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Acute Pain and Depressive Symptoms: Independent Predictors of Insomnia Symptoms among Adults with Sickle Cell Disease

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Abstract

Background—No studies to-date have systematically investigated insomnia symptoms among adults with sickle cell disease (SCD). The purpose of this study was to 1) describe the prevalence of insomnia symptoms and 2) identify bio-psychosocial predictors in community-dwelling adults with Sickle Cell Disease.

Methods—Cross-sectional analysis of baseline data from 263 African-American adults with SCD (aged 18 years or older). Measures included the Insomnia Severity Index (ISI), Center for Epidemiologic Studies in Depression scale, Urban Life Stress Scale, Brief Pain Inventory, and a chronic pain item. SCD genotype was extracted from the medical record.

Results—A slight majority (55%) of the sample reported clinically significant insomnia symptomatology (ISI ≥10), which suggests that insomnia symptoms are prevalent among community-dwelling African-American adults with SCD. While insomnia symptoms were associated with a number of bio-psychosocial characteristics, depressive symptoms and acute pain were the only independent predictors.

Conclusion—Given the high number of participants reporting clinically significant insomnia symptoms, nurses should screen for insomnia symptoms and to explore interventions to promote better sleep among adults with SCD with an emphasis on recommending treatment for pain and depression. In addition, current pain and depression interventions in this population could add insomnia measures and assess the effect of the intervention on insomnia symptomatology as a secondary outcome.

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Conflict of Interest/Disclosures

All authors declare that they have no financial or personal conflicts of interest to disclose.

Keywords

Insomnia Symptoms; Depression; Pain; Sickle Cell Disease

Background

An estimated 90,000 to 100,000 people in the United States (US) are reported to have sickle cell disease (SCD), a serious genetic disorder resulting from an abnormal hemoglobin gene, *HbS* (National Heart Lung and Blood Institute, 2012). SCD is characterized by intermittent "sickling" of red blood cells (National Heart Lung and Blood Institute, 2012). Sickled red blood cells can slow or block transport of oxygen and nutrients to the tissues and organs (National Heart Lung and Blood Institute, 2012). As a result, individuals with SCD may experience a number of serious consequences related to tissue and organ damage. Prior to the 1970s, individuals with SCD did not survive past the 2nd decade of life; however in the US, major advances in care, specifically comprehensive preventive care and awareness of early signs of complications, have improved the life expectancy of individuals with SCD to the seventh decade and beyond (National Heart Lung and Blood Institute, 2002, 2012). A shift to chronic disease management has brought an expressed desire by patients to focus on Health-Related Quality of Life (HR-QOL) across the lifespan (National Heart Lung and Blood Institute, 2002). The experience of symptoms, especially when distressing, has been negatively correlated with HR-QOL in adults with SCD (Sogutlu, Levenson, McClish, Rosef, & Smith, 2011).

While pain remains a major distressing symptom for patients with SCD (W R Smith et al., 2008), attention to other burdensome symptoms associated with decreased HR-QOL have increased in the literature during the past few years. Among these symptoms are sleep abnormalities, broadly described in the literature as "sleep disturbances." Sleep disturbance is a general term for a variety of subjective and objective sleep complaints associated with alterations in sleep/wake patterns and sleep disorders (Cormier, 1990). Sleep disturbances are common among patients with chronic disease (National Center on Sleep Disorders Research, 2003) and prevalence estimates of 40–70% (Barker et al., 2012; Jacob et al., 2006; Sogutlu et al., 2011; Wallen et al., 2014) have been reported among children and adults with SCD. In children, specific sleep disturbances associated with the sleep disorder insomnia, namely difficulty falling asleep and staying asleep, have been identified using daily sleep diaries (Valrie, Gil, Redding-Lallinger, & Daeschner, 2007a). In addition, using retrospective chart review, one recent study found insomnia symptoms were documented for 47% of the adults with SCD in that study (Mann-Jiles, Thompson, & Lester, 2015). Qualitative literature on sleep disturbances suggests that sleep disturbances have been reported as a distressing symptom (Panepinto, Torres, & Varni, 2012; Weisberg, Balf-Soran, Becker, Brown, & Sledge, 2013) and a quality of life priority for individuals with SCD (Treadwell, Hassell, Levine, & Keller, 2014). In addition, obtaining adequate sleep has been reported as a self-management strategy for preventing painful crises (Anderson & Asnani, 2013; Tanabe et al., 2010). Despite the prevalence and impact of sleep disturbances on quality of life among patients with SCD, there is a paucity of literature on factors that are associated with this symptom and especially among adults with the disease.

