HHS Public Access

Author manuscript

*Int J Behav Med*. Author manuscript; available in PMC 2020 November 30.

Published in final edited form as:

*Int J Behav Med*. 2018 December ; 25(6): 698–705. doi:10.1007/s12529-018-9751-y.

# The Nature of Trauma Pain and Its Association with Catastrophizing and Sleep

**Michelle C. Accardi-Ravid**[1], **Joshua R. Dyer**[2], **Sam R. Sharar**[3], **Shelley Wiechman**[4], **Mark P. Jensen**[4], **Hunter G. Hoffman**[5], **David R. Patterson**[4]

[1]Department of Anesthesiology & Pain Medicine, Harborview Medical Center, University of Washington, 325 9th Avenue, Seattle, WA 98104, USA

[2]Department of Family Medicine, Peace Health Medical Group, Vancouver, USA

[3]Department of Anesthesiology & Pain Medicine, University of Washington, Seattle, WA, USA

[4]Department of Rehabilitation Medicine, University of Washington, Seattle, WA, USA

[5]Department of Mechanical Engineering, University of Washington, Seattle, WA, USA

## Abstract

**Background—**Nearly 2.8 million people are hospitalized in the USA annually for traumatic injuries, which include orthopedic and internal organ injuries. Early post-injury pain is predictive of poor outcomes, including inability to eventually return to work, and long-term psychological distress. The goal of the present study was to improve our scientific understanding of trauma-related pain by examining (1) the nature and frequency of inpatient trauma pain and (2) the associations between inpatient trauma pain, education, opioid analgesic equivalent use, pain catastrophizing, and sleep quality.

**Method—**The study included 120 patients hospitalized at a major level I regional trauma center for the care of (1) closed long bone or calcaneus fractures and/or (2) an intraabdominal injury caused by blunt force trauma and requiring surgical repair (i.e., laparotomy). Medical records were reviewed to obtain demographic information and information about opioid use during hospitalization. In addition, participants were administered measures of average pain intensity, pain catastrophizing, and sleep quality.

**Results—**Education, opioid analgesic equivalents, catastrophizing, and poor sleep quality together accounted for 28% of the variance of average pain intensity over a 24-h period ($p < .001$), with each variable making a significant independent association.

**Conclusion—**Two of the factors associated with pain intensity in the study sample—catastrophizing and sleep quality—are modifiable. It is therefore possible that interventions that target these variables in patients who are hospitalized for trauma could potentially result in better

Michelle C. Accardi-Ravid, mca9@uw.edu.

**Conflict of Interest** The authors declare that they have no conflicts of interest.

**Ethical Approval** All procedures performed in studies involving human participants were in accordance with the University of Washington Institutional Review Board and registered on ClinicalTrials.gov (NCT00739076).

**Informed Consent** Informed consent was obtained from all individual participants included in the study.

long-term outcomes, including a reduced risk for developing chronic pain. Research to evaluate this possibility is warranted.

**Keywords**

Trauma; Pain; Catastrophizing; Sleep quality; Opioid analgesic equivalents; Education

## Introduction

Pain is a global phenomenon, with the prevalence of acute and postoperative pain remaining generally stable across developed nations [1], despite advances in medical knowledge. In the USA alone, nearly 2.8 million people are hospitalized each year for traumatic injuries [2], with the highest proportion of injury type represented by orthopedic fractures and internal organ injuries [3–6]. Trauma-related pain has been a consistently reported factor in the development of persistent pain, particularly among patients with orthopedic injuries [7]. Traumatic injuries account for significant length of stay, with one study finding an average of 5.7 bed days for at least one fracture diagnosis [5]. At the time of discharge from the hospital, up to 97% of patients with traumatic orthopedic injuries continue to report persistent pain [8], with 48 to 59% reporting moderate to severe pain [8, 9]. This is significant, as pain at hospital discharge predicts chronic pain at 6 months after discharge [9], and up to 63% of those with trauma pain continue to report pain 1 year after discharge [10]. Early post-injury pain is also predictive of poor outcomes, including inability to eventually return to work [11–13], and long-term psychological distress [14]. This is consistent with previously reports that injuries account for a substantial burden of disease across the world [15], musculoskeletal injuries, in particular [2, 16].

