

COGNITIVE MECHANISMS IN COMORBID INSOMNIA  
AND CHRONIC PAIN

by

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A THESIS

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## ABSTRACT

The present study examined the relation between pain catastrophizing, pre-sleep arousal (PSA), and insomnia severity in chronic pain patients. Forty-eight outpatients with chronic pain (duration  $\geq$  6 months) completed self-report measures of health, mood, pain, and sleep. A hierarchical regression analysis was conducted to determine the relative contributions of pain catastrophizing, cognitive PSA, and somatic PSA to the prediction of scores on the Insomnia Severity Index, while controlling for age, sex, education level, depression severity, symptoms of Restless Legs Syndrome (RLS), and pain intensity. Results showed that pain catastrophizing accounted for unique variance in insomnia severity, independent of pain intensity and other control variables. However, when cognitive and somatic PSA were taken into account, the significance of cognitive PSA rendered pain catastrophizing non-significant. Research and clinical work should explore how cognitive variables (such as pain catastrophizing) may be linked to the pre-sleep arousal in comorbid insomnia.

## DEDICATION

This thesis is dedicated to everyone who stood by me throughout the time taken to complete this project. In particular, I am thankful for my family and close friends, who never stopped encouraging me.

## LIST OF ABBREVIATIONS AND SYMBOLS

$a$	Cronbach's index of internal consistency
$\beta$	Beta: a standardized regression unit
$F$	Fisher's $F$ ratio: A ratio of two variances
$M$	Mean: the sum of a set of measurements divided by the number of measurements in the set
$SD$	Standard deviation
$p$	Probability associated with the occurrence under the null hypothesis of a value as extreme as or more extreme than the observed value
$r$	Pearson product-moment correlation
$t$	Computed value of $t$ test
$R^2$	Coefficient of determination: overall proportion of variance in the criterion that is jointly explained by the independent variables
$sr^2$	Squared semipartial correlation coefficient: proportion of total variance in the criterion that is uniquely explained by an independent variable
$<$	Less than
$\leq$	Less than or equal to
$=$	Equal to

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## INTRODUCTION

Insomnia is the most common sleep complaint in the general population, with an estimated prevalence of 9-15% and well documented chronicity (Ford & Kamerow, 1989; Ohayon, 2002). Symptoms of insomnia include difficulty falling or staying asleep, early morning awakenings, or chronic nonrestorative sleep, and are often accompanied by daytime impairment and distress. Persistent insomnia is associated with more health problems (Daley et al., 2009), increased risk of depression (Ohayon, 2002), and reduced quality of life (Zammit, Weiner, Damato, Sillup, & McMillan, 1999). A 1994 estimate of the annual cost of insomnia due to direct (e.g., increased healthcare utilization) and indirect (e.g., missed work) costs was between \$92.5 and \$107.5 billion (Stoller, 1994).

Insomnia related to another mental or physical disorder is the most frequent diagnosis of insomnia (Buysse et al., 1994; Ohayon, 1997). Estimates of insomnia symptoms comorbid with chronic pain range from 53-88% (Smith, Perlis, Smith, Giles, & Carmody, 2000; Tang, Wright, & Salkovskis, 2007; Wilson, Eriksson, D'Eon, Mikail, & Emery, 2002). Individuals reporting chronic pain are significantly more likely to have insomnia than those without chronic pain, even when controlling for symptoms of depression, anxiety, and other medical or sleep disorders (Taylor et al., 2007). Individuals with these comorbid conditions also report longer durations of insomnia and more daytime consequences of insomnia when compared to individuals with primary insomnia (Ohayon, 2005). In addition, there is growing evidence that the relationship between pain and sleep disturbance is reciprocal, such that pain can disturb sleep quantity/quality and sleep disruption can exacerbate pain (Smith & Haythornthwaite, 2004). Even more

concerning is the increased costs, disability, and reduced quality of life that accompany co-occurring pain and sleep problems. With more than one-quarter of adult Americans (an estimated 76.5 million) reporting pain that persists longer than 24 hours, there is high potential for insomnia comorbid with chronic pain.

Pain is defined by the International Association for the Study of Pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (IASP Task Force on Taxonomy, 1994). The current understanding of pain as a complex and dynamic process is largely the product of Melzack and Wall’s revolutionary Gate Control Theory of pain (Melzack & Wall, 1965). The Gate Control Theory states that the transmission of pain signals is controlled by a gating mechanism in the spinal cord that widens or narrows (thereby increasing or decreasing pain) in response to signals from the brain. Notably, this theory was the first to suggest a role for psychological variables in modulating the perception of pain.

Since the introduction of the Gate Control Theory of pain, research has identified a number of behavioral, affective, and cognitive variables that impact the pain experience. In particular, catastrophic thinking about one’s pain has emerged as an important predictor of poor adjustment to pain. Pain catastrophizing refers to a wide variety of negative, exaggerated thoughts about actual or anticipated painful experiences. Proposed psychological mechanisms through which catastrophizing influences the pain experience include increased attention and negative appraisals of pain, which can lead to hypervigilance and information-processing biases, maladaptive coping responses, and heightened emotional distress (Sullivan et al., 2001).

Catastrophizing has been consistently linked with higher levels of pain intensity (e.g., Sullivan, Stanish, Waite, Sullivan, & Tripp, 1998; Sullivan, Thorn, Rodgers, & Ward, 2004; Tan,

Jensen, Robinson-Whelen, Thornby, & Monga, 2001; Thorn et al., 2004), more post-surgical pain (Granot & Ferber, 2005), and increases in pain over time (Burton, Tillotson, Main, & Hollis, 1995; Keefe, Brown, Wallston, & Caldwell, 1989). This relation between catastrophizing and heightened pain has been observed across diverse pain populations, as well as in healthy individuals undergoing painful medical/dental procedures or experimental pain tasks (see Sullivan et al., 2001 for review). Pain catastrophizing has also been linked to other pain-related outcomes, such as increased pain and illness behaviors, heightened disability, and emotional distress (Severeijns, Vlaeyen, van den Hout, & Weber, 2001; Sullivan et al., 1998; Tan et al., 2001; Turner, Jensen, Warm, & Cardenas, 2002), and has been targeted in cognitive-behavioral treatment for chronic pain (Thorn et al., 2007).

Pain catastrophizing has rarely been investigated as it relates to non-pain-specific outcomes. Devoulyte et al. (2003) investigated whether the effects of pain catastrophizing could generalize beyond pain-specific domains in a sample of adults with respiratory illnesses and found that pain catastrophizing scores predicted the severity of non-pain physical symptoms. Another recent study developed a self-report instrument to assess behaviors and cognitions related to pain and poor sleep, and suggested that catastrophizing is a shared psychological process in both problems (MacDonald, Linton, & Jansson-Frojmark, 2008).

