizing and WOMAC clinical pain severity in this sample.

Pain Catastrophizing, Insomnia Symptoms on the ISI, and WOMAC Clinical Pain Severity. The overall mediation model adjusted for covariates shown in **Figure 12** accounted for 35% of the total variance in WOMAC clinical pain severity ($p < .001$). The mediation effect (path a × b) of pain catastrophizing on WOMAC clinical pain severity through insomnia symptoms on the ISI had a point estimate of .055 and a 95% confidence interval of -.0855 to 2.006. This confidence interval suggests that after statistically controlling for covariates, the mediated effect represented by a × b is not significantly different from zero (i.e., the null effect) at $P < 0.05$. The directions of paths a ($t = .832$, $p = 0.407$) and b ($t = 3.08$, $p = .003$) are inconsistent with the interpretation that increased pain catastrophizing is associated with increased insomnia symptoms on the ISI but consistent that it is associated with increased WOMAC clinical pain severity. There was also a direct effect of pain catastrophizing on WOMAC clinical pain severity, with a point estimate of 1.403 and a 95% confidence interval of .860 to 1.938. Thus,

insomnia symptoms on the ISI were not a significant partial mediator of the association between pain catastrophizing and WOMAC clinical pain severity in this sample.

Hypothesis 4b: *The Moderated Mediated Effect of Racial/Ethnic Group.* The hypothesized moderated mediation model was tested using the two models presented in hypothesis 4a. The first model tested if racial/ethnic groups moderate the effect of path a between pain catastrophizing and actigraphic sleep efficiency. This model demonstrated that racial/ethnic groups did not moderate the relationship between pain catastrophizing and actigraphic sleep efficiency, unconditional interaction F (1, 131) = .073, p = .788. The overall moderated mediation model, adjusted for covariates, was not supported as the index of moderated mediation was -.031 with a 95% confidence interval of -.366 to .199. Thus, the indirect effect of pain catastrophizing on WOMAC clinical pain severity via actigraphic sleep efficiency was not statistically significantly different for NHW (point estimate of .149, 95% CI of -.037 to .441) or NHB (point estimate of .118, 95% CI of -.076 to .342) individuals living with symptomatic knee OA.

The second model (shown in **Figure 13**) tested if racial/ethnic groups moderate the effect of path a between pain catastrophizing and insomnia symptoms on the ISI. This model demonstrated that racial/ethnic groups did moderate the relationship between pain catastrophizing and insomnia symptoms on the ISI, unconditional interaction F (1, 157) = 4.144, p = .044. The overall moderated mediation model, adjusted for covariates, was supported as the index of moderated mediation was -.233 with a 95% confidence interval of -.543 to -.002. The conditional indirect effect was significant for NHWs with a point estimate of .221 with a 95% CI of .014 to .4895 but not NHB (point estimate of -.012 with a 95% CI of -.188 to .144). Thus, the indirect effect of pain catastrophizing on

WOMAC clinical pain severity via insomnia symptoms on the ISI was statistically significant only for NHW individuals living with symptomatic knee OA.

The Moderated Mediated Effect of NADI Group. The hypothesized moderated mediation model was tested using the two models presented in hypothesis 4a. The first model tested if NADI groups moderate the effect of path a between pain catastrophizing and actigraphic sleep efficiency. This model demonstrated that NADI groups did not moderate the relationship between pain catastrophizing and actigraphic sleep efficiency, unconditional interaction $F(1, 131) = .037$, $p = .847$. The overall moderated mediation model, adjusted for covariates, was not supported as the index of moderated mediation was -.021 with a 95% confidence interval of -.308 to .233. Thus, the indirect effect of pain catastrophizing on WOMAC clinical pain severity via actigraphic sleep efficiency was not statistically significantly different for the low NADI group (point estimate of .160, 95% CI of -.039 to .421) or the high NADI group (point estimate of .139, 95% CI of -.029 to .366) individuals living with symptomatic knee OA.

The second model tested if NADI groups moderate the effect of path a between pain catastrophizing and insomnia symptoms on the ISI. This model demonstrated that NADI groups did not moderate the relationship between pain catastrophizing and insomnia symptoms on the ISI, unconditional interaction $F(1, 157) = 1.46$, $p = .228$. The overall moderated mediation model, adjusted for covariates, was not supported as the index of moderated mediation was -.013 with a 95% confidence interval of -.391 to .112. Thus, the indirect effect of pain catastrophizing on WOMAC clinical pain severity via insomnia symptoms on the ISI was not statistically significantly different for the low

NADI group (point estimate of .157, 95% CI of -.058 to .391) or high NADI group (point estimate of .023, 95% CI of -.136 to .191) individuals living with symptomatic knee OA.

DISCUSSION

Few studies have investigated the impact of objective and subjective sleep quality on chronic pain outcomes in knee OA populations. This study endeavored to characterize objective/subjective sleep quality across time to elucidate the role sleep disturbance may have in this knee OA sample. It then examined the impact objective/subjective sleep quality has on clinical pain severity, movement-evoked pain (MEP), and physical function scores. Results provided mixed results for our hypotheses. In our initial aim, we found that individuals living with symptomatic knee OA had significantly worse objective/subjective sleep outcomes than healthy controls at two time points; however, no increased rate of change across time. We then found associations between our objective/subjective sleep measures and our pain outcomes: clinical pain severity, MEP, and physical function, depending on the mode of measurement. However, objective/subjective sleep quality did not predict the change scores of our four pain outcomes. Adjusted models showed that racial/ethnic group differences were present for clinical pain severity, MEP, and physical function, specifically that NHB reported significantly more pain and reductions to physical function. Neighborhood disadvantage had the most significant impact on physical function score in that individuals living in areas with the most neighborhood disadvantage had significant reductions to score. The role of multimodal sleep disturbance in the relationship between pain catastrophizing and pain was examined, and significant mediating effects were found. Our results supported

the notion that sleep disturbance is essential in the relationship between pain catastrophizing and clinical pain severity. It also provides initial evidence that neighborhood disadvantage is a risk factor for physical disability in knee OA populations. Health providers should assess and target pain catastrophizing and multimodal sleep disturbance as clinical interventions when managing individuals living with symptomatic knee OA.

Specific Aim 1

This study sought to examine objective and subjective sleep quality in a knee OA population compared to healthy controls. The results were consistent with our first hypothesis and demonstrated that individuals living with symptomatic knee OA are experiencing more problematic sleep as reflected on both objective and subjective sleep quality measures compared to similarly aged healthy controls. The objective sleep differences between healthy controls and individuals with knee OA individuals can be attributed to increased awakenings as demonstrated by differences in wake-after-sleep onset (WASO) that alter sleep efficiency for those experiencing chronic pain. Notably, knee OA participants differed significantly from controls on all actigraphic sleep measures except total sleep time (TST). This finding suggests that although they are sleeping around the same amount of time, it takes individuals with symptomatic knee OA a significantly longer time frame to reach that TST. Alternately, subjective or perceived insomnia severity may be impacted by the affective states maintained by reductions to sleep and the associated symptoms such as daytime fatigue, mood dysregulation, decreased productivity, and general declines in quality of life (Baglioni et al., 2010). This

finding is consistent with prior research demonstrating the impact of the chronic pain experience and its bi-directional relationship with sleep (Whibley et al., 2019).

Our second result was inconsistent with our hypothesis as the objective sleep quality rate of change at two years was not significantly different between knee OA individuals and healthy controls. However, our study demonstrated a total between-group difference across time, meaning that individuals living with knee OA consistently reported more significant reductions in objective sleep quality during this timeframe. However, in both groups, there was a similar rate of decline in objective sleep quality at timepoint two. This finding suggests that temporal alterations to objective sleep quality may be attributed to factors not specific to living with a chronic pain condition. One such factor relevant to both groups is the process of aging which has been shown to disrupt sleep. Sleep disruptions related to aging are attributed to changes in neurobiological mechanisms and the subsequent changes to sleep architecture. Neurobiological changes to sleep are associated with reduced neurons in the preoptic area, suprachiasmatic nucleus, lateral hypothalamic area, and locus coeruleus (Mander et al., 2017). These neurobiological changes and atrophy within the prefrontal cortex led to architectural changes, such as the decline in the density of slow waves and sleep spindles. Similarly, our subjective sleep measure demonstrated that individuals living with knee OA had increased insomnia symptoms but almost no change across time. This finding suggests that the negative affective states contributing to perceived insomnia remain stable over time.

This study revealed that although a trend is consistent with our hypothesis, the lack of significance may signify that the two-year time frame may not be appropriate for

this type of examination. Two years may not be the appropriate parameter to show how the impact of living with chronic pain may change the rate at which objective or subjective sleep quality is altered. Almost no studies have evaluated pain and objective sleep quality in a macro-longitudinal design, and few studies to this date have analyzed subjective sleep quality and pain. A study by Nitter et al. (2012) revealed that perceived sleep disturbance was a predictor of chronic pain and the maintenance and worsening of pain at six and 17-year intervals. Another study evaluated self-reported sleep efficiency clusters up to 27 years later and revealed that low sleep efficiency was associated with the prevalence of comorbid health conditions (Didikoglu et al., 2020). The findings of our study and others suggest that individuals with alterations to sleep quality may be more likely to endorse pain symptoms and be diagnosed with chronic pain conditions. Studies attempting to determine psychosocial factors that might contribute to chronic pain profiles suggest that these individuals are more likely to have illness intrusiveness and low pain acceptance (Simoncsics, Konkolý Thege, & Stauder, 2022). As demonstrated by later aims in this study, these factors, alongside maladaptive coping strategies such as pain catastrophizing, may be risk factors for developing chronic pain and insomnia.