While the majority of hospitalized patients experience acute pain [17], the available literature has found only a weak relationship between injury severity and persistent pain [18]. Therefore, it is important to identify other factors that predict how acute pain, beyond a categorical diagnosis of trauma, might increase the likelihood of developing persistent pain. We know that biopsychosocial factors are consistently predictive of chronic pain in trauma populations. For example, in addition to a categorical diagnosis of trauma, higher pain intensity, higher anxiety and distress, and less certainty that pain will resolve during hospitalization have all been found to predict the development of chronic pain following surgery [19]. A systematic review found that alcohol consumption prior to traumatic injury, peritraumatic pain, anxiety, and depression, eligibility for compensation, lower educational status, and older age were significantly and consistently predictive of persistent pain [18]. The existing literature also suggests that lower education has been found to be associated with higher trauma-related pain intensity [8, 9], though this finding has not been consistent [20]. Many of these factors can be identified during inpatient hospitalization, thus offering an important opportunity for further assessment and potential intervention.

Although there are a number of implicated factors between acute pain and the development of persistent pain that leads to disability following traumatic injury, two stand out as particularly important because of the possibility that they could be targets for intervention. First, pain-related catastrophizing cognitions have a significant association with the

development and maintenance of acute and chronic pain in outpatient samples [21]. Such cognitions (e.g., “I will never recover from this pain”; “This pain means there is something seriously wrong with me”) have been shown to be associated with adverse postsurgical outcomes including pain ratings, opioid medication usage, depression, activity interference, and disability levels [21]. Hypothesized mechanisms of action for catastrophizing involve cognitive appraisals, attentional bias, learning theory, central nervous system alterations, and physiological pathways [21]. In fact, the authors conclude that “high levels of catastrophizing about pain should be considered a ‘risk marker’ for adverse immediate and long-term pain-related outcomes” [21].

The second potentially important modifiable factor that could contribute to long-term pain outcomes in hospitalized patients (particularly those with trauma) is sleep quality. The definition of sleep quality varies considerably in the literature; however, one study [22] found that, in addition to traditional descriptions of sleep (onset, maintenance, total time) and disruptions (early awakenings, environmental disturbance), sleep quality is also associated with subjective feelings upon awakening (mood, feeling refreshed, or restored), daytime symptoms (tiredness, alertness), coping behaviors, and cumulative effects of other recent sleep experiences. In addition to a desire for adequate pain control, tiredness, fatigue, and sleep are among the most commonly reported needs of medical patients [23]. Among those with traumatic burn injuries, distress from trouble falling asleep in the days leading up to hospital discharge has been shown to be associated with pain during hospitalization and predicts long-term difficulties with pain [24]. Further support comes from evidence indicating that factors that disrupt sleep, such as hospitalization, can worsen pain [25]. It has also been suggested that the presence of sleep disturbance during the acute hospitalization period is likely to have durable neurophysiologic changes that then impact longer term problems with both pain and sleep [24].

This body of literature suggests that early identification of factors that predict a higher likelihood of acute pain during initial hospitalization for traumatic injury (i.e., identification prior to discharge) is a critical window for identifying potential treatment targets that could improve long-term pain outcomes. Such information is important in order to identify (1) those most likely to experience trauma-related pain (and who therefore may require more close monitoring and treatment) as well as (2) modifiable factors that could be targeted with treatments. As mentioned previously, early post-injury pain may be a determinant of poor long-term outcomes, including subsequent chronic pain [9, 10], inability to return to work [11–13], and long-term psychological distress [14].

Given these considerations, the aim of the present study was to improve our scientific understanding of trauma-related pain by examining (1) the nature and frequency of inpatient trauma pain and (2) the associations between inpatient trauma-related pain, demographic and treatment factors, pain catastrophizing, and sleep problems. Specifically, we hypothesized that lower education and higher levels of opioid analgesic equivalent use, catastrophizing, and sleep problems would all make significant and independent contributions to the prediction of trauma pain in a sample of patients hospitalized for significant traumatic injuries.