Two known predictors of sleep disturbances in patients with SCD include depression (Palermo & Kiska, 2005; Wallen et al., 2014) and pain (Valrie, Gil, et al., 2007a; Wallen et al., 2014). Two other potential predictors, identified through studies in patients with SCD and other chronic diseases, are psychological stress and disease severity. In a qualitative study by Weisberg et al (2013), a participant with SCD was quoted as saying "And I would be scared to go to sleep, because I would think I was going to die in my sleep" suggesting sleep disturbances may arise from psychological stress (Weisberg et al., 2013). In addition, across studies disease severity and disease activity are consistently identified as risk factors for sleep problems among chronic disease populations (Chandrasekhara, Jayachandran, Rajasekhar, Thomas, & Narsimulu, 2009; Frech et al., 2011; Martínez De Lapiscina, Aguirre, Blanco, & Pascual, 2012).

While a few studies have investigated sleep disturbances in adults with SCD (Sogutlu et al., 2011; Wallen et al., 2014), no studies to-date have systematically investigated insomnia symptoms in this population. Given sleep disturbances, specifically disturbances associated with insomnia, have been identified in individuals with SCD, examining the prevalence of insomnia symptoms and exploring potential risk factors (or predictors) should be undertaken to understand the scope and complexity of this sleep abnormality among adult patients with SCD. Guided by Theory of Unpleasant Symptoms, which posits that physiologic, psychological, and situational factors influence the development and experience of symptoms (Lenz, Pugh, Milligan, Gift, & Suppe, 1997), this study had two purposes. The first was to describe the prevalence of insomnia symptoms and the second was to identify biological and psychosocial predictors of the development and severity of insomnia symptoms among community-dwelling adults with sickle cell disease.

Methods

Participants

This study is a cross-sectional analysis of baseline data provided by 263 African-American adults with SCD (aged 18 years or older) enrolled in the Improving Patients Outcomes with Respect and Trust (IMPORT) study. The IMPORT study was a prospective cohort study, which examined the experiences of care received by adolescents and adults with SCD (age 15 years or older). Participants were conveniently recruited from SCD outpatient clinics in the Mid-Atlantic region. Individuals were eligible to participate if they were 1) 15 years or older, 2) had HbSS, HbSC, Hb SS/β^0-thalassemia, or Hb SS/β^+-thalassemia sickle hemoglobinopathies, and 3) no plan to move within three years of enrolling in the study. A total of 292 participants provided informed consent and were enrolled in the parent study between May 2010 and December 2011.

All study procedures were approved by Institutional Review Boards at each study site. At baseline, participants completed a battery of comprehensive validated questionnaires used to assess self-reported demographic, health-related, and psychosocial data. All self-report baseline data were collected using an audio computer-assisted self-interview (ACASI) system. In addition to baseline questionnaires, a trained research assistant extracted clinical health data (e.g., medial procedures, lab results, and comorbidities) from the participant's medical record.

Measures

Insomnia symptoms include difficulty initiating and/or maintaining sleep, unintended early morning awakening, and non-restorative sleep with daytime consequences (Roth, 2007; Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). We used the *Insomnia Severity Index (ISI)* to measure perceived severity of insomnia symptoms, concern or distress, and interference with daily functioning experienced during the past two weeks (Bastien, Vallières, & Morin, 2001; Morin, Belleville, Bélanger, & Ivers, 2011). Each item on the 7-item ISI is scored on a 5-point Likert scale from 0 (“not at all”) to 4 (“extremely”). Overall, higher total scores (range zero to 28) indicate increased presence and severity of insomnia symptoms. Scores of 10 or higher can be used to identify clinically significant insomnia symptoms, while scores over 14 identify clinical insomnia cases with a high degree of accuracy (Gagnon, Bélanger, Ivers, & Morin, 2013; Morin et al., 2011; Savard, Savard, Simard, & Ivers, 2005). Overall, we have found that the ISI is a valid and reliable measure of insomnia symptoms in adults with SCD (Moscou-Jackson, Allen, Smith, & Haywood, in press) and had good internal consistency among participants in our sample (Cronbach’s alpha was 0.91).