Specific Aim 2

In this study, poor sleep quality was associated with increased clinical pain severity, MEP, and impairments to physical function. This study was unique in examining the sleep-pain relationship using multimodal methodology. Our initial hypothesis was partially supported as the associations of these variables were present for most objective and subjective measures of sleep quality. This finding is consistent with prior research demonstrating the impact of reported sleep disturbances on clinical pain

outcomes within knee OA and other pain populations (Craner & Flegge, 2022; Liu et al., 2022; Parmalee et al., 2015). These relationships between multimodal sleep and pain outcomes could be explained by other studies highlighting dysfunctional beliefs (Ravyts et al., 2022) and pain catastrophizing (Campbell et al., 2015) as common risk factors among individuals with co-morbid pain and sleep symptoms.

It should be noted that the only exception was the relationship between objective sleep quality and reported movement-evoked pain. That finding suggests that actigraphic sleep efficiency may not contribute to MEP, a measurement attempting to capture the pain that occurs when engaged in physical activity during waking periods. It should be noted that although MEP measures attempt to standardize a pain response, ultimately, the pain the individual reports while engaged in these activities is still a perceptual construct. This finding suggests that the detrimental impact of poor objective sleep quality may not be captured in this form of pain reporting. Similarly, an unintended observation during our analyses revealed a minimal association between objective and subjective sleep quality, suggesting how an individual objectively sleeps may differ from their perception of insomnia symptoms. Objective sleep quality had a stronger association with objective physical function measure, suggesting that it may serve a role in predicting physical disability, such as how an individual performs with activities of daily living or contributions to sedentary behavior. This finding is impactful considering the beneficial effects of increased physical activity on sleep health (Kubala et al., 2020) and the negative impact of sedentary behavior on sleep (Kline et al., 2017). Thus, interventions that target sleep disturbance may be beneficial in addressing future pain outcomes in individuals living with symptomatic knee OA.

Our second hypothesis attempted to examine the cross-sectional associations demonstrated in hypothesis 2a by evaluating the predictive utility of objective/subjective sleep quality at baseline on the change scores of pain/physical function outcomes over variables of influence. We found that objective sleep quality at baseline did have some ability to predict change scores in our WOMAC clinical pain severity and WOMAC physical function measures; however, the overall models were insignificant. Objective sleep quality at baseline had no relationship with change scores of our SPPB MEP and SPPB physical function measures. Our subjective sleep quality measure at baseline had no relationship with the change scores of our four pain/physical function outcomes. Thus, while baseline sleep measures may reflect the existing sleep-pain relationship, they may not capture the potential changes in this bi-directional relationship over time.

We hypothesize a few possible explanations for the lack of association between multimodal sleep measures and pain/physical function changes over time. Change scores revealed minimal change for our four pain/physical outcomes at timepoint two. This finding suggests that a two-year time frame may not be an appropriate window to examine meaningful observed pain/physical function change in a knee OA population, not dissimilar from our findings for sleep variables in Aim 1. Other recent studies consistent with our original hypothesis have demonstrated both the worsening of knee pain and the relationship this worsening has with sleep using different parameters. One study demonstrated a worsening of knee pain at seven years when tibiofemoral contact stress was present at baseline (Rabe et al., 2021). A study by Song et al. (2021) demonstrated that factors specific to knee OA patients, such as the presence of depressive symptoms, less physical activity, and increased knee pain contribute to restless sleep

trajectories across eight years. We also believe that change score analysis may not fully capture how the relationship between sleep and pain may evolve. Longitudinal assessments considering how these relationships evolve, such as those in structural equation modeling, may provide a more accurate prediction of pain outcomes.

Specific Aim 3

Few studies have evaluated how the neighborhood environments of individuals with these socioeconomic limitations may contribute to adverse sleep, pain, and physical function outcomes. This gap in the literature is relevant in samples with racial/ethnic diversity as environmental and social risk factors, such as those related to low socioeconomic status, which may play a role in exacerbating the perception of both clinical pain severity and sleep disturbance (Fuller-Rowell et al., 2016; Thompson et al., 2019). For these reasons, this study attempted to evaluate group differences in race/ethnicity stratified by neighborhood disadvantage in relation to sleep, pain, and physical function outcomes. Our first hypothesis was partially supported, indicating that racial/ethnic group main effects were present and revealed that NHB individuals reported greater clinical pain severity, SPPB MEP, and WOMAC physical function-related pain than NHW individuals in the adjusted models. Main effects were absent for our objective/subjective sleep and SPPB physical function measures for racial/ethnic groups. The main effects for our neighborhood disadvantage groups were absent for all variables except SPPB physical function score. There were no significant interactions between racial/ethnic groups and neighborhood disadvantage groups for all outcomes.

NHB individuals reported increased clinical pain severity, MEP, and physical function-related pain compared to NHW individuals. It should also be noted that the main

effect of racial/ethnic groups narrowly missed significance for SPPB physical function ($p = .051$), with NHB individuals demonstrating greater reductions to physical function score than NHW individuals. Socioeconomic disparities play a significant role, as NHB individuals often experience lower socioeconomic status, limited access to healthcare, and higher levels of stress, all of which can contribute to the experience and reporting of chronic pain (Mickel et al., 2023; Murphy et al., 2019; Rios et al., 2011; Thompson et al., 2019). Cultural and social factors also come into play, as differences in pain expression norms and healthcare utilization may influence pain symptom reporting (Peacock & Patel, 2008). Furthermore, NHB individuals have historically faced systemic racism, discrimination, and healthcare disparities, leading to potential disparities in pain management and healthcare access (Green & Hart-Johnson, 2012). Biological factors are another contributing factor, as genetic and physiological variations can influence pain perception and sensitivity (Cruz-Almeida et al., 2014; Edwards et al., 2001; Goodin et al., 2014). It is crucial to approach these observations with sensitivity, as individual experiences of pain can vary greatly within and across racial/ethnic groups, and further research is necessary to understand the multifaceted nature of these disparities fully.

This lack of significance concerning our objective/subjective sleep variables may indicate that individuals in this specific knee OA sample may not have alterations to their perception of how they are sleeping or their objective sleep when stratified by racial/ethnic group and neighborhood disadvantage group. The lack of main effects for racial/ethnic groups may indicate that sleep is impacted similarly for NHB and NHW individuals when controlling clinical variables of influence. It should also be noted that the main effect of neighborhood disadvantage on objective sleep quality narrowly missed

the significance (p = .08). This trend suggests more significant reductions in objective sleep for individuals living in areas with high neighborhood disadvantage. Conversely, there is prior literature supporting the notion that neighborhood factors contribute to alterations in sleep quality due to a variety of factors that include safety, noise, cohesion, co-morbid health conditions, and modifications to mood states (Fuentes et al., 2007, Nahmod et al., 2022; Troxel et al., 2018). Other literature has demonstrated that neighborhood disadvantage can impact objective/subjective sleep quality based on contingencies. One study by Simonelli et al. (2017) revealed that reductions in subjective sleep quality were contingent on whether individuals perceived neighborhoods as noisy. Another study found that factors such as post-traumatic stress disorder symptomology and sleep fears could explain the association between subjective sleep quality and neighborhood stress (Hall Brown & Mellman, 2013). Future studies should factor in these specifiers when evaluating the impact of neighborhood disadvantage on sleep outcomes.

SPPB physical function, on the other hand, demonstrated a significant main effect of neighborhood disadvantage, revealing that those living with high neighborhood disadvantage had significant reductions to their overall function score compared to those living with low neighborhood disadvantage. Neighborhood disadvantage can significantly impact physical function due to various interconnected factors (Millar, 2020). Firstly, neighborhoods with higher levels of disadvantage often lack access to essential resources and amenities, such as quality healthcare facilities, recreational spaces, and nutritious food options. Limited access to healthcare services and healthy environments can contribute to higher rates of chronic health conditions, physical limitations, and decreased overall physical function within these neighborhoods.

Secondly, neighborhood disadvantage is often associated with higher crime levels, violence, and social disorder (Loh et al., 2018). These factors can lead to insecurity and fear, reducing opportunities for physical activity and engagement in outdoor spaces. Lack of safe and accessible recreational areas can hinder individuals' ability to exercise and engage in physical activities, leading to a decline in physical function over time. Furthermore, neighborhood disadvantage is often accompanied by social and economic inequalities, which can impact residents' ability to access education, employment opportunities, and socioeconomic resources (Fitzpatrick et al., 2015; Nyblade et al., 2019). In summary, neighborhood disadvantage can adversely affect physical function through limited access to resources, diminished opportunities for physical activity, increased exposure to crime and violence, and the amplification of social and economic disparities. Addressing neighborhood disadvantages and promoting equitable access to resources and supportive environments are crucial for improving physical function and overall health outcomes in disadvantaged communities.