## Materials and Methods

### Participants

The study participants were a sample of patients hospitalized at a major level I regional trauma center for the care of (1) closed long bone or calcaneus fractures and/or (2) an intraabdominal injury caused by blunt force trauma and requiring surgical repair (i.e., laparotomy). This subset of patients was chosen for addressing the study questions in order to ensure that the sample was as homogeneous as possible, while also allowing for a large enough sample to perform the planned analyses. These two groups of injury (1) represent the two most frequent US hospital admissions [3] while at the same time (2) enabled us to avoid a broad range of other trauma etiologies (e.g., burn injuries, gunshot wounds, facial trauma) that have been studied elsewhere [14, 24] or introduce substantial heterogeneity. Subjects were part of a National Institute of Health (NIH) randomized trial of nonpharmacologic interventions for pain control after trauma; however, the data presented in this study were gathered prior to undergoing any of the analgesic interventions specific to the trial. The inclusion criteria for the study included the following: age 12 years or older, hospitalization for orthopedic injuries (closed long bone or calcaneus fractures), and/or intraabdominal injuries requiring a laparotomy or for a minimum of 2 days in the hospital, decisional capacity to consent to study, able to communicate verbally, and English-speaking. Exclusion criteria consisted of evidence of traumatic brain injury, currently receiving prophylaxis for alcohol or drug withdrawal, face/head/neck injuries preventing helmet use, extreme susceptibility to motion sickness, seizure history, body substance isolation procedures, incarcerated, homeless, or pregnant. Injuries that prevent helmet use, motion sickness, seizure history, and body substance isolation procedures were exclusion criteria because a subset of this sample subsequently participated in a virtual reality analgesia intervention later in their hospitalization. The study was approved by our Institutional Review Board and registered on ClinicalTrials.gov (NCT00739076).

### Procedures

Upon hospital admission and triage to an orthopedic or general surgery trauma inpatient service, patients who met exclusion/inclusion criteria were identified by the research team as potential study participants. The identified patients were first approached by a clinical care team member for permission to be approached by a research staff member. Those who expressed an interest in potentially participating then met with a research study coordinator or assistant, who discussed and explained the study further and provided the patients with the consent form for their review. The research coordinator/assistant then obtained written, informed consent from those eligible patients who expressed a willingness to participate, using a procedure approved by the Institutional Review Board. In case of minors, written permission to participate was obtained from both a parent or guardian and the patient. Data were gathered over the next 24 h. Participants were consented and studied within an average of 6.5 days following their initial hospital admission.

Patient demographic information was collected for all participants. These data were extracted from the participant's medical records and included the following information: age, sex, ethnicity, cause of injury, type of injury, date of injury, date of hospital admission,

date of study initiation, education, vocational and marital status, and length of hospitalization. For patients not interested in participating, basic non-identifying demographic information relevant to the study (age, sex, type of injury) and respectful of the patient privacy, was extracted from their records (in compliance with HIPAA restrictions).

All eligible patients were approached in person in their hospital room. Patients had to be at least 12 years old, be able to read and understand the consent/assent forms, provided only in English, and be able to complete the questionnaires. The data presented in this paper was collected from October of 2008 to December of 2012.

## Measures

The study measures administered to participants included (1) a Graphic Rating Scale of pain intensity, (2) the Pain Catastrophizing Scale (both administered within the first 48 h of consent), and (3) a modified version of the Medical Outcomes Study Sleep Problem Index I (administered in the first 24 h of consent). All response categories in this study were used based on previously published versions of the scales, unless otherwise noted. Opioid analgesic equivalents and demographic information were obtained via a combination of medical records review and patient interview.

The Graphic Rating Scale (GRS) is a 0–10 numerical scale with word descriptors indicating varying levels of pain intensity associated with the different numbers along the scale (i.e., 0 = “No pain at all,” 2 = “Mild pain,” 8 = “Severe pain,” 10 = “Excruciating pain”). Here, the tool was used to assess average pain intensity over the past 24 h. The GRS is more sensitive than simple categorical pain scales, and patients can easily use them despite having no previous experience with the measure [26].

The Pain Catastrophizing Scale (PCS) is a 13-item measure of pain catastrophizing commonly used in pain research [27]. The items describe a variety of catastrophic thoughts and feelings individuals might experience, and respondents are asked to indicate the frequency that they have the responses described when they are in pain on a 5-point Likert scale (i.e., 0 = “Not at all,” 1 = “To a slight degree,” 2 = “To a moderate degree,” 3 = “To a great degree,” 4 = “All the time”). The PCS Total scale has been shown to have adequate to excellent internal consistency [27] (Cronbach’s $\alpha = 0.87$). In the current sample, the internal consistency of the PCS total scale score was $\alpha = 0.93$, indicating excellent reliability.