Baseline socioeconomic and demographic characteristics were included in this study as potential predictors of insomnia symptomatology. Socioeconomic and demographic characteristics including age, sex, educational attainment, and employment status were self-reported by participants.

Depressive symptomatology was a psychological predictor in this study. The 10-item *Center for Epidemiologic Studies in Depression (CESD)* scale was used to measure depressive symptomatology. Item response choices, on a 4-point Likert scale, range from 0 (“Rarely or none of the time”) to 3 (“Most or all of the time) with a time frame of the past week. A summary score across items, which can range from zero to 30, is calculated. Participants who score 10 or higher may be at risk for clinical depression (Andresen, Malmgren, Carter, & Patrick, 1994). Since the CESD-10 includes the item “My sleep was restless,” results using the CESD-10 scale with and without the sleep item were compared. Overall, the CESD-10 is a valid measure of depressive symptoms among African-American adults with SCD (Laurence, George, & Woods, 2006) and had good internal consistency reliability among participants in our sample (Cronbach’s alpha = 0.83).

Perceived stress was another psychosocial predictor in this study. Perceived stress was measured using the 21-item *Urban Life Stress Scale (ULSS)*. The ULSS scale measures the degree of daily psychological and emotional stress experienced by defined contextual community-level stressors (Sanders-Phillips & Harrell, n.d.). Each item is measured on a 5-point Likert scale from 0 (“no stress at all”) to 4 (“extremely stressful”). Scores from each item are summed to produce a total score from zero to 84, where higher scores indicate greater perceived life stress (Sanders-Phillips & Harrell, n.d.; Jaffee et al., 2005). The Cronbach’s alpha for the ULSS was 0.89 for our sample.

Pain has been identified as a consistent risk factor for insomnia symptoms in other populations, thus was included as a biological predictor in this study. Given individuals with SCD experience both acute and chronic SCD pain (Smith & Scherer, 2010), we included a

measure of both in this study. A single item, “Do you have daily chronic pain?” with response options of “yes” or “no” was asked. This item was not bound by a specific timeframe therefore participants were free to interpret whether they experienced chronic SCD pain. This item has been used in several studies of SCD by the IMPORT research team. In a prior study, the IMPORT team demonstrated that this item performed as well as “the number of good days [the participant] experienced each week” (higher score equals a lower burden of pain during the week) and “self-reported pain on a good day” (higher score equals more severe pain on a good day) (Haywood et al., 2014), which are other items used to measure the burden of chronic pain in adults with SCD. Therefore, we selected this item as a valid measure of chronic SCD pain for the current study.

To measure acute pain, we used the *Brief Pain Inventory (BPI).* The BPI is an instrument used to measure two dimensions of pain, pain severity (4-items) and the degree to which pain interferes with daily functioning and feelings (7-items; Cleeland, 2009). The pain severity items (i.e. “worst,” “least,” “average,” and “current” pain rating in the past 24 hours) were used to measure acute pain severity. The response options ranged from 0 (“no pain”) to 10 (“as bad as you can imagine”). A composite pain severity score is created from the four BPI severity items (range zero to 10). The Cronbach’s alpha for the BPI-severity questions was 0.87 in this study sample.

In the absence of a SCD-specific disease severity index (Coelho et al., 2012), SCD genotype (an individual’s inherited genetic information) has been used (Grant, Gil, Floyd, & Abrams, 2000; Wison Schaeffer et al., 1999) and was used in this study as a measure of disease severity. *SCD genotype* (HbSS, HbSC, HbS/β^0-Thalassemia, and HbS/β^+-Thalassemia) was extracted from the participant’s medical chart by trained research assistants using a medical record extraction form at baseline. In general, individuals with the HbSS or HbS/β^0-Thalassemia genotypes have similar phenotypic expressions (observable traits) and are generally more severely affected than individuals with HbSC or HbS/β^+-Thalassemia. Due to similarities in their severity profile, SCD genotype was dichotomized by phenotype expression (HbSS or HbS/β^0-Thalassemia versus HbSC or HbS/β^+-Thalassemia) for the present analyses.