Our second hypothesis was not supported as the interaction of racial/ethnic group by sleep measure at baseline did not predict change scores of pain/physical function outcomes over variables of influence for most variables. Similarly, the interaction of neighborhood disadvantage group by sleep measure at baseline did not predict pain/physical function outcomes over variables of influence. Similar to hypothesis 2b, we believe two years was not an appropriate parameter to examine meaningful change in these pain/physical function outcomes. The only model with a significant interaction effect was subjective sleep quality on the ISI by racial/ethnic group. This model demonstrated that increased insomnia symptoms had a positive relationship with

improvements in SPPB physical function scores for NHWs but not NHBs. However, this finding indicates that individuals with low SPPB physical function scores and increased insomnia symptoms at baseline regressed towards the mean at two years, a common limitation of change score analysis. However, this finding indicates that cultural and social factors may explain differences across racial/ethnic groups. We hypothesize that healthcare-seeking behavior could also be a factor, as NHW individuals may be more likely to seek medical help for sleep-related concerns, resulting in familiarity with sleep disorders/measures. Conversely, underdiagnosis or underreporting among NHB individuals may be due to a lack of awareness about sleep disorders or limited access to healthcare services, considering our consistent findings showing that NHB individuals have more significant reductions in objective sleep quality (Johnson et al., 2019).

Specific Aim 4

This study was one of the first to examine the role of pain catastrophizing with multimodal sleep and pain variables. Our initial aim revealed that pain catastrophizing was associated with objective/subjective sleep variables and clinical pain severity. This finding was consistent with previous literature recognizing pain catastrophizing as a psychosocial variable that may play a significant role in maintaining negative mood states (Edwards et al., 2011) in chronic pain populations. When individuals engage in catastrophic thinking about pain, it can heighten emotional distress and exacerbate pain perception. Heightened emotional distress and negative affect can disrupt sleep patterns and lead to sleep disturbance, increased pain sensitivity, and the development or worsening of chronic pain symptoms. Other literature has shown that sleep disturbance may be the underlying mechanism in the relationship between pain catastrophizing and

experimental pain (Campbell et al., 2015). In a different conceptualization of the interrelations between these variables, Tighe et al. (2020) used a framework suggesting that pain catastrophizing may mediate between sleep disturbance and knee OA symptom severity. A third study argued that pain mediates the relationship between sleep and levels of pain catastrophizing (Wilt et al., 2016). These conceptualizations demonstrate the interchangeability of these variables and the need to elucidate how these processes relate.

In hypothesis 4a, we chose to highlight the mediating role of objective/subjective sleep disturbance in the relationship between pain catastrophizing and clinical pain severity resembling the framework by Campbell et al. (2015). Only objective sleep quality partially mediated the relationship between pain catastrophizing and clinical pain severity. Subjective sleep quality on the ISI did not partially mediate the direct relationship between pain catastrophizing and clinical pain severity. This finding provides initial evidence that underlying health factors, such as poor objective sleep, play a significant role in explaining the impact of pain catastrophizing on clinical pain severity. These findings provide further evidence to the growing literature suggesting that sleep disturbance is essential in explaining various pain outcomes. Our findings lend credence to the value of interventions that target sleep disturbance, pain catastrophizing, or both (Da Baets et al., 2023). A growing amount of literature has demonstrated that the efficacy of short-term psychological interventions that address insomnia in chronic pain populations often leads to improved pain outcomes (Tang et al., 2012; Vitiello et al., 2014). A study that factored both sleep disturbance and pain catastrophizing by Lerman et al., (2017) demonstrated that cognitive behavioral therapy could result in meaningful

reductions in pain catastrophizing as a byproduct of improving sleep disturbance in a knee OA sample. It should be noted that these interventional studies only relied on self-reported subjective sleep data. Our models further strengthen the need for studies that evaluate the efficacy of targeting sleep disturbance using multimodal measurements such as actigraphy or polysomnographic outcomes for sleep.

Our second hypothesis examining the moderating effects of racial/ethnic or neighborhood disadvantage groups on the simple mediations from above was not supported. Only one model demonstrated a moderating effect. Although our initial mediation model in hypothesis 4a revealed that subjective sleep quality as measured by the ISI did not have a partial mediating effect in the relationship between pain catastrophizing and WOMAC clinical pain severity, there is a moderated mediating effect for NHWs and not NHBs. This finding is consistent with our finding in 3b, which revealed a similar relationship between insomnia symptoms on the ISI and change scores of physical function for NHW individuals. This finding suggests that subjective sleep measures like the ISI may be beneficial diagnostic measures for NHW individuals and may lack specificity for NHB individuals. This finding may be a potential disparity as our study consistently revealed that NHB individuals are reporting significantly greater pain severity, reductions to objective sleep quality, and living in areas with more neighborhood disadvantage. Thus, we hypothesize that this group difference and the discrepancy may be attributed to access to resources and understanding of sleep-related issues/diagnoses that may be more salient in NHW communities. Underreporting of insomnia symptoms by NHBs may be related to a lack of familiarity with sleep disorders/measures, limited access to healthcare services, repressive coping, or possible

protective factors that include resilience when dealing with hardships due to poverty, racism, and other life stresses (Rios et al., 2011). However, individual insomnia experiences can vary significantly within and across racial/ethnic groups, and further research is needed to understand these disparities comprehensively.

Strengths and Limitations

Several strengths are present in our examination of sleep, clinical pain, MEP, physical function, and neighborhood disadvantage. Global strengths of our study used a diverse sample of individuals with knee OA, included covariates in our analytic models, and used multimodal measurements of our sleep, pain, and physical function variables. This study augmented our sleep analysis by including measures such as actigraphic sleep efficiency, which reduce the reliance on self-reporting, which can be subject to biases and inaccuracies. Actigraphy improves ecological validity by accurately representing their typical sleep habits and reducing potential variables associated with lab-based testing such as polysomnography. Similarly, we used comprehensive pain outcomes such as SPPB physical function score and SPPB MEP to increase the ecological validity of our pain profile. A strength of our first aim was the ability to capture sleep data over time. With repeated measures, we improved accuracy when assessing within-person changes, enhancing the precision and accuracy of our findings. Our second aim attempted to examine pain/physical function change across time in relation to our sleep variables. A strength of our third aim was incorporating neighborhood disadvantage, a novel but often-neglected risk factor, when comparing our racial/ethnic groups on variables of interest. Using variables such as neighborhood disadvantage promotes a research framework emphasizing diverse contexts, allowing researchers to identify consistent

patterns and understand the broader social determinants of individual outcomes. Finally, a strength of our final aim included these diverse contexts when examining a possible sleep as an underlying influence on the pain catastrophizing and pain relationship.

Several limitations were also present in this study. Our longitudinal analyses in aims 2 and 3 relied on using change scores, which have notable limitations that researchers should consider. Firstly, it relies on the assumption that the difference between two-time points accurately represents the true change, disregarding potential fluctuations within that period. Moreover, measurement error can influence change scores, leading to imprecise estimates. Another limitation is the possibility of regression to the mean, where extreme initial scores tend to move closer to the mean, potentially distorting the observed change. Additionally, selective attrition and suppression effects can introduce bias and obscure underlying relationships. Change scores also discard individual values, potentially losing valuable information. Lastly, change score analysis assumes independence among changes, disregarding potential correlations.

A few limitations exist regarding our neighborhood disadvantage variable. Firstly, we chose to dichotomize our NADI variable, which leads to a loss of information by collapsing a range of values into two categories. This reduction in variability can reduce statistical power and hinder the ability to detect meaningful relationships. Dichotomization may also create artificial dichotomous relationships, obscuring more nuanced and complex associations within the continuous data. Another issue with our neighborhood disadvantage is that individuals who may be the most disadvantaged may also be the most transient regarding living situations. Although we know where they were living at the time of data collection, we do not know if their living situation was different

leading up to that point. Future studies should assess time lived at the address and implement consistent address checks at any further assessments. Another limitation of neighborhood disadvantage variables is that they often encompass a wide range of environmental factors in their measurement (i.e., safety, noise). While neighborhood disadvantage composite measures provide a general indication of the area, they lack specificity regarding which factors within the environment have the most impact.

Summary

The current study revealed that individuals living with knee OA reported significant reductions in objective/subjective sleep quality over healthy controls at baseline. These reductions to objective/subjective sleep quality compared to controls were consistent at time point two; however, the rate of change was similar. It was then revealed that objective/subjective sleep quality was associated with clinical pain severity, MEP, and physical function depending on the mode of measurement. Objective/subjective sleep measures did not predict change scores of our pain/physical function outcomes on their own. Racial/ethnic group differences were demonstrated in that NHB individuals reported increased clinical pain severity, MEP, and reductions to physical function in adjusted models compared to NHW individuals. The interaction of racial/ethnic group by insomnia symptoms on the ISI predicted change scores of physical function, suggesting that NHWs with increased insomnia symptoms at baseline had reductions to physical function scores across time; however, this was not true for NHBs. Analyses examining neighborhood disadvantage showed that individuals living in areas with high disadvantage had significant reductions in physical function scores. Multimodal sleep disturbance mediated the relationship between pain catastrophizing and

clinical pain severity. Furthermore, moderated mediation was present for NHW individuals in the relationship between pain catastrophizing and clinical pain severity via subjective sleep on the Insomnia Severity Index. Future research should elucidate the applied clinical utility of neighborhood disadvantage in these clinical populations. This study, among others, reinforces the notion that multimodal measurement can provide helpful context when treating individuals with symptomatic knee OA. At the same time, interventions that target variables such as sleep and pain catastrophizing could improve chronic pain outcomes, including physical function.