The Medical Outcomes Study (MOS) Sleep Problem Index I is one of the indices and subscales included in the MOS measures of sleep battery [28]. This 6-item index describe different aspects of sleep quality such as difficulty falling asleep, feeling rested upon awakening, and getting the amount of sleep needed. In the original version of the measure, participants indicate the frequency of each sleep quality domain on a 6-point Likert scale (i.e., 1 = “All the time,” 2 = “Most of the time,” 3 = “A good bit of the time,” 4 = “Some of the time,” 5 = “A little of the time,” 6 = “None of the time”). The various indices and subscales of the MOS measures of sleep have been shown to be reliable and valid [28], and are frequently used in populations with pain [29, 30]. The Sleep Problem Index I has a documented internal consistency of $\alpha = 0.78$ [28]. In the present study, we adapted the index by changing the time frame from the original “the past four weeks” to “last night” as we

wanted to capture sleep quality during hospitalization only. Consistent with this, the responses were adapted from the original 6-point Likert scale to a 4-point Likert scale (1 = “Yes, definitely”, 2 = “Yes, somewhat”, 3 = “Yes, a little”, 4 = “No”) to match the “last night” time frame. Lower scores indicated poor sleep quality. The adaptations to the MOS scale from a previous 4-week to a “last night” hour time frame was justified as the patients were consented and studied within an average of 6.5 days of hospital admission. In the present study, the measure evidenced marginal reliability (Cronbach’s $\alpha$ = 0.59).

### Opioid Analgesic Equivalents

Because all patients received systemic analgesics for standard clinical pain control consisting of opioid and non-opioid medications at the discretion of the physicians on the primary clinical service, we wanted to account for this in our analyses. Opioid analgesics reported used by the study participants, as documented in their medical records, were converted to opioid equivalents using the American Pain Society’s standard equivalency recommendations [31]. One opioid equivalent is theoretically equipotent in analgesic effect with morphine 10 mg intravenously. In the sample investigated, the opioid analgesics used were codeine, fentanyl, hydrocodone, hydromorphone, meperidine, metha-done, morphine, oxycodone, and tramadol, while the non-opioid analgesics included acetaminophen, aspirin, ibuprofen, and ketorolac.

### Statistical Analyses

Means and standard deviations of the study variables were computed for descriptive purposes. Pearson correlation coefficients were computed to evaluate the zero order relationships between the study variables. Variable skew, kurtosis, normality, multicollinearity, and homoscedasticity were examined to ensure they met the assumptions for the planned parametric (i.e., Pearson correlation and linear regression) analyses. The Bonferroni test was used to correct for multiple post hoc comparisons of average pain intensity. Given the inconsistent relationship between education and pain reported in the literature, differences in reported pain based upon education level were assessed. Finally, a linear regression analysis was conducted to test the relationship between the independent variables of education (block 1), opioid analgesic equivalents (block 1), catastrophizing cognitions (block 2), and sleep problems (block 2), and the dependent variable of average pain intensity over the past at 24 h, entered stepwise. The regression was followed with a one-way ANOVA to determine if there was a significant effect of education on reported levels of average pain intensity.

## Results

### Demographic Characteristics

The patient demographic characteristics are shown in Table 1. One hundred and twenty-three patients were recruited to the study; however, two were excluded from analyses due to incomplete data. The sample was predominantly male (70%) and Caucasian (74%) with a mean age of 34.6 years (SD = 13.6). Seventy-four percent had at least a high school education. Thirty-eight percent suffered orthopedic injuries, 24% suffered intraabdominal injuries, and 36% had both. Another 115 patients were approached for consent, but declined

to enter the study. They did not vary significantly on demographic variables of age ($t(220) = 1.572$, $p = 0.117$) and gender ($X^2(1) = 0.032$, $p = 0.858$) (Table 1).

### Mean Pain Intensity and Relevant Correlations

As expected, participants reported moderate pain (see Table 2)—with average pain intensity over the past 24 h rated at 6.3/10 (SD = 2.3). With regard to severity, 21% reported average pain over 24 h in the mild range (0–4.9), 36% reported moderate pain (5.0–6.9), and 43% reported pain over 24 h in the severe range (7.0–10.0). Opioid analgesic equivalents ranged from 0.2 to 35.7 with a mean of 4.9 (SD = 4.6), and demonstrated a low but statistically significant correlation with average pain intensity over 24 h, $r = .25$ ($p = 0.007$). Catastrophizing was associated with average pain ($r = 0.38$, $p = 0.000$), lower education ($r = -0.28$, $p = 0.002$), and poor sleep quality ($r = -0.26$, $p = 0.003$), but not opioid analgesic equivalents ($r = 0.09$, $p = 0.339$). Poor sleep quality was similarly associated with average pain ($r = -0.38$, $p = 0.000$), education ($r = 0.18$, $p = 0.054$), and catastrophizing, but not opioid analgesic equivalents ($r = -0.14$, $p = 0.124$). Post hoc comparisons of average pain intensity indicated that the mean score for college graduates ($\mu = 4.6$, SD = 2.2) was significantly different than those with high school education ($\mu = 6.5$, SD = 2.1, $p = .027$) or less than a high school education ($\mu = 7.0$, SD = 2.4, $p = 0.004$), but not different from those with some college education ($\mu = 6.1$, SD = 2.1, $p = 0.134$). However, the mean score for persons with some college education ($\mu = 6.1$, SD = 2.1) was not significantly different from the other education levels (less than high school education $p = 0.680$, high school education $p = 0.999$, college graduates $p = 0.134$).