Statistical Analysis

Participant characteristics were described using means with standard deviations and frequencies with percentages where appropriate. Pearson’s product-moment correlations, t-tests, and one-way ANOVAs were used where appropriate to establish unadjusted relationships between insomnia symptom severity and potential bio-psychosocial predictors. As a final step, we used multiple linear regression analysis (backward-selection) to determine which bio-psychosocial characteristics were independently associated with insomnia symptom severity. All characteristics that were associated with insomnia symptom severity in the bivariate analyses were included in the models. Linear regression results are reported as unstandardized beta coefficients ($\hat{\beta}$), an estimate of the relative and unique contribution of each bio-psychosocial predictor to insomnia symptom severity. A probability of > 0.1 (two-tailed) was set as criterion for removal from the model and statistical

significance was determined as a probability < 0.05 (two-tailed). All analyses were conducted using Stata 13 software (StataCorp, 2013).

We conducted an *a priori power* analysis to determine if our sample size was adequate for using multiple regression method to identify a statistical model of predictors of insomnia symptoms. Power was set at the conventional 0.80 with statistical significance at 0.05. We determined that with our sample size (n= 263), we had at least 80% power to detect a small to medium effect size (Cohen's $f^2 > 0$ .08).

Results

Participant Characteristics

Table 1 displays characteristics for the 263 participants in this study. Seventy percent of the sample had SS or HbS/β^0-Thalassemia genotype. Educational attainment was low with 62.8% of the sample reporting less than a high school education. Further, a large proportion of the sample (42.6%) reported not working because of a disability. Although a slight majority of the sample reported experiencing chronic pain (56.6%), acute pain severity in the past 24 hours was low (mean 3.8, range 0 to 10). Among this sample, insomnia symptom severity was high with a slight majority (55%) of the sample reporting clinically significant insomnia symptomatology (ISI ≥10), while 41% may have clinical insomnia (ISI ≥14; data not presented).

Characteristics Associated with Insomnia Symptom Severity

Age, sex, education, employment status, depressive symptomatology, perceived stress, pain, and disease severity (as measured by SCD genotype) were analyzed for their unadjusted association with insomnia symptom severity (Table 2). Overall, individuals with a college education or higher reported less insomnia severity than individuals who had less than a college education (p = 0.03). When comparing insomnia severity by employment status, individuals who were not working because of a disability reported greater insomnia severity than individuals who were not disabled (p = 0.002).

We found paradoxical findings between SCD genotype and insomnia severity. Individuals with genotype SS or HbS/β^0-Thalassemia reported significantly lower insomnia severity than individuals with SC or HbS/β^+-Thalassemia (p = 0.02). Individuals with chronic pain reported greater insomnia severity than individuals without chronic pain (p < 0.001). In addition, acute pain, depressive symptoms, and perceived stress were positively associated with insomnia severity (p < 0.05). The relationship between depressive symptoms and insomnia symptomatology remained after excluding the sleep item from the CESD-10, therefore the full CESD-10 scale was used in all subsequent analyses.

Independent Statistical Predictors of Insomnia Symptom Severity

A stepwise backward-selection linear regression model was tested to determine which characteristics were independently associated with insomnia symptom severity (Table 3). Characteristics that were associated with insomnia symptom severity at the bivariate-level were included in the Model (i.e. education, disability status, SCD genotype, pain (acute and

chronic), perceived stress, and depressive symptoms). Stepwise regression analysis revealed that only acute pain and depressive symptoms were independent statistical predictors of greater insomnia severity. These results suggest that each unit increase in acute pain severity would result in almost a half a point increase in insomnia severity (as measured with the Insomnia Severity Index; $\hat{\beta} = 0.45$, p = 0.002). Similarly, for each unit increase in depressive symptom severity (as measured by the CESD-10), insomnia severity would increase by almost three-quarters of a point ($\hat{\beta} = 0.71$, p <0.01). Together, 49% of the variability in insomnia symptom severity can be explained by acute pain severity and depressive symptoms suggesting that acute pain severity and depressive symptoms are powerful predictors of insomnia severity. In a post-hoc sensitivity analysis, we dichotomized our outcome according to the clinical cut-point for the insomnia severity index (ISI < 10 versus ISI ≥ 10). Using logistic regression, we found no difference in our results, which supported the validity of our findings.

Discussion

To our knowledge, this is one of the first studies to explore insomnia symptomatology and potential bio-psychosocial predictors among community-dwelling adults with SCD. Our results suggest that insomnia symptoms are prevalent among adults with SCD, with 55% of participants reporting insomnia severity scores above the clinical cut-off (ISI ≥ 10; Morin et al., 2011). The prevalence of insomnia symptoms in this study is higher than the 30% prevalence of insomnia symptoms found in the general population (Schutte-Rodin et al., 2008). Based on these findings, nurses working with adult patients with SCD should routinely screen for insomnia symptoms and other signs of sleep disturbances.