A well-established model of insomnia posits that chronic insomnia results from a combination of predisposing, precipitating, and perpetuating factors (Spielman, Caruso, & Glovinsky, 1987). In the case of insomnia comorbid with chronic pain, the initial injury or pain sensations may trigger transient sleep problems in individuals predisposed to insomnia. Support for this idea comes from experimental research showing that pain induction during sleep can lead to increased arousal and wakefulness at night (Drewes, Nielsen, Arendt-Nielsen, Birket-Smith, &

Hansen, 1997; Lavigne et al., 2004). Correlational studies have also shown that a painful day is followed by poorer subjective sleep that night (Affleck, Urrows, Tennen, Higgins, & Abeles, 1996) and pain intensity can predict sleep problems two years later (Nicassio & Wallston, 1992; Smith et al., 2008). However, not all individuals with chronic pain develop persistent insomnia, suggesting that factors other than pain intensity are involved.

Hyperarousal has been conceptualized as both a predisposing and perpetuating factor of primary insomnia (e.g., Coren, 1988; Spielman et al., 1987). As a perpetuating factor, elevated arousal at bedtime is the result of learned associations between the bed and bedroom with states of wakefulness (i.e., eating, watching television, worrying, etc). Over time, repeated pairings of the bed with sleep incompatible behaviors and processes lead to a conditioned response of hyperarousal at bedtime, which serves to perpetuate the sleep disturbance. Only one study to date directly investigated conditioned arousal in insomnia (Robertson, Broomfield, & Espie, 2007), but behavioral techniques aimed at breaking these learned associations and reinstating the bed as a cue for sleep have been shown to be effective treatments for insomnia (Morin et al., 2006).

There has been substantial debate surrounding the process by which hyperarousal interferes with sleep, specifically, whether it occurs primarily through physiological and/or cognitive activation. Several studies have found that people with insomnia exhibit heightened physiological arousal just prior to and during sleep (Bonnet & Arand, 1995, 1997; Mitchell, 1979), but these findings have not always been replicated (Browman & Tepas, 1976). In contrast, research has more consistently linked cognitive arousal with sleep disturbance. When asked for the primary reason for their sleeping difficulties, people with insomnia were more likely to identify cognitive factors (i.e., overactive mind), relative to somatic ones (i.e., aware of one's body as full of tension, sweaty, etc) (Espie, Brooks, & Lindsay, 1989; Lichstein & Rosenthal,

1980). Similarly, on a self-report measure of arousal symptoms experienced at bedtime, scores on the cognitive arousal subscale were more strongly associated with self-reported sleep parameters than somatic arousal scores, particularly on overall and nightly ratings of sleep-onset latency (Nicassio, Mendlowitz, Fussell, & Petras, 1985).

One possible explanation for this heightened cognitive arousal is that cognitive activity is simply an epiphenomenon of the nighttime wakefulness inherent in insomnia. Two studies found that inducing cognitive arousal in normal sleepers before a daytime nap led to an increase in subjective sleep-onset latency (SOL) and a decrease in self-reported duration of sleep (Gross & Borkovec, 1982; Tang & Harvey, 2004). Inducing cognitive arousal at bedtime also interfered with laboratory measures of sleep (increased SOL and more nighttime awakenings) in an undergraduate sample (Hall, Buysse, Reynolds, Kupfer, & Baum, 1996). In contrast, manipulating pre-sleep cognitive stress in individuals with insomnia led to a decrease in subjective and objective SOL (Haynes, Adams, & Franzen, 1981). Haynes and colleagues suggested that their experimental stressor distracted individuals with insomnia from their usual sleep-interfering cognitions, thus allowing them to fall asleep faster.

Attempts to delineate the specific content of pre-sleep cognitions have established that thoughts and concern about sleep are extremely prevalent in people with insomnia (Harvey, 2000; Kuisk, Bertelson, & Walsh, 1989; Watts, Coyle, & East, 1994; Wicklow & Espie, 2000). However, studies found that general problem-solving and pre-sleep thoughts about a range of topics (e.g., work and family, physical sensations, environmental stimuli) are also common in people with insomnia (Harvey, 2000; Van Egeren, Haynes, Franzen, & Hamilton, 1983). Additionally, there is evidence that affect-laden cognitive activity is more arousing and sleep-interfering than neutral cognitive activity. Kuisk and colleagues (1989) documented that the

cognitive activity of people with insomnia was more negatively valenced than pre-sleep thoughts of normal sleepers. In an experimental study, Tang and Harvey (2004) induced both anxious and neutral cognitive arousal before a nap. Results showed that only the anxious cognitive arousal group reported longer subjective sleep-onset latency than the control group; the neutral cognitive arousal group did not differ from controls on subjectively reported sleep.

Due to the important role of arousal in the maintenance of primary insomnia, a few researchers have investigated its role in insomnia comorbid with pain. Research has shown that chronic pain patients generally report higher levels of pre-sleep arousal (cognitive and somatic) than normal sleepers (Palermo, Toliver-Sokol, Fonareva, & Koh, 2007; Smith et al., 2000). One study found that, similar to primary insomnia, pre-sleep cognitive arousal was more strongly related to sleep quality in chronic musculoskeletal pain patients, relative to somatic arousal (Smith et al., 2000). Cognitive arousal uniquely predicted sleep quality in this sample, over and beyond levels of depression and pain intensity. Pre-sleep worry also predicted ratings of sleep quality in a sample of adolescents with mixed chronic pain conditions (Palermo et al., 2007).

A follow-up study by Smith and colleagues (2001) used in-vivo thought sampling procedures to investigate the content of pre-sleep cognitions in pain patients. The most frequent topics included general thoughts about pain, negative thoughts about sleep, and thoughts about the experimental procedure, but only pain-related thoughts predicted longer sleep-onset latencies. Given that affect-laden cognitive activity appears to impact sleep more than neutral cognitive activity (Tang & Harvey, 2004), it follows that pain patients' thoughts about their pain would be more salient and arousing for them, thus having more potential to disrupt their sleep.

There is a need for a closer examination of the psychological and cognitive processes involved in the complex relationship between insomnia and chronic pain. Limitations of previous

research include a lack of standardized and validated sleep outcome measures. Studies often ask only about pain-related sleep disturbance or use a single survey item to assess overall sleep problems without identifying the specific symptoms experienced. In addition, potential confounding variables that can influence pain and sleep (e.g., other sleep disorders, comorbid medical and psychological conditions, medication usage) have rarely been measured and controlled for.