### Regression Analyses Predicting Pain Intensity

Given that education and opioid analgesic equivalents were related to pain, we controlled for this by entering them into the first block of the regression (see Table 3). Education, opioid analgesic equivalents, catastrophizing, and poor sleep quality together accounted for 28% of the variance of average pain intensity over the past 24 h ($p = 0.001$), with each variable making a significant independent association.

## Discussion

The present study sought to increase our knowledge of the nature of inpatient pain in the early days following significant traumatic orthopedic and/or intraabdominal injuries, with a focus on identifying potentially modifiable predictors of pain, catastrophizing, and sleep quality. A notable 79% of those with traumatic injuries reported moderate to severe average pain over 24 h despite receiving opioid analgesics. The reported mean average pain over 24 h was 6.3 on a scale that was converted to a 0–10 for ease of interpretation. Consistent with our first hypothesis, higher catastrophizing and poor sleep quality were associated with higher levels of pain intensity.

Those reporting higher levels of pain also had lower education, received more opioid analgesic medication, tended to catastrophize more, and reported poor sleep quality. Although the association with lower education has been reported in other samples from a population of individuals hospitalized for trauma-related pain [8, 9], the findings regarding

catastrophizing and sleep problems have not yet been reported in this population. Catastrophizing refers to the tendency of patients to “think the worst of their pain.” There have been many studies demonstrating an association between pain intensity and catastrophizing in samples of individuals with chronic pain [32], but this variable has received little attention in the acute pain literature, particularly in populations with acute traumatic injury pain. The findings here identify a potential target for intervention (i.e., catastrophizing thoughts) in this population, as such thoughts are both amenable to change and predictive of those who might suffer [33].

The association between pain and poor sleep quality is well established in the chronic pain literature [34], but has received minimal attention with respect to acute pain. Similarly, poor sleep quality is a frequent complaint in the hospital setting [23–25], but its association with pain has received little attention. Given the potential impact of sleep quality on adjustment and recovery, the importance of improved pain control becomes even more substantial. Our findings are consistent with findings from the pain literature indicating that pain and sleep quality seem to have bidirectional impacts on one another [24, 25]. We cannot assume that interventions for sleep quality that would be implemented at home are similarly effective during inpatient hospitalization; there are significant differences between the two settings, including situational factors (acuity of medical needs, disturbances from other patients/families), environmental factors (sounds, lighting), and individual factors (changes in routine, cognition, mood). Future studies might investigate potential interventions for sleep quality during hospitalization, in order to directly evaluate the effects on peritraumatic pain and catastrophizing in this population.

Our sample reported higher levels of acute pain than what is reported in other similar cohorts in other studies [8, 9]. The high levels of pain in the present sample may reflect a number of factors. First, given that the study was conducted at a level I regional trauma center with an expertise in orthopedics, the nature of the injuries in the sample were likely more severe, with rates of both surgical interventions and those with multiple traumatic injuries higher possibly higher than previous reports. Second, as discussed below, pain was associated with less education, and the participating hospital has one of the highest rates of low socioeconomic status in the region. Third, several measurements of pain were made over a 24-h period as part of a larger study, and the findings may reflect an artifact of the frequency of measurement. In any case, the high levels of pain intensity in this sample are of concern and indicate the need to be vigilant of this problem in any level I trauma center, particularly since it can be associated with chronic pain after trauma at follow-up [9, 10]. Further, we acknowledge that without repeated assessment at later stages, it is not possible to generalize that the acute pain experienced in this study will necessarily lead to the development of chronic or persistent pain.