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Table 1 Descriptive characteristics and group differences between Controls and Knee OA (N = 253) for study variables at baseline

	Controls (n = 65)	**Knee OA (n=188)**	**p-value**
Variable			
Demographic Characteristics			
Age - Years	57.25 (8.81)	57.96 (7.76)	.265
Sex			
Male	23 35%	69 37%	
Female	42 65%	119 63%	
Race			
non-Hispanic Black (NHB)	25 38%	97 52%	
non-Hispanic White (NHW)	40 62%	91 48%	
National area deprivation index (NADI)	57.37 (25.48)	66.95 (22.07)	**.004**
1-20	7 11%	1 1%	
21-40	15 23%	30 16%	
41-60	7 11%	39 20%	
61-80	22 34%	50 27%	
80-100	14 21%	68 36%	
Clinical Characteristics			
PROMIS Depressive symptoms	10.98 (1.33)	13.26(6.40)	**.000**
Pain Catastrophizing	0.37 (0.55)	1.34 (1.27)	**.000**
WOMAC Clinical Pain Severity	0.60 (1.89)	7.84 (4.29)	**.000**
WOMAC Physical Function	1.82 (6.21)	25.10 (14.58)	**.000**
SPPB Movement-Evoked Pain (MEP)	1.44 (5.03)	23.97 (26.35)	**.000**
SPPB Physical function	10.69 (1.33)	9.37 (1.71)	**.000**
Body mass index (BMI)	29.38 (7.00)	32.04 (7.66)	.305
Kellgren-Lawrence (KL) Index	1.08 (1.11)	1.77 (1.47)	**.000**
Grade 0	25 40%	54 29%	
Grade 1	17 27%	29 16%	
Grade 2	14 22%	38 21%	
Grade 3	5 8%	30 16%	
Grade 4	2 3%	33 18%	
Sleep Metrics			
Sleep efficiency (%)	83.34 (8.26)	78.48 (10.69)	**.024***
Wake after sleep onset (WASO)	42.13 (19.69)	55.17 (29.56)	**.047***
Total sleep time (mm)	378.77 (57.80)	365.03 (71.95)	.348*
Insomnia Severity Index (ISI)	5.33 (5.36)	9.45 (6.44)	**.001***

PROMIS = Patient-reported Outcomes Measurement Information; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; SPPB = Short Physical Performance Battery *Adjusted for age, race, BMI, KL index, PROMIS depressive symptoms, and site

Table 2 Group differences between non-Hispanic Blacks and non-Hispanic Whites (N = 188) at baseline

	NHB (n = 97)	**NHW (n=91)**	**p-value**
Variable			
Demographic Characteristics			
Age - Years	56.44 (6.52)	59.57 (8.65)	**.007**
Sex			
Male	39 40%	30 33%	
Female	58 60%	61 67%	
National area deprivation index (NADI)	73.80 (19.14)	59.64 (22.74)	**.000**
1-20	1 1%	0 0%	
21-40	6 6%	24 26%	
41-60	15 16%	24 26%	
61-80	30 31%	20 22%	
80-100	45 46%	23 25%	
Clinical Characteristics			
Depressive symptoms (PROMIS)	13.86 (6.92)	12.62 (5.76)	.212
Pain Catastrophizing	1.73 (1.30)	.93 (1.09)	**.000**
WOMAC Pain Severity	9.09 (3.99)	6.49 (4.21)	**.000**
WOMAC Physical Function	30.11 (13.66)	19.75 (13.66)	**.000**
SPPB Pain Severity	32.06(29.89)	15.36 (18.56)	**.000**
SPPB Physical function	9.07 (1.77)	9.68 (1.60)	**.014**
Body mass index (BMI)	32.80 (7.76)	31.23(7.51)	.163
Kellgren-Lawrence (KL) Index	1.89 (1.50)	1.65 (1.45)	.265
Grade 0	28 30%	26 30%	
Grade 1	8 8%	21 24%	
Grade 2	24 25%	14 16%	
Grade 3	16 17%	14 16%	
Grade 4	19 20%	14 16%	
Sleep Metrics			
Sleep efficiency (%)	76.61 (10.52)	80.28 (10.61)	**.025**
Wake after sleep onset (WASO)	59.72 (32.09)	50.80 (26.39)	.068
Total sleep time (mm)	346.30 (66.42)	383.03 (72.87)	**.002**
Insomnia Severity Index (ISI)	9.58 (5.36)	9.32 (6.66)	.820

PROMIS = Patient-reported Outcomes Measurement Information; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; SPPB = Short Physical Performance Battery

Table 3 Group differences between NADI groups on sleep quality, clinical pain, and physical function measures at baseline

	Low NADI	**High NADI**	**Comparison**	
	M (SD)	***M (SD)***	***F***	**p**
Actigraphic Sleep Efficiency	80.22(10.03)	77.51(9.77)	2.74	.100
Insomnia Symptoms on the ISI	9.33(6.49)	9.69 (6.44)	0.14	.712
WOMAC Clinical Pain Severity	7.20 (4.16)	8.58(4.31)	4.93	**.028**
WOMAC Physical Function	22.54(14.42)	28.15(14.01)	7.22	**.008**
SPPB MEP	20.82(23.22)	27.50(29.03)	3.01	.085
SPPB Physical Function	9.76(1.74)	8.96(1.61)	10.77	**.001**

Note: **N**ADI=national area deprivation index; ISI=Insomnia Severity Index; WOMAC=Western Ontario and McMaster Universities Osteoarthritis Index; SPPB=Short Physical Performance Battery; MEP=movement-evoked pain

Table 4 Correlation coefficients at baseline

		1	2	3	4	5	6	7	8	9	10	11	12	13	14
1.	NADI	—													
2.	Sleep Efficiency	**-.21***	—												
3.	Insomnia Severity Index	.02	-.10	—											
4.	WOMAC Clinical Pain Severity	**.17***	**-.31****	**.41****	—										
5.	WOMAC Physical Function	**.23****	**-.31****	**.37****	**.87****	—									
6.	SPPB MEP	**.18***	-.11	**.37****	**.58****	**.62****	—								
7.	SPPB Physical Function	**-.24****	**.32****	-.15	**-.30****	**-.41****	**-.23****	—							
8.	Pain Catastrophizing	**.24****	**-.24****	**.35****	**.55****	**.57****	**.41****	**-.38****	—						
9.	PROMIS Depressive Symptoms	.14	-.12	**.45****	**.37****	**.38****	**.26****	**-.31****	**.52****	—					
10.	Race/ethnicity	**.31****	**-.19***	.02	**.31****	**.36****	**.32****	**-.18***	**.32****	.09	—				
11.	KL Index	-.03	-.09	.01	**.16***	**.26****	**.28****	**-.16***	.14	-.09	.08	—			
12.	BMI	.12	-.10	.11	**.22****	**.28****	.14	**-.29****	**.22****	.10	.10	**.35****	—		
13.	Age	**-.16***	.07	**-.33****	**-.31****	**-.26****	**-.18***	.00	**-.32****	**-.26****	**-.20****	**.15***	**-.25****	—	
14.	Sex	-.06	**.28****	.02	-.04	-.05	-.08	-.07	.01	.00	-.09	.03	**.25****	.12	—

Race Coded: 0 = Non-Hispanic White, 1 = Non-Hispanic Black; Sex Coded: 1 = Male, 2 = Female

NADI = National Area of Deprivation Index; SPPB = Short Physical Performance Battery; MEP = Movement-evoked Pain; WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index; PROMIS = Patient-reported Outcomes Measurement Information BMI = Body Mass Index; KL = Kellgren-Lawrence; *p < .05; **p < .01

Table 5: Predictors of clinical pain, movement-evoked pain, and physical function from controls, sleep efficiency, race/ethnicity, and sleep efficiency by racial/ethnic group. Hierarchical linear regression analyses.