The current study examined the strength and predictive value of the relationships among pain intensity, pain catastrophizing, and pre-sleep arousal with insomnia severity in individuals with chronic pain. Previous research has shown that pain catastrophizing is predictive of negative pain-related outcomes. However, this pain-specific cognitive process has not yet been explored in relation to sleep disturbance. Therefore, our first aim was to establish whether pain catastrophizing is associated with severity of insomnia symptoms in chronic pain patients. It was expected that higher levels of pain catastrophizing would predict more severe insomnia, independent of pain intensity ratings. Next, we extended Smith and colleague's (2000) findings predicting sleep quality in pain patients to the prediction of insomnia symptom severity. Specifically, we expected cognitive arousal, as opposed to somatic arousal, to better predict insomnia severity. Since pain catastrophizing and cognitive arousal both involve intrusive and negative thoughts about affect-laden topics, a high degree of overlap was expected between the two constructs. However, despite this overlap, both pain catastrophizing and cognitive pre-sleep arousal were expected to account for unique variance in insomnia severity. The study used a cross-sectional, correlational research design. A hierarchical regression analysis was conducted to determine the relative contribution of each predictor above and beyond pain intensity and other control variables.

## METHODOLOGY

### **Participants**

Adult outpatients were recruited from five pain and medical clinics in Tuscaloosa and Birmingham, Alabama. All participants were receiving outpatient treatment for chronic pain (pain duration of at least 6 months). Both normal and poor sleepers were included to capture the full range of insomnia symptoms in pain patients. Participants reporting fibromyalgia as their primary source of pain were excluded due to the symptom overlap with chronic fatigue and sleep disorders. Participants reporting diagnoses or severe symptoms of sleep apnea or narcolepsy were excluded due to fundamental differences in symptom presentation and underlying mechanisms. Participants reporting other sleep disorders (e.g., restless leg syndrome) were not excluded, but these symptoms were assessed. Participants reporting headaches as their primary pain problem were initially excluded, but due to slow accrual of participants, this exclusion criterion was discontinued mid-way through recruitment.

During the 1.5 year recruitment period (August 2009 through December 2010), 158 patients were informed of the study. Ninety-one people met initial eligibility criteria and agreed to participate. Ninety-one questionnaire packets were sent and 52 were returned (response rate 57%). Participants received \$10 monetary compensation when they returned the questionnaires. Four participants were later found to be ineligible due to probable sleep apnea and their data were excluded. The final sample included 48 participants (75% female) between the ages of 23 and 70 ( $M = 51.6$  years,  $SD = 11.9$ ). The majority of participants were Caucasian (81.3%), followed by African American (8.3%) and multiracial classifications (10.4%). Ninety-two



percent of the sample reported pain in more than one site. The most commonly reported types of pain were back and neck pain (96%), arthritis pain (71%), and knee/hip/limb pain (71%). Over half of the sample (58%) reported back and neck pain as their primary pain complaint (see Table 1). Mean duration of the primary pain complaint was 11.9 years ( $SD = 8.8$ ), ranging from 9 months to 33 years. Over one third of the sample (35%) reported being on disability. The remainder of the sample was currently working or in school (27.1%), retired (20.8%), unemployed due to pain (8.3%), or unemployed for other reasons (8.3%). All 48 participants reported a high school degree or GED and approximately 46% held a college degree. Forty-four percent of the sample reported their current income was inadequate to meet basic needs for food, clothing, and medical care.

Table 1

*Primary Pain Complaints (N=48)*

Pain Type	<i>n</i>	%
Back/ neck pain	28	58.3
Knee/ hip/ limb pain	7	14.6
Neuropathy/ RSD	5	10.4
Arthritis pain	3	6.3
Headache pain	3	6.3
IBS/ Pelvic pain	2	4.2

*Note.* RSD = Reflexive Sympathetic Dystrophy;

IBS= Irritable Bowel Syndrome

## Measures

**Demographics and health variables.** Participants completed a background information form and brief health history to measure sample characteristics and control variables. Basic information was collected on socio-demographic variables (i.e., age, sex, race/ethnicity, highest level of education, employment/disability status, and perceived income adequacy). The health history gathered information about comorbidity (i.e., other physical health problems, psychological disorder diagnoses), and pain and sleep problems (i.e., specific symptoms, duration of problems, symptoms of other sleep disorders). Body Mass Index (BMI) was calculated from self-reported height and weight. Additional items assessed caffeine consumption, tobacco use, and alcohol use over the previous two week period. Participants also provided a list of prescription and over-the-counter medications currently being used, the purpose of each medication, and typical dosage. Prescription medication usage was categorized into the following categories: (1) Opioid analgesics, (2) Anti-inflammatories, (3) Muscle relaxants, (4) Anti-convulsants, (5) Antidepressants, (6) Benzodiazepines, (7) Hypnotics (including benzodiazepines taken for sleep).

**Depressive symptoms.** The Beck Depression Inventory-II (BDI-II; Beck, Brown, & Steer, 1996) is a 21-item self-report measure of cognitive and behavioral symptoms of depression experienced over the past two weeks. Participants rate each item from 0-3 with a higher summed score representing higher symptomatology. Total scores range from 0 - 63 where scores of 0 - 13 are considered normal, scores of 14 - 19 represent mild to moderate depression, scores of 20 - 28 represent moderate to severe depression, and scores of 29 - 63 represent severe depression. Beck and colleagues (1996) reported a Cronbach's alpha of .92 for an outpatient sample, which exceeds coefficient alphas for preceding versions of the BDI. The scale had high

internal consistency in our sample (Cronbach's alpha = .91). Due to high rates of depression comorbid with both insomnia and pain, the BDI-II was used in this study to control for the possible confound of mood disturbance.

**Pain-related measures.** The Brief Pain Inventory (BPI; Cleeland, 1989) is a self-report instrument that measures pain intensity and pain interference with life activities. The four pain intensity items on the BPI ask about current pain intensity, worst, least, and average pain over the previous two weeks. Participants rate each of these items on a numerical rating scale (0 = *No pain*, 10 = *Pain as bad as you can imagine*), and the four ratings are averaged to yield a composite index of pain intensity. The BPI pain intensity scale has been validated with chronic pain samples (Tan, Jensen, Thornby, & Shanti, 2004) and had acceptable internal consistency in our sample (Cronbach's alpha = .85). An additional numerical rating scale was used to rate usual pain at bedtime during the past two weeks. The bedtime pain item has unknown validity data at this time.

The Pain Catastrophizing (CAT) Scale (PCS; Sullivan, Bishop, & Pivik, 1995) is a 13-item self-report instrument that measures the frequency and extent to which one engages in negative pain-related cognitions. (See Appendix for scale items). Participants respond to each item on a 5-point Likert scale (0 = *not at all*, 4 = *all the time*) according to the degree to which they experience each statement when in pain. The PCS yields a total summed score ranging from 0 to 52, with higher scores indicating greater levels of catastrophizing. Research suggests a PCS score of 30 or higher represents a clinically relevant level of catastrophizing (Sullivan, 2004). The PCS is composed of three well-correlated subscales. The 4-item rumination scale reflects an excessive focus on pain (i.e., "I keep thinking about how much it hurts"), the 3-item magnification scale reflects an exaggeration of the seriousness and consequences of pain (i.e., I

wonder whether something serious might happen”), and the 6-item helplessness scale reflects helplessness about one’s ability to deal with pain (i.e., “I feel I can’t stand it anymore”). This three-factor structure of the PCS has been replicated (Osman et al., 1997) and the reliability and validity of the scale has been established in clinical and experimental samples (Sullivan et al., 2001). The PCS total score was used in this study and had high internal consistency (Cronbach’s  $\alpha=.90$ ).