We used the Theory of Unpleasant Symptoms (TOUS) and literature to guide our selection of bio-psychosocial characteristics believed to be independently associated with insomnia symptoms and severity in adults with SCD. Similar to other studies, we found that acute pain and depressive symptoms were independent predictors of insomnia symptom severity (Buenaver et al., 2012; Chandrasekhara et al., 2009; Heffner, France, Trost, Ng, & Pigeon, 2011; Huang & Lin, 2009; Palermo & Kiska, 2005; Palesh et al., 2007); however, among other potential risk factors, we found weak or non-significant associations.

Of note, we found that disease severity, as measured by SCD genotype, was not an independent risk factor of insomnia symptom severity. Further, we found individuals with the SC or HbS/β^{+}-Thalassemia genotype reported more severe insomnia symptomatology than individuals with SS or HbS/β^{0}-Thalassemia which are considered the most severe genotypes. This finding is largely unsupported in the literature. However, anecdotally clinicians working with adults with SCD have noted that although higher morbidity is associated with individuals with SS or HbS/β^{0}-Thalassemia, the day-to-day experience is similar for everyone. Overall, a small number of studies have investigated the relationship between symptoms including sleep disturbances and disease severity with mixed results. Among these studies, and in our study, it was believed that disease severity would be positively associated with insomnia severity (Ameringer, Elswick, & Smith, 2014). It is possible that the measure of disease severity used in our study did not accurately capture disease severity. It is also possible that the association between disease severity and

insomnia severity was weak and our study was underpowered to detect a predictive relationship.

There are a few notable limitations in this study that cannot be ignored. First, this was a cross-sectional examination of insomnia symptoms in adults with SCD, which provides a snapshot of the symptom experience. Second, this study was a secondary analysis of an existing dataset, thus only variables collected by the parent study were explored. Other potential explanatory variables of insomnia symptom severity (e.g., personal habits and obstructive sleep apnea) could not be examined. Finally, in post-priori power analysis, we determined our sample size was likely too small to detect significant independent associations for bio-psychosocial characteristics that were very weakly associated with insomnia symptom severity. However, their weak association may suggest they are not a strong predictor of insomnia symptoms. Despite these limitations, we used an established theory to guide the selection of potential explanatory variables. Furthermore, although conveniently sampled, our study population is representative of the target population of persons diagnosed with sickle cell disease, which enhances the generalizability of our results. We believe that the findings in this study can, at the least, be a spring board for clinical and research discussions about insomnia in adults with SCD.

Conclusion

Overall, this study found that clinically significant insomnia is prevalent among community-dwelling African-American adults with SCD. Furthermore, while insomnia symptoms may be associated with a number of bio-psychosocial factors, depression and acute pain may the only independent risk factors.

Clinical Implications

Given the high number of participants who reported clinically significant insomnia symptoms, clinical nurses should proactively screen for insomnia symptoms when working patients with SCD. To screen for insomnia symptoms, brief self-report measures (e.g. Insomnia Severity Index) could be administered during an outpatient clinical visit. When feasible, the use of sleep diaries should be encouraged. Sleep diaries illustrate fluctuations in sleep patterns over time, which then provides a better assessment of the quality and quantity of one’s sleep (Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006).

Awareness of and referrals to primary care clinicians to treat pain and depression, when present, are also important for nurses working with patients with SCD. According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR), insomnia is the most common sleep disturbance associated with a major depressive episode (*American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders*, 2013). In our study, approximately 40% of participants scored above the clinical cut-off (CESD-10 ≥ 10) for clinically relevant depression, suggesting insomnia and depression may be comorbid conditions in this population. Similarly, numerous studies and ours have found a relationship between pain and sleep disturbances in patients with chronic pain conditions including SCD (Valrie, Bromberg, Palermo, & Schanberg, 2013) thus suggesting that nurses working with patients with SCD should continue to routinely assess clinical pain severity. When clinical

pain is not well controlled, administering pain medications (as ordered) or advocating for pain management treatments (e.g. pharmacological or non-pharmacological) is encouraged.