	Model 1: WOMAC Pain Change	Model 2: WOMAC Physical Function Change	Model 3: SPPB MEP Pain Change	Model 4: SPPB Physical Function Change
Independent Variables				
Block 1: Control Variables				
Age	.081	.071	.160	-.127
BMI	.111	.159	-.017	.084
KL Index	-.073	-.097	-.073	.048
Depressive Symptoms	-.074	.107	-.059	.099
Site	.077	.053	.249*	-.105
R^2	-.030	-.012	.024	-.001
F	.387	.754	1.520	.975
Block 2: Sleep				
Sleep Efficiency	.259*	.206*	.114	-.133
R^2	.027	.020	.027	.007
ΔR^2	.063	.040	.012	.017
F	1.494	1.364	1.495	1.121
ΔF	6.919*	4.291*	1.345	1.815
Block 3: Race				
Race	.082	.083	.010	-.160
R^2	.024	.017	.082	.021
ΔR^2	.006	.006	.000	.023
F	1.369	1.261	1.271	1.335
ΔF	.648	.668	.009	2.514
Block 4: Interaction				
Sleep Efficiency x Race	-.195	-.163	.115	-.199
R^2	.032	.020	.014	.031
ΔR^2	.017	.012	.006	.018
F	1.436	1.264	1.188	1.433
ΔF	1.824	1.258	.644	2.024

Race Coded: 0 = Non-Hispanic White, 1 = Non-Hispanic Black

SPPB = Short Physical Performance Battery, WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index, MEP = Movement-evoked Pain, BMI = Body Mass Index, KL = Kellgren-Lawrence

*p < .05; **p < .01

Table 6: Predictors of clinical pain, movement-evoked pain, and physical function from controls, insomnia severity, race/ethnicity, and insomnia severity by racial/ethnic group. Hierarchical linear regression analyses.

	Model 5: WOMAC Clinical Pain Change	Model 6: WOMAC Physical Function Change	Model 7: SPPB MEP Pain Change	Model 8: SPPB Physical Function Change
Independent Variables				
Block 1: Control Variables				
Age	.038	.131	.160	-.048
BMI	.073	.164	-.017	.133
KL Index	-.067	-.122	-.073	.055
Depressive Symptoms	-.011	.054	-.059	.122
Site	.616	.112	.248*	-.119
R^2	-.014	-.004	.028	.011
F	.666	.899	1.714	1.269
Block 2: Sleep				
Insomnia Severity	-.150	-.164	-.134	-.047
R^2	-.006	.006	.033	.004
ΔR^2	.015	.018	.013	.002
F	.871	1.127	1.707	1.084
ΔF	1.869	2.224	1.628	.202
Block 3: Race				
Race	.033	.053	-.050	-.203*
R^2	-.014	.006	.028	.090
ΔR^2	.001	.003	.003	.038
F	.759	1.008	1.501	1.335
ΔF	.123	.332	.324	4.940*
Block 4: Interaction				
Insomnia Severity x Race	.092	-.033	-.082	-.337*
R^2	-.018	-.008	.023	.143
ΔR^2	.004	.000	.003	.053
F	.722	.883	1.335	2.445*
ΔF	.485	.061	.390	7.280*

Race Coded: 0 = Non-Hispanic White, 1 = Non-Hispanic Black

SPPB = Short Physical Performance Battery, WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index, MEP = Movement-evoked Pain, BMI = Body Mass Index, KL = Kellgren-Lawrence

*p < .05; **p < .01

Table 7: Predictors of clinical pain, movement-evoked pain, and physical function from controls, sleep efficiency, NADI group, and actigraphic sleep efficiency by NADI group. Hierarchical linear regression analyses.

	Model 1: WOMAC Pain Change	Model 2: WOMAC Physical Function Change	Model 3: SPPB MEP Pain Change	Model 4: SPPB Physical Function Change
Independent Variables				
Block 1: Control Variables				
Age	.081	.071	.160	-.127
BMI	.111	.159	-.017	.084
KL Index	-.073	-.097	-.073	.048
Depressive Symptoms	-.074	.107	-.059	.099
Site	.077	.053	.249*	-.105
R^2	-.030	-.012	.024	-.001
F	.387	.754	1.520	.975
Block 2: Sleep				
Sleep Efficiency	.259*	.206*	.114	-.133
R^2	.027	.020	.027	.007
ΔR^2	.063	.040	.012	.017
F	1.494	1.364	1.495	1.121
ΔF	6.919*	4.291*	1.345	1.815
Block 3: NADI				
NADI	.108	.174	.129	-.096
R^2	.026	.104	.098	.070
ΔR^2	.045	.028	.015	.008
F	1.711	1.68	1.529	1.088
ΔF	2.844	3.038	1.670	.897
Block 4: Interaction				
Sleep Efficiency x NADI	.120	-.034	-.009	.166
R^2	.115	.105	.098	.083
ΔR^2	.026	.028	.000	.013
F	1.589	1.418	1.325	1.130
ΔF	.762	.061	.004	1.394

NADI = National Area of Deprivation Index: Coded: 0 = Low NADI, 1 = High NADI

SPPB = Short Physical Performance Battery, WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index, MEP = Movement-evoked Pain, BMI = Body Mass Index, KL = Kellgren-Lawrence

*p < .05; **p < .01

Table 8: Predictors of clinical pain, movement-evoked pain, and physical function from controls, insomnia severity, NADI group, and insomnia severity by NADI group. Hierarchical linear regression analyses.

	Model 5: WOMAC Pain Change	Model 6: WOMAC Physical Function Change	Model 7: SPPB MEP Pain Change	Model 8: SPPB Physical Function Change
Independent Variables				
Block 1: Control Variables				
Age	.038	.131	.160	-.048
BMI	.073	.164	-.017	.133
KL Index	-.067	-.122	-.073	.055
Depressive Symptoms	-.011	.054	-.059	.122
Site	.616	.112	.248*	-.119
R^2	-.014	-.004	.028	.011
F	.666	.899	1.714	1.269
Block 2: Sleep				
Insomnia Severity	-.150	-.164	-.134	-.047
R^2	-.006	.006	.033	.004
ΔR^2	.015	.018	.013	.002
F	.871	1.127	1.707	1.084
ΔF	1.869	2.224	1.628	.202
Block 3: NADI				
NADI	.119	.138	.065	-.120
R^2	.055	.072	.084	.009
ΔR^2	.013	.018	.484	.013
F	.982	1.296	1.402	1.169
ΔF	1.628	2.235	.484	1.647
Block 4: Interaction				
Insomnia Severity x NADI	-.137	-.210	-.107	-.324*
R^2	.064	.092	.089	.107
ΔR^2	.013	.006	.005	.042
F	.993	1.476	1.402	1.752
ΔF	1.057	2.616	.573	5.518*

NADI = National Area of Deprivation Index: Coded: 0 = Low NADI, 1 = High NADI
SPPB = Short Physical Performance Battery, WOMAC = Western Ontario and McMaster Universities Osteoarthritis Index, MEP = Movement-evoked Pain, BMI = Body Mass Index, KL = Kellgren-Lawrence
*p < .05; **p < .01