**Pre-sleep arousal.** The Pre-Sleep Arousal (PSA) Scale (Nicassio et al., 1985) is a 16-item self-report questionnaire designed to measure symptoms of cognitive and somatic arousal during the pre-sleep period. (See Appendix for scale items). Participants rate each item on a 5-point Likert scale (1 = *not at all*, 5 = *extremely*) based on how intensely they experience the symptom as they attempt to fall asleep in bed every night. The scale is organized into an 8-item subscale for cognitive pre-sleep arousal (PSA) and an 8-item subscale for somatic PSA. Example items from the cognitive subscale include “review or ponder events of the day” and “worry about falling asleep.” Items from the somatic subscale include “heart racing, pounding, or beating irregularly” and “a tight, tense feeling in your muscles.” Each subscale has a total summed score ranging from 8-40, with higher scores indicating higher levels of arousal. The PSA Scale total score ranges from 16- 80. Both subscales (Cognitive PSA and Somatic PSA) were used in this study with a time frame of the previous two weeks. Adequate internal consistency has been reported for the PSA subscales (Cronbach’s  $\alpha = .81$  and  $.76$  for the cognitive and somatic scales respectively). In our sample, internal consistency was higher for the PSA total (Cronbach’s  $\alpha = .90$ ) and cognitive PSA subscale ( $\alpha = .93$ ), but somewhat lower for the somatic PSA subscale (Cronbach’s  $\alpha = .69$ ).

**Sleep-related measures.** The Insomnia Severity Index (ISI; Morin, 1993) is a 7-item self-report measure used to evaluate the nature, severity, and impact of sleeping difficulties over the previous two weeks. Participants rate their perception of each item on a 5-point Likert scale (0 = *not at all*, 4 = *very much*), and the summed items yield a total severity score ranging from 0-28. The total score can also be classified into categories of severity: score of 0-7 (absence of insomnia), 8-14 (subthreshold insomnia), 15-21 (moderate clinical insomnia), and 22-28 (severe clinical insomnia). The validity of the ISI is considered high, and it has been used as both a screening and outcome measure in insomnia treatment research (Bastien, Vallieres, & Morin, 2001). The ISI was the primary outcome variable for this study and had adequate internal consistency in our sample (Cronbach's alpha = .81).

Sleep diaries were included as an additional measure of self-reported sleep. Participants complete the sleep diary each morning upon awakening for two weeks. The diary takes approximately 2 minutes to complete each day and provides a record of the time the participant entered bed the night before, final morning exit from bed, sleep-onset latency, number of awakenings during the night, wake time after sleep onset, and time spent napping the day before. The diary also asks for medications taken at bedtime and a rating of perceived quality of sleep ranging from 1 (*very poor*) to 5 (*excellent*). Sleep diaries provide a more valid and detailed measure of sleep by collecting multiple data points over time, and this method has been used previously with chronic pain patients (Haythornthwaite, Hegel, & Kerns, 1991). From the sleep diary data, daily values were calculated for sleep-onset latency (SOL), number of awakenings (NWAK), wake time after sleep-onset (WASO), total sleep time (TST), and sleep efficiency (SE; which is the ratio of time sleeping in bed to total time spent in bed). Daily values for these five

variables and daily ratings of sleep quality (SQ) were averaged across the 14 days of data to provide summary measures of self-reported sleep.

## **Procedure**

Recruitment took place at five local outpatient pain and medical clinics. Flyers advertising a study on “sleep patterns in people with chronic pain” were posted in clinic lobbies and exam rooms. Interested patients filled out flyers with their contact information or contacted the researchers directly. When patients expressed interest in the study, they spoke with the investigator (in person or over the telephone) to learn more about study requirements and answer screening questions about age, type of pain, pain duration, and possible sleep disorders. Eligible volunteers who wished to participate received study packets in person or by mail. The packets included a cover letter with instructions, informed consent documents, study questionnaires, sleep diaries, and a stamped return envelope. Participants receiving packets by mail were contacted approximately one week later to ensure the mailing was received and to answer any questions about the informed consent document or questionnaires.

Participation involved filling out six self-report measures that took a total of 30-45 minutes to complete and a 14 day record of sleep habits that took approximately 2 minutes each day. The questionnaires included a background information and health history form followed by the Beck Depression Inventory-II, Brief Pain Inventory, Pain Catastrophizing Scale, Pre-Sleep Arousal Scale, and the Insomnia Severity Index. The sequence of the last five questionnaires was randomized to minimize possible order effects. To ensure confidentiality, participants were assigned research identification numbers that were kept separate from their signed consent forms. Participants received \$10 compensation, which was delivered once the completed questionnaires were returned. For questionnaires that were returned with missing or incomplete

data, a researcher attempted to contact the participant to gather the missing data. Due to greater respondent burden, sleep diaries were considered a secondary measure and not required for participant compensation.

### **Statistical Analysis**

Sample size was based on power analyses for the hypothesis that cognitive PSA will be a significant and unique predictor of insomnia severity. In previous research with chronic pain patients, Smith and colleagues (2000) found a medium effect of cognitive PSA on sleep quality. Using the effect size found by Smith et al. for cognitive arousal ( $f^2 = .16$ ), 9 predictor variables, power of .70, and alpha of .05, a sample size of 48 participants is adequate (Cohen, 1987). Summary statistics were calculated for all study measures to describe the sample. Pearson correlations were conducted to examine the bivariate relations among predictor variables and insomnia severity. A hierarchical regression analysis was then conducted to examine the unique and shared contributions of pain catastrophizing and pre-sleep arousal to the prediction of insomnia severity, while controlling for demographics (i.e., age, sex, education level), depression severity, restless legs syndrome (RLS), and pain intensity.

## RESULTS

### Description of Sample

Scores on the Insomnia Severity Index (ISI) ranged from 4 to 26, with a mean score (see Table 2) indicating moderately severe clinical insomnia. Two-thirds (67%) of participants scored above the cut-off score ( $ISI \leq 15$ ) suggesting clinically significant insomnia (see Table 2 for breakdown of ISI categories and frequency of selected ISI items). However, when participants were asked a single item question on whether or not they had current difficulties sleeping, 47 of the 48 participants (98%) gave an affirmative response. Furthermore, when asked about the frequency of sleeping difficulties, 42% responded between 3-5 nights a week and 48% responded 6-7 nights a week. Average duration of sleep problems was reported to be 12.8 years ( $SD = 13.4$ ,  $n = 45$ ). Eighteen participants (38%) reported having sleep problems prior to their pain problem. Regarding specific sleep complaints, responses to the health history form indicated that daytime sleepiness was experienced by 81% of the sample, dozing off during the day by 56%, regular snoring by 50%, and symptoms of restless legs by 54%.