This study is a preliminary investigation of the nature of trauma pain early during hospitalization, as well as associated variables, following orthopedic and intraabdominal injuries. As such, number of limitations should be noted. First, the sample size was relatively small. Although pain was prospectively assessed with standardized tools, it was only assessed over a single 24-h period an average of 6 days after hospital admission. It would be preferable to monitor pain over the entire hospitalization in order to increase the reliability

of assessment. Scores on the Pain Catastrophizing Scale were somewhat lower in this population, relative to those of outpatients with chronic pain. It is possible that the presence of around-the-clock assessment and treatment for acute pain while receiving inpatient care was a mitigating factor in the development of catastrophic cognitions. Additionally, we modified the MOS Sleep Problem Index I for the purposes of our study and it had lower than ideal reliability. However, the fact that sleep quality was still a significant predictor of pain intensity despite the relatively low reliability of this measure argues for the reliability of the finding. Future studies may benefit from using other validated measures of sleep quality and/or multimodal assessment of sleep. The use of a self-report measure for sleep quality is problematic as it is prone to retrospective biases. Future studies are needed to evaluate the effects of more objective sleep measures, such as actigraphy or polysomnography. Lastly, we did not distinguish between pain due solely to the traumatic injury and postoperative pain in those patients who had already undergone surgical treatment of their traumatic injuries.

Despite the study's limitations, the findings provide new information regarding pain and its correlates in a sample of hospitalized patients with acute (trauma-related) pain. We found that the majority of participants in this study who were hospitalized for the care of orthopedic or internal organ injuries reported high levels of pain in spite of treatment with conventional doses of opioid analgesics. We found that this under-investigated type of pain was associated with catastrophizing cognitions and poor sleep quality, as well as educational level and opioid analgesic equivalence. The findings shed light on the severity of this problem and identified two modifiable factors that have the potential to help these patients gain better control over pain. Research to evaluate the potential benefits of interventions that reduce catastrophizing cognitions and improve sleep quality in this population is warranted.

## Funding

This work was supported with funds from the National Institute of Health (R01GM042725; R01AR054115) and National Institute of General Medical Sciences.

## References

1. Waldman SD. Management of acute and postoperative pain In: Waldman SD, editor. Pain management. Philadelphia: Saunders; 2011 p. 216–27.
2. Centers for Disease Control and Prevention, National Center for Injury Prevention and Control. Web-based injury statistics query and reporting system (WISQARS). 2012 http://www.cdc.gov/injury/wisqars. Accessed 15 October 2012.
3. DiMaggio C, Ayoung-Chee P, Shinseki M, Wilson C, Marshall G, Lee DC, et al. Traumatic injury in the United States: in-patient epidemiology 2000–2011. Injury. 2016;47(7):1393–403. [PubMed: 27157986]
4. The burden of musculoskeletal diseases in the United States: Prevalence, societal, and economic cost, 2nd edition. American Academy of Orthopedic Surgeons; 2008.
5. Bradley C, Harrison J. Descriptive epidemiology of traumatic fractures in Australia. Injury research and statistics series number 17: Australian Institute of Health and Welfare; 2004.
6. Kendall JL, Kestler AM, Whitaker KT, Adkisson MM, Haukoos JS. Blunt abdominal trauma patients are at very low risk for intraabdominal injury after emergency department observation. West J Emerg Med. 2011;12(4):496–504. [PubMed: 22224146]
7. Rosenbloom BN, Khan S, McCartnet C, Katz J. Systematic review of persistent pain and psychological outcomes following traumatic musculoskeletal injury. J Pain Res. 2013;6:39–51. [PubMed: 23357964]