Figure 1

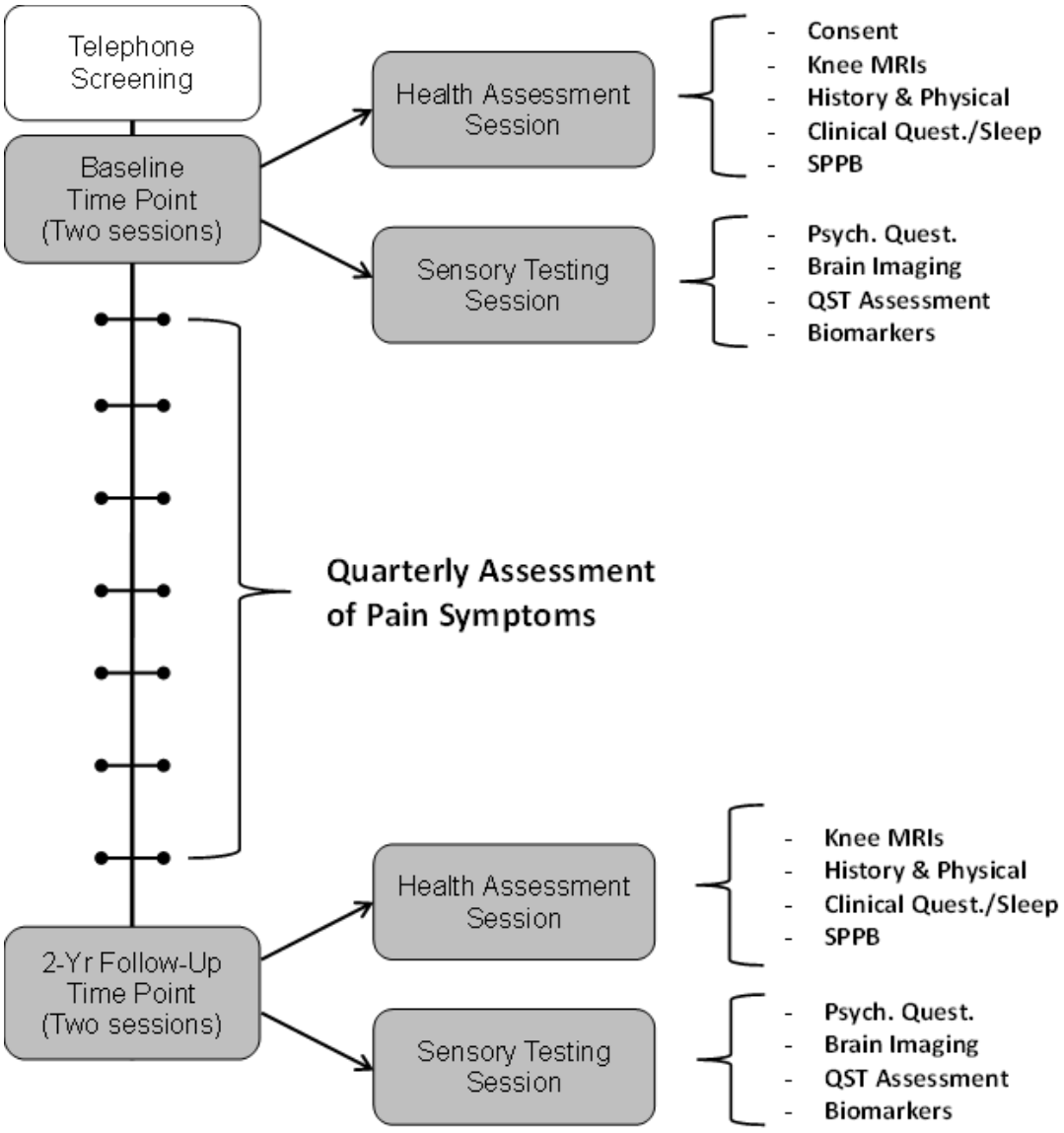

Telephone Screening
Baseline Time Point (Two sessions)
Health Assessment Session
- Consent
- Knee MRIs
- History & Physical
- Clinical Quest./Sleep
- SPPB
Sensory Testing Session
- Psych. Quest.
- Brain Imaging
- QST Assessment
- Biomarkers
Quarterly Assessment of Pain Symptoms
2-Yr Follow-Up Time Point (Two sessions)
Health Assessment Session
- Knee MRIs
- History & Physical
- Clinical Quest./Sleep
- SPPB
Sensory Testing Session
- Psych. Quest.
- Brain Imaging
- QST Assessment
- Biomarkers