Sleep diary data were available for 42 participants (89%). Daily values for sleep-onset latency (SOL), number of awakenings (NWAK), wake time after sleep-onset (WASO), total sleep time (TST), sleep efficiency (SE), and sleep quality (SQ) were averaged across the two weeks. One participant did not complete both weeks of the sleep diary so his responses were averaged across the seven days that were completed. Mean SOL for the sleep diaries was 52 minutes ( $SD = 35.2$ ) with mean WASO of 51 minutes ( $SD = 34.5$ ). Participants reported an average of 2.5 awakenings per night ( $SD = 1.2$ ) and a mean TST of just over 6 hours ( $M = 367$



minutes,  $SD = 100.7$ ). Mean SE was 72.3% ( $SD = 12.8$ ). The mean sleep quality rating was 2.6 ( $SD = 0.6$ ), on a scale from 1 (*very poor*) to 5 (*excellent*). These sleep parameters support the summary findings from the ISI and health history, which indicate this sample of pain patients is experiencing significant symptoms of insomnia, as well as other sleep complaints.

The most commonly reported physical problems other than pain were hypertension (44%), gastrointestinal problems (42%), and respiratory problems (21%). The mean number of comorbid physical problems endorsed (out of 11 categories) was 1.56 ( $SD = 1.30$ ). Fifteen participants (31%) smoked cigarettes ( $M = 16.4$  cigarettes/day,  $SD = 6.1$ ), and all of them reported smoking within 2 hours of bedtime almost every night for the past 2 weeks. Thirty-five percent of the sample reported alcohol use, but alcohol consumption close to bedtime was rarely endorsed. In contrast, the majority of participants (88%) reported caffeine consumption and 46% of the sample consumed caffeine after 2pm almost everyday. Mean BMI was 29.0 ( $SD = 6.6$ ), indicating our sample was overweight. Regarding mental health, 63% of participants reported current or past diagnosis of a psychological/ learning disorder and 27% reported they are currently seeing a mental health professional. As shown in Table 2, the mean BDI-II score indicates this sample reported a moderate to severe level of depression. Means and standard deviations for other study variables are also presented in Table 2.

Medication lists were obtained from 47 participants. All 47 of these participants reported taking prescription medications. The number of reported medications ranged from 2 to 18, with a median of 7 and mean of 6.5 ( $SD = 2.9$ ). Thirty-two participants (68%) reported taking opiate analgesics. Of these 32 participants, 13 (40%) were prescribed two or more opiates. Twenty-three percent of the total sample reported taking non-steroidal anti-inflammatory drugs (NSAIDs), 47% muscle relaxants, 40% anticonvulsants, 64% antidepressants, and 38%

benzodiazepines. Across the three major classes of medications prescribed for pain (opioids, muscle relaxants, and NSAIDs), 85% of the total sample reported medication from at least one of these categories. Forty-five percent of the total sample took pain medications from at least two different categories. Finally, 24 participants (51%) reported taking hypnotics (including benzodiazepines and sedating antidepressants that were reportedly prescribed for sleep). Twenty-three (96%) of these 24 participants were also taking pain medications.

Table 2

*Means and Standard Deviations for Study Variables (N=48)*

Variable	<i>M</i>	<i>SD</i>
Insomnia Severity Index	17.1	5.2
Beck Depression Inventory-II	21.1	10.7
BPI Pain Intensity	5.7	1.5
Usual Bedtime Pain Ratings	5.9	2.0
PCS total	22.5	13.8
PSA Scale total	41.4	13.0
Cognitive PSA	25.2	9.0
Somatic PSA	16.1	5.3

*Note.* BPI= Brief Pain Inventory; PCS= Pain Catastrophizing Scale; PSA= Pre-Sleep Arousal.

Table 3

*Sleep Characteristics of Participants (N= 48)*

Selected ISI Items	<i>n</i>	%
Categories for ISI total score		
No insomnia	1	2.1
Sub-threshold Insomnia	15	31.3
Moderate Clinical Insomnia	21	43.8
Severe Clinical Insomnia	11	22.9
Item 1: Difficulty falling asleep <sup>a</sup>		
None	2	4.2
Mild	11	22.0
Moderate	14	29.2
Severe	18	37.5
Very Severe	3	6.3
Item 2: Difficulty staying asleep <sup>a</sup>		
None	0	0
Mild	4	8.3
Moderate	19	39.6
Severe	17	35.4
Very Severe	8	16.7
Item 3: Waking up too early <sup>a</sup>		
None	5	10.4
Mild	4	8.3
Moderate	15	31.3
Severe	18	37.5
Very Severe	6	12.5

*Note.* ISI = Insomnia Severity Index.

<sup>a</sup> Please rate the current (past 2 week's) SEVERITY of your insomnia problem.

## Associations Among Predictors

Correlational analyses (see Table 4) indicated that pain intensity, pain catastrophizing, cognitive PSA, somatic PSA, and depression severity were all positively associated with insomnia severity ( $p \leq .001$ ). As expected, BDI-II scores were also moderately to highly correlated with cognitive PSA, somatic PSA, PCS (all  $p < .001$ ), and pain intensity ( $p = .01$ ). Examination of the correlation matrix in Table 4 reveals a significant degree of overlap among the primary predictor variables. The PSA subscales (cognitive PSA and somatic PSA) were strongly correlated ( $r = .61, p < .001$ ) with one another. As expected, PCS scores were highly correlated with cognitive PSA ( $r = .70, p < .001$ ). PCS scores were also strongly correlated with somatic PSA ( $r = .66, p < .001$ ). This indicates that pain catastrophizing is associated with higher levels of both cognitive and somatic arousal at bedtime. Pain intensity was moderately correlated with somatic PSA ( $r = .35, p = .01$ ), but not cognitive PSA. Ratings of bedtime pain, which were strongly correlated with overall pain intensity ( $r = .76, p < .001$ ), showed a similar association with somatic PSA ( $r = .46, p = .001$ ), but not cognitive PSA. Pain intensity was moderately correlated with PCS ( $r = .30, p = .04$ ). Bedtime pain ratings showed a stronger association with PCS ( $r = .42, p = .003$ ).

Correlational analyses were also conducted with available sleep diary data ( $n = 42$ ). There were no significant associations between the six sleep diary parameters and measures of pain catastrophizing, cognitive PSA, or somatic PSA. However, greater pain intensity was associated with longer SOL ( $r = .34, p = .03$ ), shorter TST ( $r = -.45, p = .003$ ), and lower SE ( $r = -.40, p = .008$ ). Bedtime pain ratings were also associated with shorter TST ( $r = -.39, p = .01$ ) and lower SE ( $r = -.34, p = .03$ ), but they were not significantly related to SOL. Mean SOL, TST, and SE were all correlated with ISI scores in the expected direction ( $p < .05$ ).