8. Archer KR, Castillo RC, Wegener ST, Abraham CM, Obremskey WT. Pain and satisfaction in hospitalized trauma patients: the importance of self-efficacy and psychological distress. J Trauma Acute Care Surg. 2012;72(4):1068–77. [PubMed: 22491629]
9. Williamson OD, Epi GD, Gabbe BJ, Physio B, Cameron PA, Edwards ER, et al. Predictors of moderate or severe pain 6 months after orthopaedic injury: a prospective cohort study. J Orthop Trauma. 2009;23(2):139–44. [PubMed: 19169107]
10. Rivara FP, Mackenzie EJ, Jurkovich GJ, Nathens AB, Wang J, Scharfstein DO. Prevalence of pain in patients 1 year after major trauma. Arch Surg. 2008;143(3):282–7. [PubMed: 18347276]
11. MacKenzie EJ, Bosse MJ, Kellam JF, Pollak AN, Webb LX, Swiontkowski MF, et al. Early predictors of long-term work disability after major limb trauma. J Trauma. 2006;61(3):688–94. [PubMed: 16967009]
12. Clay FJ, Fitzharris M, Kerr E, McClure RJ, Watson WL. The association of social functioning, social relationships and the receipt of compensation with time to return to work following unintentional injuries to Victorian workers. J Occup Rehabil. 2012;22(3):363–75. [PubMed: 22278297]
13. Iakova M, Ballabeni P, Erhart P, Seichert N, Luthi F, Dériaz O. Self perceptions as predictors for return to work 2 years after rehabilitation in orthopedic trauma inpatients. J Occup Rehabil. 2012;22(4):532–40. [PubMed: 22562093]
14. Wiechman Askay S, Patterson DR. What are the psychiatric sequelae of burn pain? Curr Pain Headache Rep. 2008;12(2):94–7. [PubMed: 18474187]
15. Krug EG, Sharma GK, Lozano R. The global burden of injuries. Am J Public Health. 2000;90(4):523–6. [PubMed: 10754963]
16. Mock C, Cherian MN. The global burden of musculoskeletal injuries. Clin Orthop Relat Res. 2008;466:2306–16. [PubMed: 18679760]
17. Wadensten B, Fröjd C, Swenne CL, Gordh T, Gunningberg L. Why is pain still not being assessed adequately? Results of a pain prevalence study in a university hospital in Sweden. J Clin Nurs. 2011;20(5–6):624–34. [PubMed: 21320191]
18. Rosenbloom BN, Khan S, McCartney C, Katz J. Systematic review of persistent pain and psychological outcomes following traumatic musculoskeletal injury. J Pain Res. 2013;6:39–51. [PubMed: 23357964]
19. White CL, LeFort SM, Amsel R, Jeans ME. Predictors of the development of chronic pain. Res Nurs Health. 1997;20:309–18. [PubMed: 9256877]
20. Clay FJ, Newstead SV, Watson WL, Ozanne-Smith J, Guy J, McClure RJ. Bio-psychosocial determinants of persistent pain 6 months after non-life-threatening acute orthopaedic trauma. J Pain. 2010;11(5):420–30. [PubMed: 20439055]
21. Quartana PJ, Campbell CM, Edwards RR. Pain catastrophizing: a critical review. Expert Rev Neurother. 2009;9(5):745–58. [PubMed: 19402782]
22. Harvey AG, Stinson K, Whitaker KL, Moskovitz D, Virk H. The subjective meaning of sleep quality: a comparison of individuals with and without insomnia. Sleep. 2008;31(3):383–93. [PubMed: 18363315]
23. Lauri S, Lepistö M, Käppeli S. Patients' needs in hospital: nurses' and patients' views. J Adv Nurs. 1997;25(2):339–46. [PubMed: 9044009]
24. Smith MT, Klick B, Kozachik S, Edwards RE, Holavanahalli R, Wiechman S, et al. Sleep onset insomnia symptoms during hospitalization for major burn injury predict chronic pain. Pain. 2008;138(3):497–506. [PubMed: 18362052]
25. Young JS, Bourgeois JA, Hilty DM, Hardin KA. Sleep in hospitalized medical patients, part 1: factors affecting sleep. J Hosp Med. 2008;3(6):473–82. [PubMed: 19084897]
26. Scott J, Huskisson EC. Graphic representation of pain. Pain. 1976;2(2):175–84. [PubMed: 1026900]
27. Sullivan MJL, Bishop SR, Pivik J. The pain catastrophizing scale: development and validation. Psychol Assess. 1995;7(4):524–32.
28. Hays R, Stewart A. Sleep measures In: Stewart A, Ware JE Jr, editors. Measuring functioning and well-being: the medical outcomes study approach. Durham: Duke University Press; 1992 p. 235–59.

29. Koroschetz J, Rehm SE, Gockel U, Brosz M, Rainer F, Tolle TR, et al. Fibromyalgia and neuropathic pain–differences and similarities. A comparison of 3057 patients with diabetic painful neuropathy and fibromyalgia. BMC Neurol. 2011;11:55. [PubMed: 21612589]

30. Kim JS, Bashford G, Murphy TK, Martin A, Dror V, Cheung R. Safety and efficacy of pregabalin in patients with central post-stroke pain. Pain. 2011;152(5):1018–23. [PubMed: 21316855]

31. Max M, Payne R. Principles of analgesic use in the treatment of acute pain and cancer pain. 3rd edition. American Pain Society 1992:12–20.