Figure 2

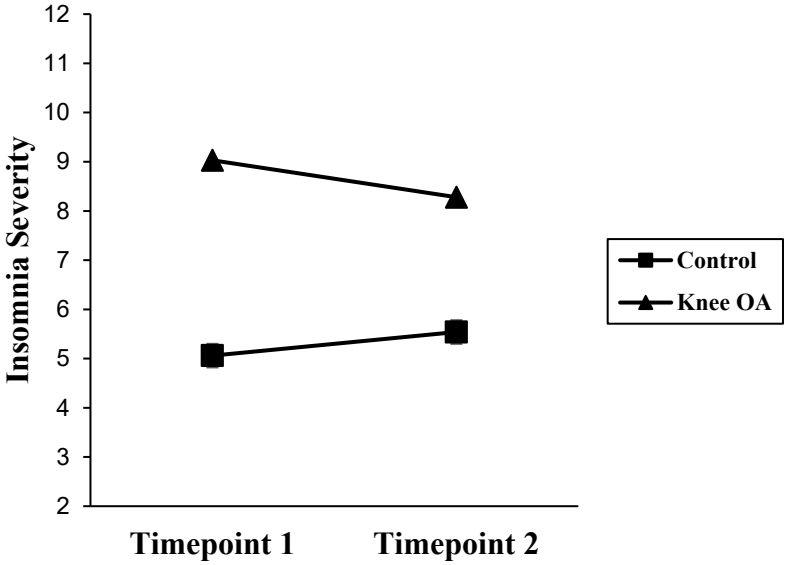


Figure 3

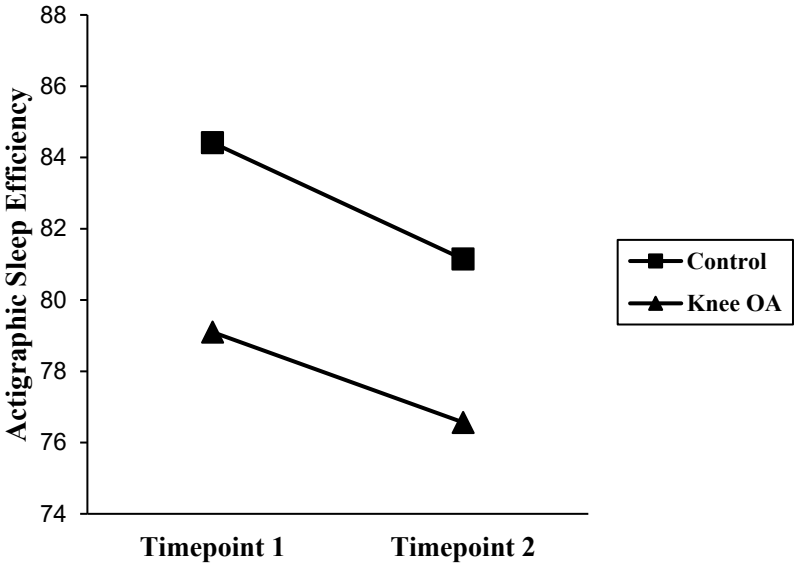


Figure 4

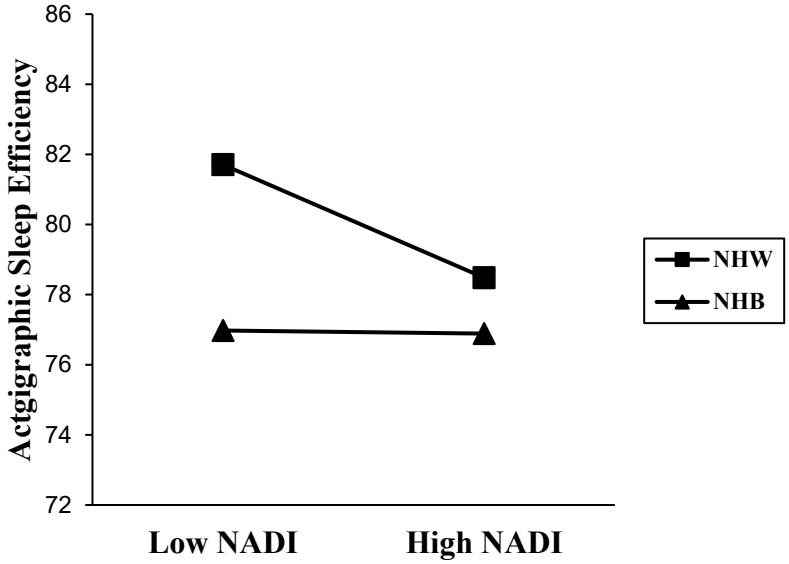


Figure 5

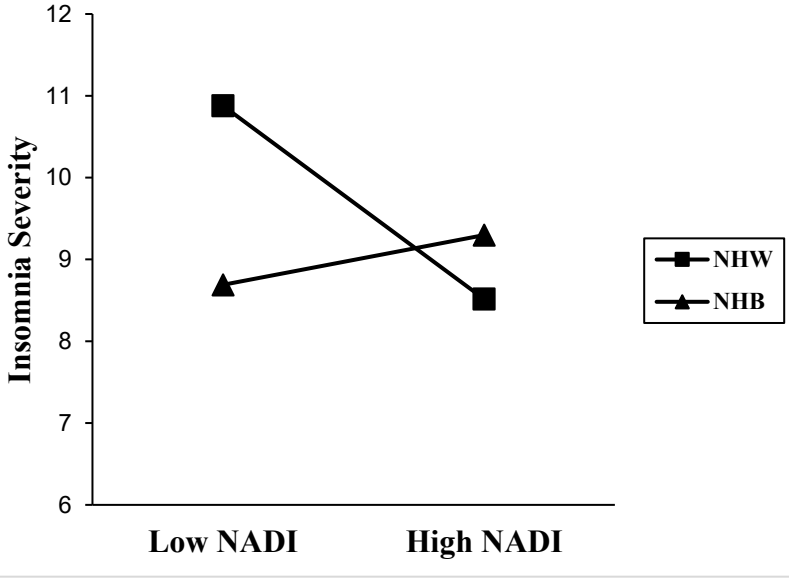


Figure 6

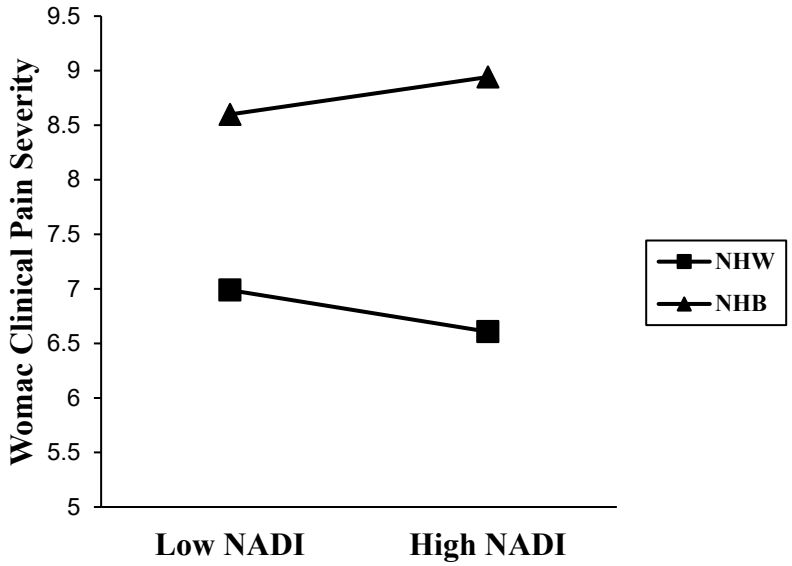


Figure 7

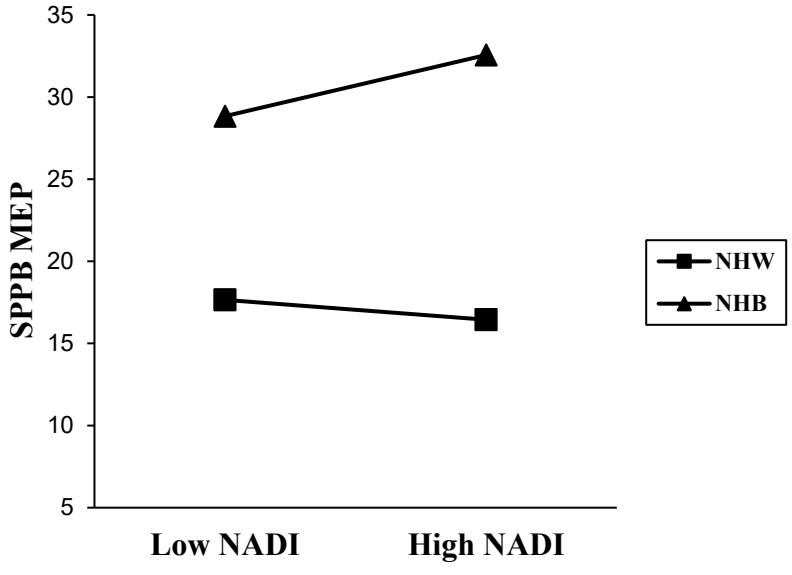


Figure 8

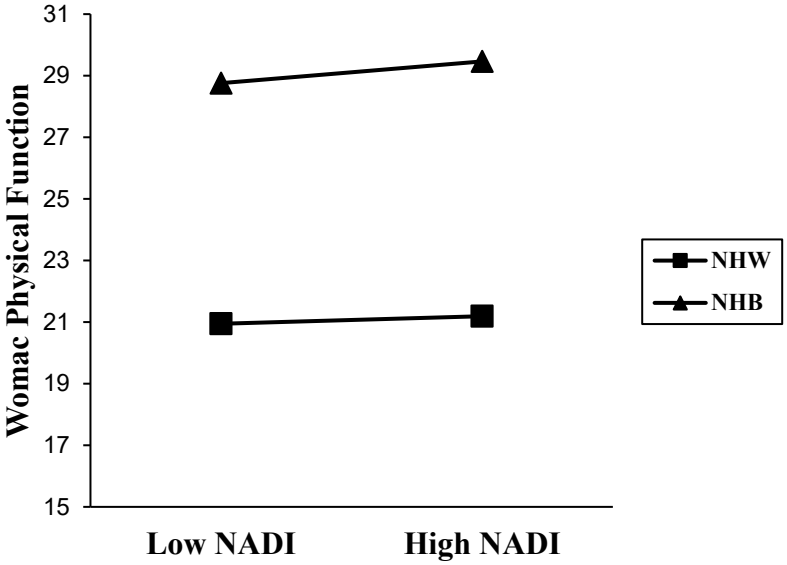


Figure 9

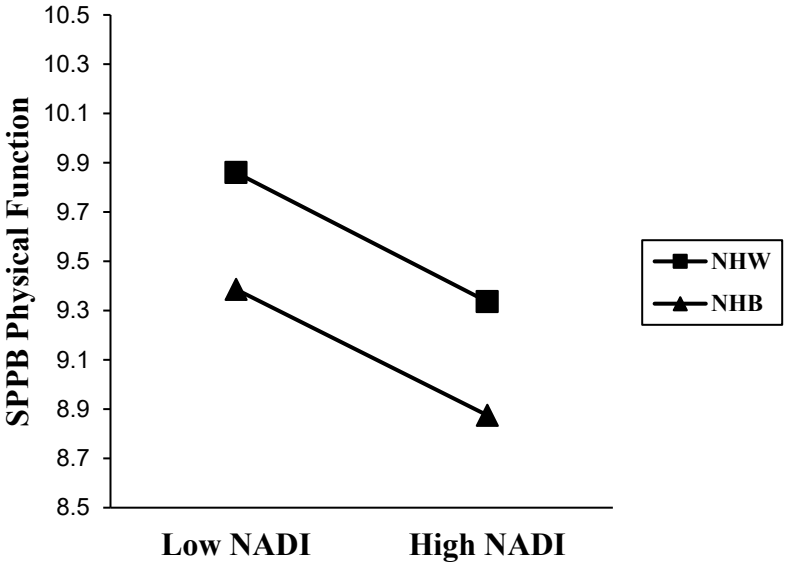
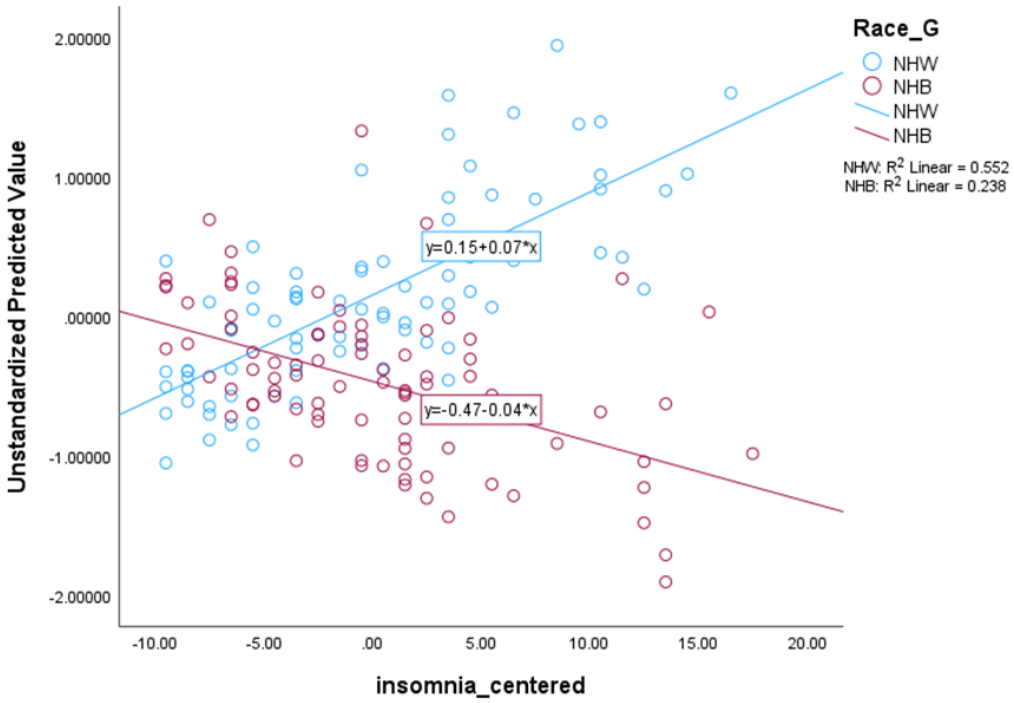


Figure 10


Race_G
NHW
NHB
NHW
NHB
NHW: R2 Linear = 0.552
NHB: R2 Linear = 0.238
y=0.15+0.07*x
y=-0.47-0.04*x
Unstandardized Predicted Value
2.00000
1.00000
.00000
-1.00000
-2.00000
-10.00
-5.00
.00
5.00
10.00
15.00
20.00
insomnia_centered