Table 4

*Intercorrelations Between Insomnia Severity and Predictor Variables (N = 48)*

Variable	1	2	3	4	5	6	7	8	9	10
1. ISI total	--									
2. BDI-II total	.53**	--								
3. Pain Intensity	.47**	.36*	--							
4. PCS total	.50**	.70**	.30*	--						
5. Cognitive PSA	.52**	.54**	.07	.70**	--					
6. Somatic PSA	.54**	.55**	.35*	.66**	.61**	--				
7. Age	-.10	-.33*	-.05	-.30*	-.23	-.23	--			
8. Sex	.13	-.11	.14	-.13	-.24	-.37**	-.19	--		
9. Education	.04	-.07	-.28	-.30*	-.09	-.29*	-.09	-.05	--	
10. RLS symptom	.34*	.08	.24	.11	-.00	.28	-.35*	-.05	-.16	--

*Note.* ISI= Insomnia Severity Index; BDI= Beck Depression Inventory; PCS= Pain Catastrophizing Scale; PSA= Pre-Sleep Arousal; RLS= Restless Legs Syndrome.

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

### **Predictors of Insomnia Severity**

A hierarchical regression analysis was conducted to examine the relative contribution of pain catastrophizing and PSA to ISI scores. Pain intensity and other control variables were entered in step one, followed by PCS scores in step two, and both cognitive and somatic PSA scores in step three. As previously mentioned, there was moderate to high overlap among predictor variables. However, this collinearity was expected due to the nature of our research question. There were no significant violations of normality, linearity, and homoscedasticity.

The first step in the analysis included a set of control variables; age, sex (1= female, 2= male), education level (0= no college degree, 1= college degree), BDI-II scores, presence or absence of Restless Legs Syndrome (RLS) symptoms, and pain intensity scores. Together, these variables accounted for 52% of variance in ISI scores,  $F(6,41) = 7.37, p < .001$ . Entering PCS scores in step two provided a 7% increment in explained variance,  $F(1,40) = 6.40, p = .02$ . Finally, adding cognitive and somatic PSA in step 3 further increased explained variance by almost 13%,  $F(2,38) = 8.20, p = .001$ . Cognitive PSA was the only significant predictor in this step,  $\beta = .38, t(38) = 2.77, p = .009$ , uniquely accounting for 6% of variance in insomnia severity. Somatic PSA was not significantly associated with ISI scores in the presence of cognitive PSA. The full regression model was significant, accounting for 71% of total variance in ISI scores,  $F(9,38) = 10.35, p < .001$ . Importantly, PCS was no longer a significant predictor once cognitive PSA was entered into the model. Table 5 presents fully partialled values for all variables in the final model. Along with pain intensity, several control variables emerged as significant predictors of ISI in the final model; age, sex, education, and RLS symptoms.

Table 5

*Final Hierarchical Regression Analysis Predicting Insomnia Severity with Pain Catastrophizing and Pre-Sleep Arousal (N=48)*

Step and predictor	$\Delta R^2$	B	SE B	$\beta$	<i>t</i>	<i>sr</i> <sup>2</sup>
Step 1	.519**					
Age		.095	.045	.219	2.11*	.034
Sex		3.61	1.20	.305	2.99**	.068
Education		3.38	1.04	.329	3.26**	.081
BDI-II total		.083	.065	.171	1.28	.013
RLS symptoms		3.32	1.05	.323	3.15**	.076
Pain Intensity		.896	.361	.263	2.48*	.047
Step 2	.066*					
PCS total		.006	.061	.016	.097	.000
Step 3	.125**					
Cognitive PSA		.215	.078	.375	2.77**	.059
Somatic PSA		.275	.144	.283	1.91	.028

*Note.* Values for individual predictors reflect fully partialled values from final regression model.

*sr*<sup>2</sup> = squared semipartial correlation coefficient; BDI= Beck Depression Inventory; RLS=

Restless Legs Syndrome; PCS= Pain Catastrophizing Scale; PSA= Pre-Sleep Arousal.

Total  $R^2 = .709$ .

\*  $p < .05$ ; \*\*  $p < .01$ .

## DISCUSSION

This study provides further support for the role of cognitive factors in insomnia comorbid with chronic pain. After controlling for pain intensity and other covariates, cognitive PSA was the most important predictor of insomnia severity in our sample of chronic pain patients. Cognitive PSA also accounted for the relation between pain catastrophizing and somatic PSA with insomnia severity.

Together, the cognitive and somatic pre-sleep arousal subscales explained an additional 13% of variance in insomnia severity in chronic pain patients. In support of our hypothesis, cognitive PSA appeared to drive this effect, uniquely explaining 6% of variance, while somatic PSA was non-significant. This finding is consistent with Smith et al's (2000) study that found cognitive PSA to be more salient to poor sleep quality in chronic pain patients than somatic arousal. It is also consistent with previous research on cognitive arousal and primary insomnia. However, these results do not imply that somatic arousal is an unimportant component of comorbid insomnia. There is a strong association between the cognitive and somatic dimensions of arousal, as evidenced by the high correlation ( $r = .61$ ) between the two PSA subscales. Cognition and physiology are interrelated, such that cognitive hyperarousal (particularly when it involves negatively valenced or threatening material) induces physiological arousal, and in turn, physiological arousal triggers cognitive activity as individuals search for an explanation of their somatic symptoms.

An aim of the present study was to explore the potential association between pain catastrophizing and insomnia. In support of our first hypothesis, PCS scores made a unique



contribution to the prediction of insomnia severity after controlling for the effects of pain intensity, depression, RLS symptoms, and demographics. However, once pre-sleep arousal was taken into account, pain catastrophizing was no longer a significant predictor of insomnia severity. Thus, our hypothesis regarding the independent contributions of cognitive arousal and pain catastrophizing was partially supported, as only cognitive PSA accounted for unique variance in insomnia severity. This last finding is likely due to shared explained variance. Cognitive PSA and PCS scores were highly correlated with each other, which could represent item-content overlap. For instance, several items assessing general cognitive pre-sleep arousal (e.g., “thoughts keep running through your head” or “worry about problems other than sleep”) may also be tapping into the more specific construct of pain catastrophizing. One might also argue that this overlap exists because the two scales are essentially measuring the same construct. However, the current results support some degree of conceptual independence between pain catastrophizing and cognitive arousal. If the PCS and the cognitive PSA subscale were measuring the same construct, then the addition of cognitive PSA would not have improved the amount of variance explained in insomnia over and above the effects of PCS scores.