Finally, nurses should explore insomnia interventions with their patients. Understanding the role of sleep hygiene education or cognitive behavioral therapy directed at insomnia (CBT-I) may improve quality of life indicators in SCD patients. CBT-I in particular, is a tailored behavior change technique, which changes one's belief about their sleep as well as their sleep behaviors. Both sleep hygiene education and CBT-I are alternatives to prescription sleep medications and can be easily incorporated into nursing practice to increase the quality and quantity of sleep of SCD patients.

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Table 1

Participant characteristics[a]

	Total (N = 263) n (%)	Males (n = 119) n (%)	Females (n = 144) n (%)	p
Age, years*	35.6 (11.8)	34.9 (11.5)	36.2 (12.1)	0.38
Education				
High school or less	161 (62.2)	69 (59.5)	92 (64.3)	0.73
Some college education	44 (17.0)	21 (18.1)	23 (16.1)	
College Grad or greater	54 (20.9)	26 (22.4)	28 (19.6)	
Employment				
Working	94 (35.7)	45 (37.8)	49 (34.0)	0.66
Unemployed	36 (13.7)	15 (12.6)	21 (14.6)	
Disability	113 (43.0)	52 (43.7)	61 (42.4)	
Retired	9 (3.4)	2 (2.0)	7 (4.9)	
Other	11 (4.2)	5 (4.2)	6 (4.2)	
Genotype				
SS or HbS/β^0-Thalassemia	186 (70.7)	83 (69.8)	103 (71.5)	0.75
SC or HbS/β^+-Thalassemia	77 (29.3)	36 (30.3)	41 (28.5)	
Chronic Pain				
No	115 (43.4)	49 (41.2)	66 (46.2)	0.42
Yes	147 (56.1)	70 (58.8)	77 (53.9)	
BPI severity*	3.8 (2.5)	3.7 (2.6)	3.9 (2.4)	0.62
ULSS*	16.8 (12.0)	17.4 (12.4)	16.4 (11.7)	0.52
CESD-10*	8.9 (6.0)	9.1 (5.8)	8.8 (6.2)	0.68
ISI	11.1 (7.0)	11.0 (6.7)	11.3 (7.3)	0.73

Note: BPI = Brief Pain Inventory; CESD-10 = Center for Epidemiologic Studies in Depression; ISI = Insomnia Severity Index; ULSS = Urban Life Stress Scale

*Mean (SD)

[a]N = 263, smaller n's are related to missing data

Table 2

Association between insomnia symptom severity and potential risk factors: Pearson's correlation coefficients and student's t-test results

		t-test	Pearson's Correlation	
	M (SD)	t(*df*)	*r*	p-value
Age			0.06	0.34
Gender				
Males	10.97 (6.6)	−.34 (258.2)		0.073
Females	11.27 (7.3)			
Education				
Less than college	11.57 (6.9)	2.2 (81.3)		0.03
College or greater	9.19 (7.1)			
Disability Status				
Not Disabled	9.96 (7.0)	−3.1 (246.5)		0.002
Disabled	12.7 (6.7)			
Genotype				
SS or Beta (0) Thalassemia	10.44 (6.5)	−2.4 (123.7)		0.02
SC or Beta (+)Thalassemia	12.83 (7.7)			
Chronic Daily Pain				
No	8.29 (6.5)	−6.2 (245.3)		< 0.001
Yes	13.31 (6.6)			
BPI Severity			0.41	< 0.001
ULSS			0.45	< 0.001
CESD-10			0.68	< 0.001

Note: BPI = Brief Pain Inventory; CESD-10 = Center for Epidemiologic Studies in Depression; ISI = Insomnia Severity Index; M = Mean; SD = Standard Deviation; ULSS = Urban Life Stress Scale

Table 3

Stepwise backward linear regression results for a model of independent predictors of insomnia symptomatology[a]

Variable	$\hat{\beta}$ (95% CI)	p-value
Constant	3.02 (1.73, 4.32)	0.002
Acute Pain (BPI)	0.45 (0.17, 0.73)	< 0.001
Depressive Symptoms (CESD-10)	0.71 (0.59, 0.82)	< 0.001
Disability Status		0.83
Chronic Pain		0.78
SCD Genotype		0.45
Educational Attainment		0.21
Stress (ULSS)		0.10
Final Model	F(2,236) = 113.6	<0.001

Note: BPI = Brief Pain Inventory; CESD-10 = Center for Epidemiologic Studies in Depression; SCD = Sickle Cell Disease; ULSS = Urban Life Stress Scale. Results are reported as unstandardized beta coefficients ($\hat{\beta}$).

[a] n = 239