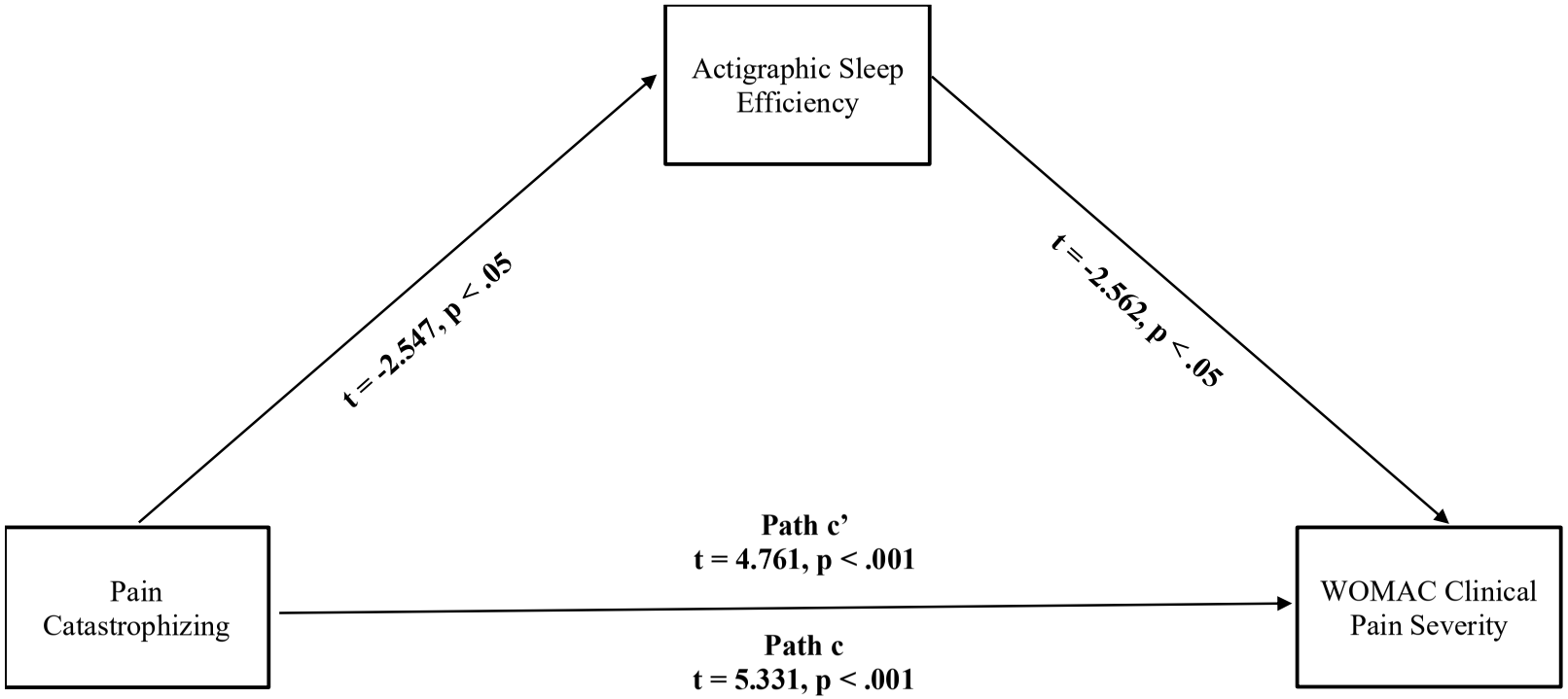


Figure 11 Actigraphic sleep efficiency significantly partially mediates the association between pain catastrophizing and WOMAC clinical pain severity.

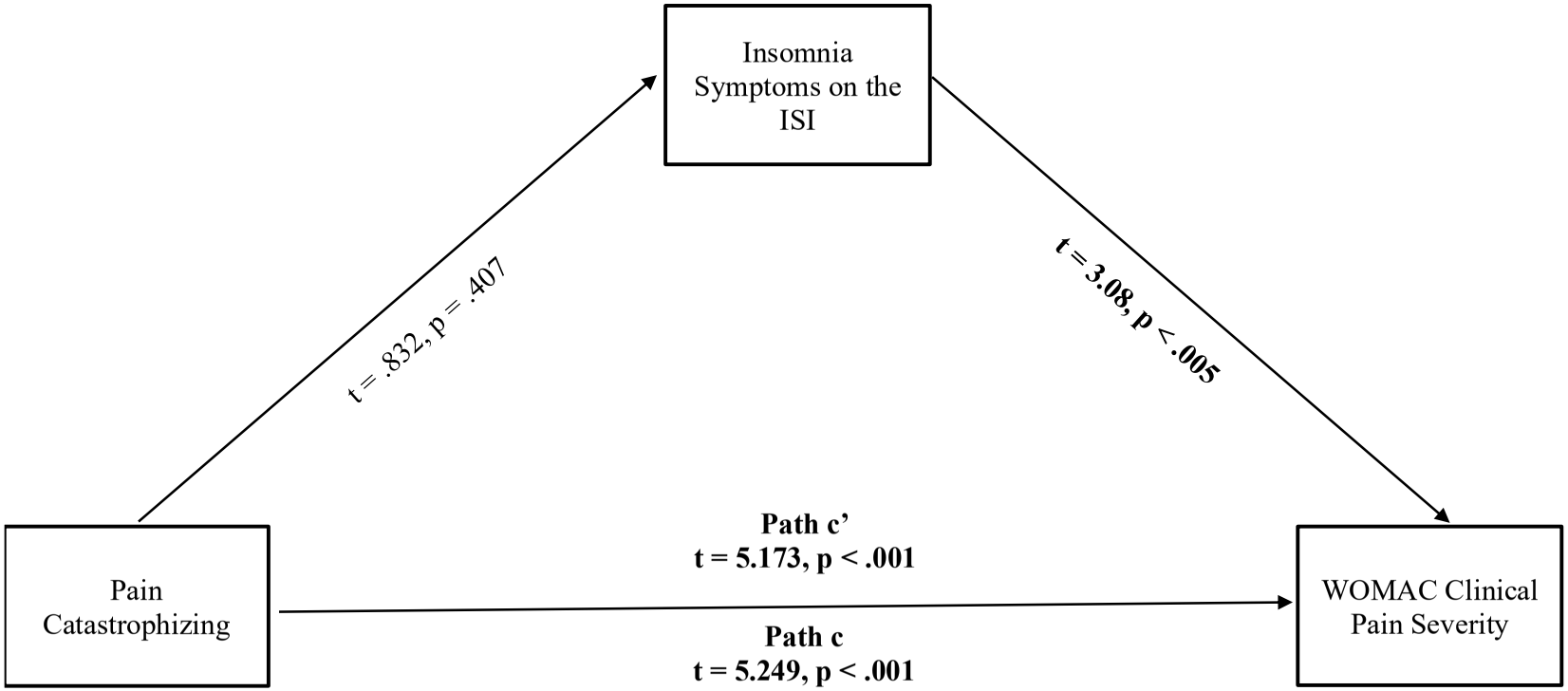


Figure 12. Insomnia symptoms on the ISI does not significantly mediate the association between pain catastrophizing and WOMAC clinical pain severity.

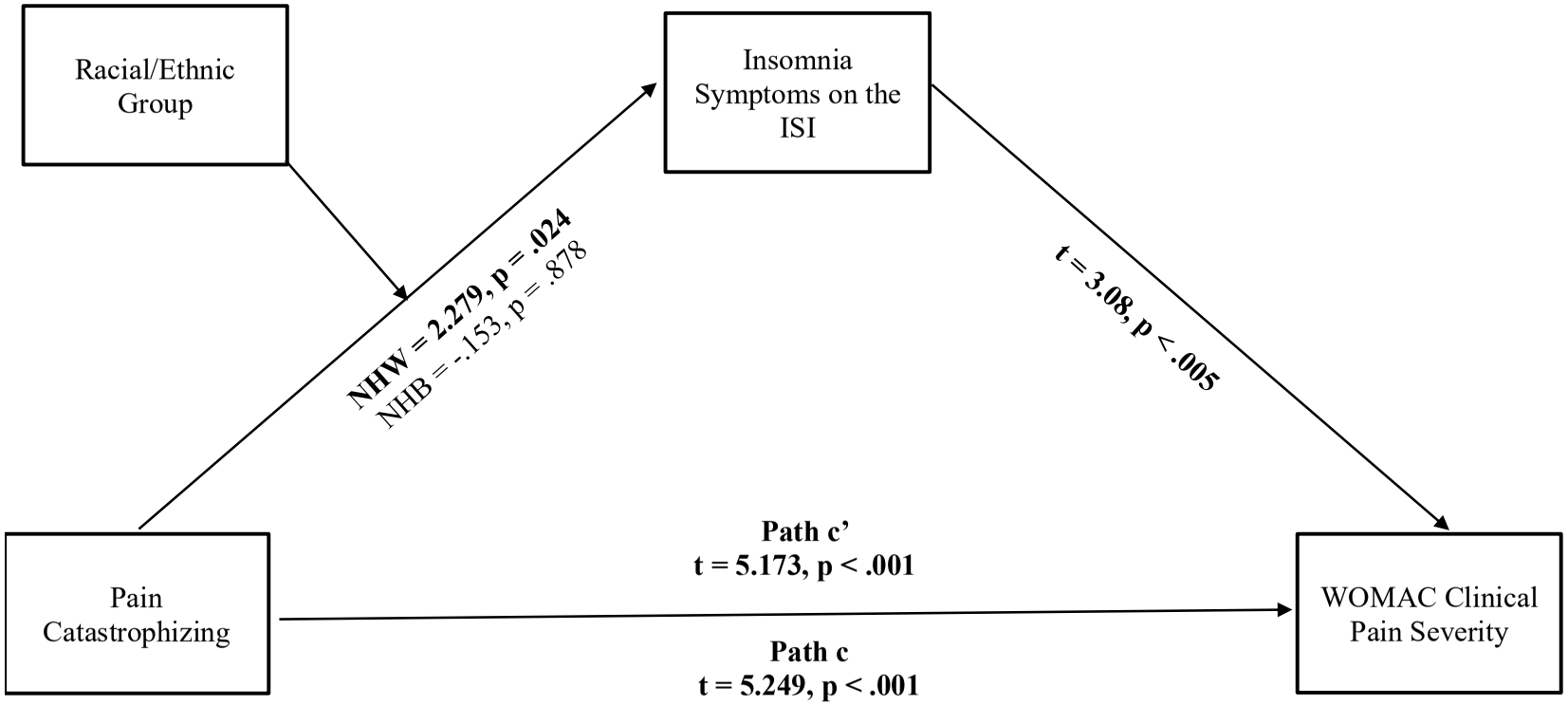


Figure 13. A mediated moderation of racial/ethnic group on pain catastrophizing on WOMAC clinical pain severity via insomnia symptoms on the ISI.