Notably, pain intensity remained a significant predictor of insomnia severity, even in the presence of pain catastrophizing, pre-sleep arousal, and other control variables. Although the contribution was small, it suggests that the experience of more intense pain is uniquely linked with insomnia symptoms. Previous studies examining the relation between pain severity and sleep disturbance in pain patients have yielded mixed results, which seem to depend on how pain was assessed. For example, a recent study found that nighttime pain ratings predicted poorer sleep quality in chronic pain patients, while daytime pain ratings made no significant contribution (Ashworth, Davidson, & Espie, 2010). In a study with back pain patients, Tang et al

(2007) investigated different dimensions of the pain experience and found that ratings of present pain intensity no longer predicted insomnia severity once affective ratings of pain (i.e., emotional evaluations of pain as unpleasant) were taken into account. Seeing as though pain is a multidimensional experience, it is likely that some of the cognitive processes involved in the perception and evaluation of painful sensations are similar to those used by people with insomnia to appraise their sleep.

An unanticipated finding was the lack of correlation between measures of pre-sleep arousal and sleep diary parameters. Previous research has consistently demonstrated a positive association between cognitive arousal and subjective sleep-onset latency (SOL), which was not replicated in our current study. One possible explanation for this inconsistency could be a mismatch of the time frames captured by the retrospective measure of pre-sleep arousal and the prospective measure of daily sleep pattern. In order to reduce participant burden and facilitate participation in our study, the timing of the retrospective questionnaires and sleep diaries was not standardized. Thus, the PSA scale could be completed immediately (pertaining to the previous two weeks), and the sleep diaries could then be completed for the following two weeks. Conversely, the PSA scale could be completed after finishing the sleep diaries so that both measures refer to the same time period.

Results of the present study are strengthened by the use of a validated, insomnia-specific outcome measure, the Insomnia Severity Index. The percentage of our sample (67%) with ISI scores suggesting clinical insomnia was similar to rates in a recent study with pain patients (Tang et al, 2007). Interestingly, a much higher percentage (98%) of our sample reported “current difficulties sleeping.” This discrepancy between the number of general sleep complaints and indications of clinically significant insomnia highlights the improved sensitivity and specificity

of the ISI. Future research should continue to implement more detailed assessments of a patient's sleep disturbance. Another strength of this research is the use of a clinical pain sample with minimal exclusionary criteria. Participants were not excluded due to psychiatric comorbidity or medication use, which improves the generalizability of these findings to other pain patients. Due to increased risk of restless legs syndrome (RLS) in certain pain groups (e.g., Viola-Saltzman, Watson, Bogart, Goldberg, & Buchwald, 2010) and similarities between patients with RLS/PLMD and patients with primary insomnia (Edinger, 2003), our sample also included participants with RLS complaints. Given the high rate (54%) of participants with affirmative responses to the question, "Do your legs jerk during the night or feel restless when you lay down to fall asleep?" and the independent positive association between these symptoms and ISI scores, further investigation into this area is recommended.

Despite the strengths of these findings, there are some limitations to consider. Since recruitment flyers and the study title mentioned sleep, there is possible self-selection bias, in that individuals with sleep problems were more likely to volunteer and show interest in this study. Thus, the current sample may over represent those with significant sleep disturbance. The use of cross-sectional data further limits our ability to draw conclusions about the direction of effects, but provides a starting point for continued discussion in the literature. Prospective, longitudinal studies utilizing daily diary measures of sleep, pain, and pre-sleep arousal would allow for examination of the temporal association between these factors.

Finally, potential confounds still remain. Although careful attempts were made to exclude individuals with probable sleep apnea, we did not conduct diagnostic studies to confirm this. Also, we included patients with restless leg complaints, but did not establish RLS diagnoses or assess RLS severity. Thus, the role of this disorder in driving the insomnia complaint could not

be definitively evaluated. Many of the participants were prescribed hypnotics or pain medications with sedating properties (e.g., opioid analgesics, muscle relaxants). Because a high percentage (81%) of the sample reported daytime sleepiness, it was unclear how much of this daytime drowsiness could be attributed to sleep disturbance and how much was due to medication effects. Conversely, inadequate pain control could disrupt sleep by allowing breakthrough pain during the night. Future research may also wish to consider other cognitive processes that have been implicated in insomnia comorbid with chronic pain, but were not assessed in this study; health anxiety (Tang et al, 2007) and dysfunctional beliefs about sleep (Ashworth et al, 2010).

Our findings highlight the important contribution of cognitive pre-sleep arousal to insomnia co-occurring with chronic pain. Although somatic arousal and pain catastrophizing were also associated with insomnia severity, they were not significant in the presence of cognitive arousal. However, our results do not negate the potential role of pain catastrophizing. Due to the strong association between measures of pain catastrophizing and cognitive pre-sleep arousal, future research could examine how catastrophic thinking about pain may fuel cognitive arousal at bedtime and contribute to insomnia in people with chronic pain.

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## APPENDIX

## Pain Catastrophizing Scale

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

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

<i>When I'm in pain ...</i>	Not at all	To a slight degree	To a moderate degree	To a great degree	All the time
1. I worry all the time about whether the pain will end.	0	1	2	3	4
2. I feel I can't go on.	0	1	2	3	4
3. It's terrible and I think it's never going to get any better.	0	1	2	3	4
4. It's awful and I feel that it overwhelms me.	0	1	2	3	4
5. I feel I can't stand it anymore.	0	1	2	3	4
6. I become afraid that the pain will get worse.	0	1	2	3	4
7. I keep thinking of other painful events.	0	1	2	3	4
8. I anxiously want the pain to go away.	0	1	2	3	4
9. I can't seem to keep it out of my mind.	0	1	2	3	4
10. I keep thinking about how much it hurts.	0	1	2	3	4
11. I keep thinking about how badly I want the pain to stop.	0	1	2	3	4
12. There's nothing I can do to reduce the intensity of the pain.	0	1	2	3	4
13. I wonder whether something serious may happen.	0	1	2	3	4

*What type of pain were you thinking about as you as you answered the previous questions?*

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## Pre-Sleep Arousal Scale

For the list below, think about your experience during the pre-sleep period (in bed with the lights out before falling asleep for the first time). **Please rate how intensely you experience each item as you attempt to fall asleep in your bed each night.**

	Not at all	Slightly	Moderately	A Lot	Extremely
1. Heart racing, pounding, or beating irregularly	1	2	3	4	5
2. A jittery, nervous feeling in your body	1	2	3	4	5
3. Shortness of breath or labored breathing	1	2	3	4	5
4. A tight, tense feeling in your muscles	1	2	3	4	5
5. Cold feeling in your hands, feet, or body in general	1	2	3	4	5
6. Have stomach upset (knot or nervous feeling in stomach, heartburn, nausea, gas, etc)	1	2	3	4	5
7. Perspiration in palms of your hands or other parts of your body	1	2	3	4	5
8. Dry feeling in mouth or throat	1	2	3	4	5
9. Worry about falling asleep	1	2	3	4	5
10. Review or ponder events of the day	1	2	3	4	5
11. Depressing or anxious thoughts	1	2	3	4	5
12. Worry about problems other than sleep	1	2	3	4	5
13. Being mentally alert, active	1	2	3	4	5
14. Can't shut off thoughts	1	2	3	4	5
15. Thoughts keep running through your head	1	2	3	4	5
16. Being distracted by sounds, noise in the environment (e.g., ticking of clock, house noises, traffic)	1	2	3	4	5