32. Jensen MP, Moore MR, Bockow TB, Ehde DM, Engel JM. Psychosocial factors and adjustment to chronic pain in persons with physical disabilities: a systematic review. Arch Phys Med Rehabil. 2011;92(1):146–60. [PubMed: 21187217]

33. Glombiewski JA, Sawyer AT, Gutermann J, Koenig K, Rief W, Hofmann SG. Psychological treatments for fibromyalgia: a meta-analysis. Pain. 2010;151(2):280–95. [PubMed: 20727679]

34. Fishbain DA, Cole B, Lewis JE, Gao J. What is the evidence for chronic pain being etiologically associated with the DSM-IV category of sleep disorder due to a general medical condition? A structured evidence-based review. Pain Med. 2010;11(2):158–79. [PubMed: 19788712]

35. Jaffe SE, Patterson DR. Treating sleep problems in patients with burn injuries: practical considerations. J Burn Care Rehabil. 2004;25(3):294–305. [PubMed: 15273471]

**Table 1**

Description of study sample

| Variable | Consented | Non-consented |
|---|---|---|
| Participants | *N* = 121 | *N* = 115 |
| Age (year) | 34.6 ± 13.6 | 37.7 ± 16.6 |
| Days since injury | 6.5 ± 8.5 | |
| Sex | | |
| Male | 85 (70.2) | 82 (71.3) |
| Female | 36 (29.8) | 33 (28.7) |
| Marital status | | |
| Married or cohabitating | 66 (54.5) | |
| Single, divorced, or widowed | 55 (45.5) | |
| Ethnicity | | |
| White/Caucasian | 91 (74.4) | |
| Black | 16 (13.2) | |
| Hispanic/Chicano | 5 (4.1) | |
| Native American | 4 (3.3) | |
| Asian | 4 (3.3) | |
| Other | 2 (1.7) | |
| Vocational status | | |
| Employed full time | 47 (38.8) | |
| Employed part time | 11 (9.1) | |
| Student | 27 (22.3) | |
| Retired | 2 (1.7) | |
| Unemployed | 34 (28.1) | |
| Education * | | |
| Did not graduate high school | 30 (24.8) | |
| High school graduate | 36 (29.8) | |
| Some college | 37 (30.6) | |
| College graduate | 17 (14.0) | |
| Type of injury | | |
| Orthopedic | 46 (38.0) | |
| Intraabdominal | 29 (24.0) | |
| Both | 43 (35.5) | |
| Other | 3 (2.5) | |

Values are mean ± SD or *n* (%)

* Does not equal 121 due to missing data/no response

**Table 2**

Means, standard deviations, and Pearson correlations for pain, education, opioid equivalents, catastrophizing, and sleep problems

| Variable | Mean ± SD | 1 | 2 | 3 | 4 | 5 |
|---|---|---|---|---|---|---|
| 1. Average pain over 24 h† | 6.3 ± 2.3 | - | | | | |
| 2. Education†† | - | −0.30*** | - | | | |
| 3. Opioid equivalents | 4.9 ± 4.6 | 0.25** | 0.07 | - | | |
| 4. Catastrophizing | 21.5 ± 13.4 | 0.38*** | −0.28** | 0.09 | - | |
| 5. Sleep | 14.2 ± 4.2 | −0.38*** | 0.18* | −0.14 | −0.26** | - |

† Range = 0–10

†† Mean and standard deviation not provided

* $p \leq 0.05$

** $p \leq 0.01$

*** $p \leq 0.001$

Lower sleep scores indicate more sleep problems

**Table 3**

Results of the linear regression predicting Average Pain Intensity at 24 h

| Step and variable | B | SE | *β* | *t* | *P* | 95% CI for *t* | |
|---|---|---|---|---|---|---|---|
| | | | | | | Lower bound | Upper bound |
| Step 1 | | | | | | | |
| Constant | 72.22 | 5.33 | | 13.55 | 0.000 | 61.66 | 82.78 |
| Education | −6.93 | 1.96 | −3.05 | −3.53 | 0.001 | −10.81 | −3.05 |
| Opioid equivalents | 1.35 | 0.43 | 0.27 | 3.11 | 0.002 | 0.49 | 2.21 |
| Step 2 | | | | | | | |
| Constant | 78.09 | 9.44 | | 8.27 | 0.000 | 59.38 | 96.79 |
| Education | −4.51 | 1.91 | −0.20 | −2.36 | 0.020 | −8.29 | −0.73 |
| Opioid equivalents | 1.04 | 0.41 | 0.21 | 2.54 | 0.012 | 0.23 | 1.86 |
| Catastrophizing | 0.40 | 0.15 | 0.23 | 2.65 | 0.009 | 0.10 | 0.69 |
| Sleep | −1.31 | 0.46 | −0.24 | −2.86 | 0.005 | −2.21 | −0.40 |

$R^2$ = 0.15 for step 1 ($p$ = 0.000); $\Delta R^2$ = 0.13 for step 2 ($p$ = 0.001)