July 2, 2010

Office for Research

Institutional Review Board for the  
Protection of Human Subjects

THE UNIVERSITY OF  
**ALABAMA**  
R E S E A R C H

Haley Dillon  
Department of Psychology  
College of Arts & Sciences  
The University of Alabama

Re: IRB # 09-OR-208-ME-R1 "Cognitive Mechanisms in Comorbid  
Insomnia and Chronic Pain"

Dear Ms. Dillon:

The University of Alabama Medical Institutional Review Board has  
granted approval for your renewal application

Your renewal application has been given expedited approval according to  
45 CFR part 46. Approval has been given under expedited review  
category 7 as outlined below:

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

Your application will expire on June 30, 2011. If your research will  
continue beyond this date, complete the relevant portions of Continuing  
Review and Closure Form. If you wish to modify the application,  
complete the Modification of an Approved Protocol Form. When the  
study closes, complete the appropriate portions of FORM: Continuing  
Review and Closure.

Please use reproductions of the IRB approved informed consent form to  
obtain consent from your participants.

Should you need to submit any further correspondence regarding this  
proposal, please include the above application number.

Good luck with your research.

Sincerely,



152 Rose Administration Building  
Box 870117  
Tuscaloosa, Alabama 35487-0117  
(205) 348-8461  
FAX (205) 348-8882  
TOLL FREE (877) 820-3066

Director & Research Compliance Officer  
Office for Research Compliance  
The University of Alabama

09-OR-208-ME-2\

UNIVERSITY OF ALABAMA  
INSTITUTIONAL REVIEW BOARD FOR THE PROTECTION OF HUMAN SUBJECTS  
REQUEST FOR APPROVAL OF RESEARCH INVOLVING HUMAN SUBJECTS

I. Identifying information

	Principal Investigator	Second Investigator	Third Investigator
Names:	Haley Dillon	Kenneth Lichstein	
Department:	Psychology	Psychology	
College:			
University:	University of Alabama	University of Alabama	
Address:	BOX #870348	BOX #870348	
Telephone:	[REDACTED]	[REDACTED]	
FAX:			
E-mail:	hrdillon@crimson.ua.edu	lichstein@ua.edu	

Title of Research Project: Cognitive Mechanisms in Comorbid Insomnia and Chronic Pain

Date Submitted: 6/28/10

Funding Source: Graduate Student Research and Travel Support Award (matched contributions from the UA Graduate School and the UA Psychology Department)

Type of Proposal  New  Revision  Renewal  Completed  Exempt

Please attach a renewal application

Please attach a continuing review of studies form

Please enter the original IRB # at the top of the page

UA faculty or staff member signature: \_\_\_\_\_

II. NOTIFICATION OF IRB ACTION (to be completed by IRB):

Type of Review: \_\_\_\_\_ Full board  Expedited

IRB Action:

- Rejected Date: \_\_\_\_\_
- Tabled Pending Revisions Date: \_\_\_\_\_
- Approved Pending Revisions Date: \_\_\_\_\_

Approved-this proposal complies with University and federal regulations for the protection of human subjects.

Approval is effective until the following date: 6-30-11 a.s.

- Items approved:
- Research protocol (dated \_\_\_\_\_)
  - Informed consent (dated \_\_\_\_\_)
  - Recruitment materials (dated \_\_\_\_\_)
  - Other (dated \_\_\_\_\_)

Approval signature

Date 7/1/2010

**Sleep and Pain Study – Informed Consent**  
**The University of Alabama**

**Title of Research: Normal and Disturbed Sleep Patterns in Chronic Pain**

**Investigator(s): Haley Dillon, Kenneth L. Lichstein, PhD**

**IRB Approval #:**

**What is the purpose of this study—what is it trying to learn?**

This study is looking at how sleep and pain experiences may be related. You have been asked to be in this study because you are an adult who has had chronic pain for at least 6 months. One hundred participants will be recruited for this study.

**What will I be asked to do in this study?**

If you agree to be in this study, you will be asked to fill out several questionnaires on your health, sleep patterns, pain experiences, and how you deal with these areas of your life. The questionnaires take about 45 minutes to fill out and can be completed at the clinic or in your home. You will also be given a questionnaire called a "Sleep Diary" to fill out at home every day for two weeks (14 days). It takes about 5 minutes to fill out each morning. The Sleep Diary asks questions about what time you went to bed the night before, what time you woke up this morning, how many times you woke up during the night, etc. When you finish the two weeks of diaries, you will mail them back to us in a stamped return envelope we will give you.

To thank you for your time and participation, you will receive \$10 payment after completing the questionnaires.

**What are the benefits of being in this study?**

Potential benefits are that you may gain some insight into how research studies in psychology work, and into how your sleep patterns and pain experiences influence each other. Participation in this study may also provide us with information that can be used to better understand and improve comprehensive health care for individuals with chronic pain.

**What are the risks to me if I am in this study?**

The only foreseeable risk of participation is that providing personal information may make you feel uncomfortable. However, any discomfort will be temporary and will likely not be more than what you would experience in a routine day.

**How will my privacy and confidentiality be protected?**

We will not tell anyone you are in this study. The only exception is that we are required to report to the appropriate authorities if you choose to inform us of: 1) serious thoughts about harming yourself or someone else and/or 2) behaviors related to abuse of a child or older

adult. Except for these severe cases, all information about you and your participation in this study will not be revealed to anyone for any reason. You do not have to answer any questions or give us any information that you do not want to.

We will protect your information by giving you an identification number. Your name will not appear on any study document besides this consent form and a payment receipt. There is no way to link consent forms and names with data. The data from the study will be kept in locked file drawers in locked offices. No one will have access to it except the investigators. Your healthcare provider will not have access to the information you provide. We will publish scientific articles on this study but no clinics, towns, or Alabama counties will be identified. No one will be able to tell who you are.

**Do I have to be in this study?**

No. If you decide to be in this study it should be because you really want to volunteer. You can refuse to be in the study. You can also start the study and decide to stop at any time. If you refuse to participate or start the study and then stop, it will not affect your medical care in any way. As an alternative to participating in this study, you may choose not to participate.

**What if we have questions, suggestions, concerns, or complaints?**

If you have questions during or following the project, you may contact the principal investigator, Haley Dillon, at [REDACTED]. If you have any questions about your rights as a research participant, please contact Ms. Tanta Myles, Research Compliance Officer at [REDACTED].

You will be given a copy of this consent form to keep. Save it in case you want to review it later or you decide to contact the investigator or the university about the study.

**I have read this consent form. I have had a chance to ask questions and my questions have been answered. I understand what I will be asked to do and I freely agree to take part in it.**

\_\_\_\_\_  
Signature of participant

\_\_\_\_\_  
Date

\_\_\_\_\_  
Signature of witness

\_\_\_\_\_  
Date